

Indoor air pollution

Kapalı ortam hava kirliliği

Duygu Kavuncuoğlu¹, Zahide Koşan¹

¹ Department of Public Health, Ataturk University
Faculty of Medicine, Erzurum, Turkey

ORCID ID of the author(s)

DK: 0000-0002-0546-5478

ZK: 0000-0002-1429-6207

Abstract

Clean air is one of the essential preconditions of life. Approximately 4.3 million people die due to indoor air pollution every year. The majority of these deaths are due to stroke (34%), ischemic heart disease (26%), and chronic obstructive pulmonary disease (22%). The principal indoor air pollutant particulate materials are carbon monoxide, tobacco smoke, formaldehyde and volatile organic compounds, nitrogen dioxide, asbestos, radon, and biological pollutants (micro-organisms and allergens). The prevention of indoor air pollution and use of clean energy sources must be regarded as an opportunity to improve health, particularly that of mothers and children, reduce poverty, and achieve environmental sustainability.

Keywords: Air pollution, Indoor air pollution, Sick building syndrome

Öz

Temiz hava hayatın temel şartlarından biridir. Her yıl kapalı ortam hava kirliliği nedeniyle 4.3 milyon insan yaşamını kaybetmektedir. Bu ölümlerin, çoğu inme (%34), iskemik kalp hastalığı (%26) ve kronik obstrüktif akciğer hastalığı (%22) kaynaklıdır. Başlıca kapalı ortam hava kirlleticileri partiküler madde, karbonmonoksit, tütün dumanı, formaldehit ve uçucu organik bileşikler, nitrojen dioksit, asbest, radon, biyolojik kirleticiler (mikroorganizmalar ve allerjenler) olarak sıralanabilir. Kapalı ortam hava kirliliğinin engellenmesi ve temiz enerji kaynaklarının kullanılması, özellikle anne-çocuk sağlığı olmak üzere sağlığı iyileştirmek, yoksulluğu azaltmak ve çevresel sürdürülebilirliğin sağlanması için bir fırsat olarak değerlendirilmelidir.

Anahtar kelimeler: Hava kirliliği, Kapalı ortam hava kirliliği, Hasta bina sendromu

Corresponding author / Sorumlu yazar:

Duygu Kavuncuoğlu

Address / Adres: Atatürk Üniversitesi Tıp Fakültesi,
Halk Sağlığı Anabilim Dalı, Yakutiye, Erzurum,
Türkiye

e-Mail: duygu_koylu@hotmail.com

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Introduction

Clean air is an essential precondition for life. Air quality in homes, schools, day care centers, health institutions and other private and public buildings where humans spend much of their lives is a basic determinant of healthy living and well-being [1].

The presence of harmful biological, physical and chemical agents such as carbon monoxide, sulfur dioxide, nitrogen oxides, formaldehyde, cigarette smoke, radon, asbestos, volatile organic compounds, various micro-organisms, and allergens, with their deleterious effects on human health, in closed environments such as homes, non-industrial workplaces, and official buildings, is defined as 'indoor pollution' [2,3].

Hazardous materials deriving from structural components of buildings and internal equipment, forming for reasons such as fuel consumption for day-to-day activities such as cooking and heating, can result in severe health problems, even with fatal consequences. Approximately 4.3 million people die due to indoor air pollution every year. The majority of these deaths are due to stroke (34%), ischemic heart disease (26%) and chronic obstructive pulmonary disease (22%) [1].

Social and economic prosperity levels are closely associated with indoor air pollution markers. There are considerable differences between developing and industrialized countries. Indoor air pollution is related to the characteristics of the structure concerned, the materials used in its construction, the heating system and fuel use, ventilation, the behaviors of the residents, and external environmental conditions [1].

Approximately 3 billion people worldwide use solid fuels (such as wood, plant wastes, charcoal, coal, and manure) to meet their basic domestic energy requirements. The majority of these live in low- and moderate-income countries. Components of indoor air pollution far exceed acceptable levels in housing in which such fuels are used without adequate ventilation. Exposure is high among women and children who spend the most time close to domestic ranges particularly used for heating and/or cooking [4]. According to World Health Organization (WHO) data, more than 50% of pneumonia-related deaths among children under five years are associated with indoor air pollution [1].

