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NURSING REVIEW OF EFFECTS OF SECOND-HAND SMOKE INHALATION IN EARLY LIFE

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Abstract:

Although early childhood tobacco smoke exposure has been extensively examined, the impact of second-hand smoke (SHS) in new-onset respiratory symptoms and lung function decrease in adults has received less attention in longitudinal investigations. We search the electronic databases; PubMed and Embase for all the studies that were published discussing the concern topic. According to newly revealed information, even quick and short-term SHS exposure has considerable negative consequences on the human respiratory system. SHS has been found to be independently related with respiratory tract symptoms such as sneeze, sore throat, cough, and nasal rhinitis. Chronic SHS exposure of those who worked in hospitality settings contributed to decreased exercise performance among healthy adult nonsmokers. Its impact on the development of chronic diseases should be investigated further.

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INTRODUCTION:

Second-hand smoke (SHS) is still one of the most common indoor contaminants in the world. According to a 2011 overview report, up to 40% of children, 35% of women, and 33% of men were regularly exposed to second-hand smoke indoors worldwide [1]. Children that are exposed to passive smoke have poor lung growth [2]. The influence of ambient cigarette smoke on respiratory diseases and lung function, on the other hand, has received little attention, and the connections are less obvious in adults [3,4]. Emerging data suggests that second-hand smoking exposure is linked to the development of chronic obstructive pulmonary disease (COPD). SHS exposure during adulthood has been linked to lung and circulatory disorders [5]. Since 1986, the Surgeon General of the United States has considered SHS to be a cause of lung cancer6, and more recently, strong evidence for a link with deadly coronary heart and cerebrovascular disease has been discovered. Associations between SHS and adult-onset asthma, chronic obstructive pulmonary disease (COPD), and other lung disorders are suggestive but not causal [5]. Because of findings that breast epithelia may be more sensitive to carcinogenic insults during this time of tissue proliferation and initial differentiation, there is growing interest that early life exposures may be associated to breast cancer risk [6]. Pregnancy and lactation cause additional differentiation, following which breast tissue appears to be more resistant to carcinogenic shocks [7,8]. Despite significant research on the relationships of childhood SHS exposure with health outcomes in children and adult SHS exposure with health outcomes in adults, there are few confirmed links with childhood SHS exposure and adult mortality [9]. It is possible that childhood SHS exposure that causes lung and cardiovascular system damage might raise the likelihood of fatal consequences in adulthood [9].

METHODOLOGY:

Narrative review of literature was conducted through electronic databases; PubMed, Embase, searching studies that were published concerning the topic of this review, up to the middle of 2021. Studies were limited to human subjects published in English language.

DISCUSSION:

Secondhand smoke (SHS) was initially identified as a concern to nonsmokers in 1981 by Hirayama and Trichopoulos et al., who discovered an elevated risk of getting lung cancer in nonsmoking women married to smokers [10]. Since then, scientific evidence has consistently linked SHS exposure to disease and early death in both adults and children [11]. SHS has been linked to CVD, stroke, lung cancer, chronic obstructive pulmonary disease (COPD), asthma aggravation, and reproductive consequences in women, such as low birth weight infants. Children who are exposed to SHS are more sensitive, risking rhinitis, eye irritation, middle ear infection, increased respiratory symptoms such as cough, chest pain, wheezing, lower respiratory infections, and sudden infant death syndrome [12].

Smoking is known to have a negative impact on the cardiovascular (CV) system and exercise performance, and SHS exposure is also linked to an increase in CV risk [13,14]. There have been few studies on occupational SHS exposure and its influence on nonsmokers' cardiorespiratory (CR) response to exercise testing. McMurray et al. [15] studied a group of 8 women by smoking status, with and without SHS exposure using simulated smoke during exercise testing, and Flouris et al. [7] studied SHS exposure using self-burning cigarettes to simulate bar-level exposure among healthy non-smoking adults via exercise testing. Thier de Borba et al. assessed CR response in the general population segregated by smoking status using submaximal exertion incremental testing on a treadmill [16]. Arjomandi et al. evaluated the flow-volume curves, diffusion capacity, and lung volumes of non-smoking flight attendants in airplane cabins prior to the adoption of commercial smoking prohibitions in 2009 [12].