In addition to fuel-based pollutants, various other elements such as asbestos, wood protection materials, volatile organic compounds, paints, adhesives, resins, polishing materials, perfumes, spray gases, and cleaning materials also cause indoor pollution [5].

Indoor air pollution also varies between rural and urban regions, in association with economic conditions and lifestyle. For example, dust and organic particles are more common in agricultural areas, and pollutants such as mites and fungi, in urban dwellings. Regional climatic conditions, architectural factors (construction materials, structure, and room distribution and characteristics), and particularly the ventilation of homes must also be considered since they are also effective. People living in urban areas who spend the majority of their time indoors are exposed to a relatively higher level of indoor air pollution.

The acute health impacts of indoor air pollutants include an increased incidence of mucosal damage, cough, wheezing,

increased thoracic pressure, airway susceptibility to allergens, and pneumonia, as well as middle ear infection, trachea-bronchiolitis, and asthma flare-ups. In the chronic period, air pollution has an adverse impact on child growth, causes increasing susceptibility to chronic obstructive lung diseases including asthma by leading to pulmonary function disorder, and diseases such as ischemic heart disease, stroke, lung cancer, cataract, and tuberculosis. Air pollution has also been linked to low birth weight in newborns [6].

Another effect on health of indoor air pollution is described as sick building syndrome. During the energy crisis in the 1970s, thermal insulation began being used to maintain environmental heat in buildings. This gave rise to increasing indoor air pollution because of inadequate ventilation in some buildings. Pollution caused by construction materials and other environmental pollutants was trapped inside buildings and increased to levels capable of producing symptoms in susceptible individuals. Sick building syndrome refers to a series of symptoms emerging in a closed environment and resolving when leaving that environment, and affecting the majority of individuals living in the building [7-9]. Probable causes include problems concerning building architecture, work-related factors, building repairs being carried out in a manner incompatible with the original design, insufficient ventilation, inadequate maintenance, and chemical and biological pollutants. In 1982, the WHO listed the symptoms observed in sick building syndrome under five categories: ear, nose and throat damage, skin damage, idiopathic hypersensitivity reactions, taste and smell-related findings, and neurological or general health symptoms [7,10].

The principal indoor air pollutants are as follows:

Particulate matter

The combustion of fossil fuels (such as coal), organic materials (such as wood and peat), and other substances such as rubber and plastic, motorized vehicles, power station emissions, and forest fires are the principal sources of particulate matter (PM). The pathogenicity of PMs is determined by their size, compounds, origins, solubility, and capacity to produce reactive oxygen. PMs with an aerodynamic diameter less than 10 μm have been found to have a greater effect on human health.

According to U.S. Environmental Protection Agency (EPA) standards, the recommended mean annual threshold for PM_{10} (breathable particles ≤ 10 micrometers in diameter) is 50 $\mu\text{g}/\text{m}^3$. Several studies have shown that biomass used as fuel causes very high particle levels in homes, that the mean 24-h PM_{10} value in environments with open fires is 1000 $\mu\text{g}/\text{m}^3$ and can even exceed 10,000 $\mu\text{g}/\text{m}^3$ when specimens are collected during open fire use. Considering that this occurs daily in homes using biomass, mean pollution levels are approximately 20 times higher than the limit recommended by the EPA. There are two important components showing individuals' exposure to indoor pollution; environmental pollutant density and length of exposure of each individual to this. Women, young girls up to the age when they begin to walk, and girls from the time when they begin to acquire culinary skills are known to be exposed to greater indoor pollution, about at least 3-5 hours a day. Exposure to pollutants may be prolonged in some societies and in cold regions [11].

The PM group defined as PM_{2.5} has small diameters and larger surface areas. They can thus pass through nasal filtration, be carried by air currents to the farthest points of the respiratory canal and accumulate there [12]. PM_{2.5} can cause asthma and respiratory tract infections, adversely affects pulmonary functions, and can even lead to cancer [13-15]. Studies have determined a positive correlation between PM_{2.5} levels and respiratory tract disease prevalence, hospitalization rates, and daily death rates [16,17]. These relations are more marked in the elderly, pregnant women, babies, adolescents, subjects with a history of cardiopulmonary disease and other susceptible populations [18-20]. One study of a seven-year period in the USA (from 2000 to 2007) showed that every 10 µg / m³ decrease in PM_{2.5} extended life spans by 0.35 years [21].

Carbon monoxide

Carbon monoxide (CO) is a colorless, odorless gas produced by the insufficient combustion of fossil-based fuels. Any situation involving inadequate combustion can produce carbon monoxide, such as gas, kerosene, wood- coal-burning stoves, chimneys, cooking ranges, leaking stove pipes and chimneys, room and water heaters, vehicle exhaust in closed garages, and tobacco smoke.

Mean threshold levels set by the US Environmental Protection Agency (EPA) are 50 ppm for 8 hours, 75 ppm for 4 hours, and 125 ppm for one hour, although there is no agreed standard for indoor air CO.

The affinity of CO for hemoglobin (Hb) is 240-270 times greater than that of oxygen, and it reduces the capacity of Hb to transport oxygen to tissues. Intoxication results in tissue hypoxia, and multiple organs, particularly the central nervous and cardiovascular systems are affected with their high metabolic rates. Intervention may be delayed since symptoms of exposure are not specific. Exposure to CO is particularly hazardous to babies and individuals with a history of asthma or heart disease. At low levels, fatigue and chest pain are exacerbated in individuals with chronic heart disease. At higher levels of exposure, breathing CO causes symptoms such as headache, dizziness, and loss of strength in healthy individuals. CO can also cause sleepiness, nausea, vomiting, confusion, and disorientation. Very high levels of exposure can result in loss of consciousness and death, and may produce irreversible sequelae [22].

Carboxyhemoglobin (COHb) measurement confirms that exposure has taken place, although there is no correlation between severity of intoxication and COHb levels. Normal COHb levels are 3-8% in smokers and 1-3% in non-smokers. The half-life of COHb is 4 h at room temperature, 1 h with 100% O₂, and 20-30 min with hyperbaric oxygen.

Tobacco smoke

Tobacco smoke consists of more than 3800 different substances such as CO, carbon dioxide, oxides of nitrogen, polyaromatic hydrocarbons, and numerous toxic materials. The PM rate in air is 2-3 times higher in homes where the residents smoke [23,24].

Environmental tobacco smoke components are divided into the smoke exhaled by the smoker, and smoke disseminated from the tip of the cigarette. Non-smokers exposed to environmental tobacco smoke are exposed to the majority of

toxins and various additional substances emitted by active smokers. For example, levels of N-nitrosodimethylamine, a proven carcinogenic substance in animals, in main smoke are 20-100 times higher than those in subsidiary smoke [25], and a 'passive' smoker 50 cm from a cigarette can breathe more than ten times the active carbonyl compounds inhaled by the active smoker [26]. Smoking one pack a day contributes approximately 20 µg/m³ to 24-h indoor particle levels. In addition, it has been concluded that the moment a cigarette is lit, it has a probable increasing effect of 500-1000 µg/m³ on short-term particle concentrations [27].

Health impacts associated with exposure to tobacco smoke range from nose and throat irritation, worsening of asthma symptoms in children and adults, and lower respiratory tract diseases to lung cancer [8,28-31]. One case-control study of 191 individuals in the USA concluded that approximately 17% of lung cancers among non-smokers were associated with exposure to environmental tobacco smoke at early ages [32].

Formaldehyde and volatile organic compounds

The sources of indoor formaldehyde vary among different countries; the main sources in developing countries are solid fuels used indoors, insect repellents, and furniture, while in developed countries they are domestic cleaning products and deodorants, glues and resins, tobacco smoke, carpets, furniture and paint materials, and insulating foams. A colorless gas with a pungent odor, formaldehyde can result in a burning sensation in the eyes and throat, nausea, and respiratory difficulty. High concentrations can trigger attacks in asthma patients. Chronic formaldehyde inhalation causes damage to the nasal and other respiratory tract mucosa, and an increased incidence of lung and nasopharyngeal cancer [33].