Preventive tobacco control initiatives, such as smoking bans in public places, have been adopted by several nations, yet an estimated 84% of the population remains unprotected [12,15]. Unfortunately, even with legislation in place, several countries have failed to enforce the bans, as is the case in Greece, and others have failed to include coverage of hospitality venues. Individuals working in SHS-polluted situations, such as hospitality venues, are thus exposed to SHS on a daily and chronic basis, emphasizing the necessity of studying the effect of long-term exposure on CR health [15,16].

The evidence for dose-dependent correlations between passive smoking and causes of death is consistent with prior findings for lung cancer and coronary heart disease, and it expands on the data for stroke. Previous research has linked passive smoking to first-time acute strokes, and we now have a dose-response relationship with stroke death. Previous research concentrated on ischemic strokes, however Chinese populations have a higher prevalence of hemorrhagic stroke than white populations, suggesting that majority of the strokes in our study were non-ischemic. Passive smoking, like

active smoking, is likely to affect all stroke subtypes [10,11,13].

Only a few research have looked into the link between second-hand smoke exposure and the development of asthma in adults. Coogan et al. found a positive connection between passive smoke exposure and the incidence of adult-onset asthma among 46,182 women aged 21 to 69 years at baseline in the prospective U.S. Black Women's Health Study [17]. Nonsmoking study participants who were exposed to second-hand smoke exhibited a 21% increase in asthma incidence (adjusted HR: 1.2; 95%-CI: 1.0-1.5) compared to those who were not exposed. Two Finnish population-based case-control studies [18,19] yielded similar results. Second-hand smoke exposure at work or at home increased the likelihood of developing asthma throughout a 2.5-year period [19].

Previous research has looked into the link between second-hand smoke exposure and respiratory symptoms and disorders, including COPD. For example, Eisner et al. [20] investigated the influence of lifetime exposure to second-hand smoke on the risk of developing COPD in 2112 persons aged 55 to 75 years in the United States (including current, former, and never smokers). It discovered a link between cumulative exposure to second-hand smoke at home (adjusted OR: 1.6; 95%-CI: 1.1-2.2) and self-reported doctor-diagnosed COPD (adjusted OR: 1.4; 95%-CI: 1.0-1.8). Based on data from the Guangzhou Biobank Cohort Study, a Chinese study [21] evaluated the connection of self-reported density and duration of passive smoking exposure with respiratory symptoms (cough, phlegm, and shortness of breath) and COPD (FEV1/FVC 0.7 assessed pre-bronchodilation). Second-hand smoke exposure at home and at work was linked to an increased risk of COPD (adjusted OR: 1.5; 95%-CI: 1.2-1.9) and any respiratory symptoms (adjusted OR: 1.2; 95%-CI: 1.1-1.3).

In a study [22] of 2182 lifelong never smokers participating in the Obstructive Lung Disease in Northern Sweden (OLIN) trials, those with second-hand smoke exposure had an elevated risk of COPD, defined using the fixed ratio of FEV1/FVC 0.7 assessed post-bronchodilation. Second-hand smoke exposure was classified into several groups depending on prior and present second-hand smoke exposure at home and at work. Those who had ever been exposed at home and at both previous and current jobs had the highest connections (adjusted OR: 3.8; 95%-CI: 1.3-11.2), as did those who were currently exposed at home and at both previous and current jobs (adjusted OR: 5.7; 95%-CI: 1.5-22.5). Another study [15] could reveal a substantial dose-dependent association between second-hand smoke exposure and mortality from several diseases, including COPD among other causes of death. In contrast, Chan-Yeung et al. [23] discovered no link between second-hand smoke exposure and an elevated risk of COPD in a small sex- and age-matched case-control study of 289 patients and controls in Hong Kong, China.