Organic chemicals are widely used in domestic products such as paint, varnish, sealing wax, cosmetic products, wood preservers, cleaning materials and disinfectants, moth repellents, sprays, and hobby materials. In addition, fuel can also be made from organic chemicals. All these products result in the release of a specific quantity of organic chemicals during use and in the area concerned. Mean indoor levels of various organic compounds are 2-5 times higher than external levels. The effects on health of volatile organic products can vary significantly; some are highly toxic, while others have no known health impacts. Exposure to volatile organic compounds can lead to symptoms such as irritation of the eyes, nose and throat, headache, loss of coordination, nausea, dizziness and eye disorders, and to impaired memory and liver, kidney, and central nervous system damage [34]. Chronic exposure to benzene can lead to bone marrow depression and associated hematological diseases and leukemia [35].

Nitrogen dioxide

The best-known sources of indoor nitrogen dioxide (NO₂) are gas and oil stoves used for cooking and cigarette smoke. The current WHO air quality guideline recommends a mean indoor annual value of 40 µg/m³ for NO₂. Indoor NO₂ levels in winter are 2-3 times higher than external levels, while the two are approximately equal in summer. NO₂ is a non-water-soluble gas. At low levels it can be expelled via mucus secretions on the upper respiratory tract but reaches the lower respiratory tract after being inhaled. If inhaled at high levels, cough,

wheezing, respiratory failure, nausea, dyspnea, fatigue and anxiety may be seen [36].

A mean increase equivalent to $28 \mu\text{g}/\text{m}^3$ in indoor NO_2 has been determined in homes using solid fuels compared to electricity, increasing the risk of respiratory difficulty in children by 20%.

Asbestos

Asbestos is a general term referring to the group of hydrated silicate minerals with an incombustible filament structure that occur in various forms [8]. In the 1970s, asbestos was widely used in various areas of industry, due to its electrical and thermal insulation properties, such as the production of pipe insulation, cement slabs, tiles, paint, and wallpaper. Concerns over the health effects of exposure to asbestos have led to legal measures prohibiting its use in many countries [37]. Exposure to asbestos generally involves respiration of the fibers and causes diseases such as lung cancer, mesothelioma, and asbestosis [38,39]. In order for asbestos fibers to affect health, they need to remain in the respiratory passages for approximately one year, and those less than $1 \mu\text{m}$ in diameter and greater than $5\text{-}10 \mu\text{m}$ in length have been shown to be particularly hazardous [37].

Radon

Radon is a colorless, odorless, radioactive gas arising from the natural breakdown of thorium and uranium in rock and soil. Radon exposure represents more than 50% of annual radiation exposure of natural origin [40]. The most important sources of indoor radon are various structural materials, emissions from the soil, rock and natural fissures beneath and around the building, and well water [41]. Radon general involves no health risk in the open air, since outdoor levels are quite low. However, it may accumulate at dangerous levels in closed environments. Differences in air pressure between the inside of a building and the surrounding soil also play a significant role in radon emissions. If the air pressure in a house is greater than that in the soil beneath, then the radon remains outside. However, if it is lower than that of the surrounding soil (as is generally the case), then the internal pressure behaves like a vacuum cleaner attracting radon gas. Since radon is emitted from the soil, geological data for the area can help predict potential indoor radon levels. Cell culture, animal experiments, and epidemiological cohort and case-control studies have proven that radon causes cancers such as leukemia and lung cancer. Radon has been classified as a Group 1 carcinogen by the International Agency for Research on Cancer [42-45]. Approximately 14,000 adults die from radon-related cancer in the USA every year. In light of the latest scientific data, the World Health Organization recommends a reference level of $100 \text{Bq}/\text{m}^3$ in order to reduce health risks associated with indoor radon exposure to a minimum [41].