Another study [24] looked at the relationship between second-hand smoke exposure and chronic bronchitis in women and found that women who were exposed to second-hand smoke had a 3.7 (95% CI: 1.2-11.3) higher risk of chronic bronchitis than those who were not exposed to second-hand smoke. Second-hand smoke exposure was also linked to mild (adjusted OR: 1.8; 95%-CI: 1.1-2.9) and moderate (adjusted OR: 3.8; 95%-CI: 1.7-8.6) COPD, as defined by GOLD.

Janson et al. [25] investigated changes and determinants of changes in active and passive smoking in the first and second surveys of the European Community Respiratory Health Survey, finding that second-hand smoke exposure was higher among subjects with lower socioeconomic status and educational level. Furthermore, those exposed to second-hand smoke were less likely to quit smoking, implying that reducing second-hand smoke exposure could be useful in reducing active smoking.

The link between SHS exposure and sinusitis has not been thoroughly examined in adults, and the majority of evidence linking SHS to middle ear infections originates from the pediatric literature [12]. However, there is a pathophysiologic basis for causality because SHS produces inflammation in the nasal mucosa and nonsmokers may be more sensitive to SHS exposure [9]. Unfortunately, and predictably, tobacco industry actions to undercut US regulatory bodies have hampered research on the negative health implications of SHS exposure [10].

Exhaled carbon monoxide (eCO) levels were not observed to differ substantially between children from smoking and non-smoking homes, despite the fact that mean eCO levels were 17.6% higher in children from smoking homes. Exhaled CO was also shown to be unrelated to the amount of cigarettes smoked per day. There are several explanations for this finding, including previous research's finding of eCO's low sensitivity in predicting SHS exposure in children, age-related ability to perform the physically demanding test, and exposure to other CO sources prior to the test, such as the road microenvironment, industry, and solid fuel burning [24]. Cotinine, a

nicotine metabolite, can be identified in the hair, saliva, urine, and blood of people who have been exposed to SHS and is a common alternative measure used in research to determine recent active and passive smoking [26].

CONCLUSION:

According to newly revealed information, even quick and short-term SHS exposure has considerable negative consequences on the human respiratory system. SHS has been found to be independently related with respiratory tract symptoms such as sneeze, sore throat, cough, and nasal rhinitis. Chronic SHS exposure of those who worked in hospitality settings contributed to decreased exercise performance among healthy adult nonsmokers. Its impact on the development of chronic diseases should be investigated further.

REFERENCES:

1. Oberg M, Jaakkola MS, Woodward A, Peruga A, Prüss-Ustün A. Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. *Lancet.* 2011;377(9760):139–146. doi: 10.1016/S0140-6736(10)61388-8.
2. Strachan DP, Cook DG. Health effects of passive smoking. 5. Parental smoking and allergic sensitisation in children. *Thorax.* 1998;53:117–123. doi: 10.1136/thx.53.2.117.
3. Strachan DP, Cook DG. Health effects of passive smoking. 6. Parental smoking and childhood asthma: longitudinal and case-control studies. *Thorax.* 1998;53:204–212. doi: 10.1136/thx.53.3.204.
4. Cook DG, Strachan DP, Carey IM. Health effects of passive smoking. 9. Parental smoking and spirometric indices in children. *Thorax.* 1998;53:884–893. doi: 10.1136/thx.53.10.884.
5. HHS. The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General. Atlanta, GA: HHS, CDC, National Center for Chronic Disease Prevention and Health Promotion, Office of Smoking and Health, 2014.
6. Trichopoulos D (1990) Hypothesis: does breast cancer originate in utero. Lancet 335: 939–940.
7. Russo J, Reina D, Frederick J, Russo IH (1988) Expression of phenotypical changes by human breast epithelial cells treated with carcinogens in vitro. Cancer Res 48: 2837–2857.
8. Russo J, Calaf G, Sohi N, Tahin Q, Zhang PL, Alvarado ME, et al. (1993) Critical steps in breast carcinogenesis. Ann NY Acad Sci 698: 1–20.
9. HHS. The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. Atlanta, GA: HHS, CDC, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office of Smoking and Health, 2006.
10. Hirayama T. Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. *Br Med J (Clin Res Ed)* 1981;282(6259):183–5. doi: 10.1136/bmj.283.6296.915.
11. Trichopoulos D, Kalandidi A, Sparros L MB. Lung cancer and passive smoking. *Int J Cancer.* 1981;27(1):1–4. doi: 10.1002/ijc.2910270102.
12. Kobayashi Y, Takeuchi T, Hosoi T, Loeppky JA. Effects of habitual smoking on cardiorespiratory responses to sub-maximal exercise. *J Physiol Anthropol Appl Human Sci [Internet]* 2004 Sep;23(5):163–9. doi: 10.1186/2049-6958-9-34.
13. Arjomandi M, Haight T, Redberg R, Gold WM. Pulmonary function abnormalities in never-smoking flight attendants exposed to secondhand tobacco smoke in the aircraft cabin. *J Occup Environ Med [Internet]. NIH Public Access.* 2009 Jun;51(6):639–46. doi: 10.1097/JOM.0b013e3181a7f048.
14. Institute of Medicine Secondhand Smoke Exposure and Cardiovascular Effects: Making Sense of the Evidence. *Institute of Medicine.* 2009
15. McMurray RG, Hicks LL, Thompson DL. The effects of passive inhalation of cigarette smoke on exercise performance. *Eur J Appl Physiol Occup Physiol.* 1985;54(2):196–200. doi: 10.1007/BF02335929.
16. Thier de Borba AT, Jost RT, Gass R, Nedel FB, Cardoso DM, Pohl HH, et al. The influence of active and passive smoking on the cardiorespiratory fitness of adults. *Multidiscip Respir Med [Internet]* 2014;9(1):34. doi: 10.1186/2049-6958-9-34.
17. Sutton P, Vergara X, Beckman J, Das R. Occupational illness among flight attendants due to aircraft disinsection. *CA Department of Health Services, Occupational Health Branch, Oakland, CA.* 2003.
18. Whelan EA, Lawson CC, Grajewski B, Petersen MR, Pinkerton LE, Ward EM, Schnorr TM. Prevalence of respiratory symptoms among female flight attendants and teachers. *Occup Environ Med.* 2003;**60**:929–934. doi: 10.1136/oem.60.12.929.

19. Hocking MB. Trends in cabin air quality of commercial aircraft: industry and passenger perspectives. *Rev Environ Health.* 2002;**17**:1–49.

20. Eisner MD, Balmes J, Katz PP, Trupin L, Yelin EH, Blanc PD. Lifetime environmental tobacco smoke exposure and the risk of chronic obstructive pulmonary disease. *Environ Health.* 2005;**4**(1):7. doi: 10.1186/1476-069X-4-7.
21. Yin P, Jiang CQ, Cheng KK, Lam TH, Lam KH, Miller MR, et al. Passive smoking exposure and risk of COPD among adults in China: the Guangzhou biobank cohort study. *Lancet.* 2007;**370**(9589):751–757. doi: 10.1016/S0140-6736(07)61378-6.
22. Hagstad S, Bjerg A, Ekerljung L, Backman H, Lindberg A, Rönmark E, et al. Passive smoking exposure is associated with increased risk of COPD in never smokers. *Chest.* 2014;**145**(6):1298–1304. doi: 10.1378/chest.13-1349.
23. Chan-Yeung M, Ho ASS, Cheung AHK, Liu RWT, Yee WKS, Sin KM, et al. Determinants of chronic obstructive pulmonary disease in Chinese patients in Hong Kong. *Int J Tuberc Lung Dis.* 2007;**11**(5):502–507.
24. Wu CF. Feng NH, Chong IW, Wu KY, Lee CH, Hwang JJ, et al. *Second-hand smoke and chronic bronchitis in Taiwanese women: a health-care based study BMC Public Health.* 2010;**10**:44.
25. Janson C, Künzli N, de Marco R, Chinn S, Jarvis D, Svanes C, et al. Changes in active and passive smoking in the European Community respiratory health survey. *Eur Respir J.* 2006;**27**(3):517–524. doi: 10.1183/09031936.06.00106605.