Biological Pollutants (micro-organisms and allergens)

Domestic animals, their fur and secretions, moisturizers, ventilation systems, drainage pipes, and pipes can constitute indoor growth areas for micro-organisms, fungi, and algae. Moisture is essential for dust mites, fungi, and bacteria to multiply. Rain or subterranean water entering a building and accumulating on internal surfaces can facilitate the growth of micro-organisms. These micro-organisms can then cause various

contagious diseases, allergic reactions, and toxic effects in those living in that environment. Adequate air flow is essential to prevent moisture problems associated with water vapor produced and exhaled by humans and domestic animals, or from cooking and cleaning. Legionella can be present and grow in ventilation system water discharge channels, stagnant water pipes, and shower pipes, and is one of the agents involved in pneumonia in humans. Higher number of people in an indoor environment facilitate the spread of infectious diseases. Mites found in floor furnishings, bed surfaces and beddings may lead to asthma, particularly in children. Pollen in the air can also lead to asthma [1-5].

Conclusion

Understanding the hazards posed by substances giving rise to indoor air pollution constitutes the first step to preventing adverse health effects and identifying the requisite precautionary measures [46]. Education and establishing individual awareness and sensibility are therefore particularly important. Education will help in the search for diverse ways of managing energy better, protecting children in the home, and reducing risks. Individuals must also be educated about using clean alternative energy sources instead of the direct burning of biomass fuel. However, in addition to the public, politicians and administrators must also reinforce their commitment and awareness of the health impacts of indoor pollution.

Fuel use is affected by sociocultural and economic factors. Habits, availability and most importantly, affordability influence fuel selection. Most low-income families directly employ biomass fuel for cooking, since this is the cheapest and simplest option available. The use of accessible, economical, and clean energy sources must be encouraged. Stoves used for heating and/or lighting, and which are traditionally smoky and leaky, must be replaced with those that are fuel efficient, non-smoke emitting, and have reliable means of eliminating indoor pollutants. Importance must be attached to adequate ventilation during the construction of dwellings, and appropriate architectural measures must be introduced for homes with inadequate ventilation.

The prevention of indoor air pollution and the use of clean sources of energy must be regarded as an opportunity to improve health, particularly that of mothers and children, to reduce poverty, and to achieve environmental sustainability. Indoor air pollution can only be controlled through coordinated and determined endeavor on the part of different sectors associated with health, energy, the environment, and rural and urban regeneration. The prevention of indoor air pollution will result in economic and social regeneration.

References

1. Indoor air pollution. 14.01.2018]; Available from: <https://www.who.int/features/qa/indoor-air-pollution/en/>.
2. Myers I, Maynard RL. Polluted air—outdoors and indoors. *Occup Med (Lond)*. 2005;55(6):432-8.
3. McKenzie JF, Pinger RR, Kotecki JE. An introduction to community health. London: Jones & Bartlett Publishers; 2011.
4. Kurmi OP, Semple S, Steiner M, Henderson GD, Ayres JG. Particulate matter exposure during domestic work in Nepal. *Annals of occupational hygiene*. 2008;52(6):509-17.
5. Cooke TF. Indoor Air Pollutants: A Literature Review. *Reviews on Environmental Health*. 1991;9(3):137-60.
6. Kırımhan S, Boyabat N. Erzurum'da hava kirliliği-Son rapor. Atatürk Üniversitesi Çevre Sorunları Sempozyumu-5, Erzurum, 1983;12:6-8.
7. Spellman FR. The science of air: concepts and applications. Boca Raton, Fla: CRC Press; 2016:300.
8. Maroni M, Seifert B, Lindvall T. Indoor air quality: a comprehensive reference book. Vol. 3. Amsterdam: Elsevier; 1995.
9. Wang LK, Pereira NC, Hung YT. Advanced air and noise pollution control; Totowa, New Jersey: Humana Press; 2005.

- 10.Chang CC, Ruhl RA, Halpern GM, Gershwin ME. The sick building syndrome. I. Definition and epidemiological considerations. 1993;30(4):285-95.
- 11.Raaschou-Nielsen O, Andersen ZJ, Beelen R, Samoli E, Stafoggia M, Weinmayr G, et al. Air pollution and lung cancer incidence in 17 European cohorts: prospective analyses from the European Study of Cohorts for Air Pollution Effects (ESCAPE). *The Lancet Oncology*. 2013;14(9):813-22.
- 12.Xing YF, Xu YH, Shi MH, LianXing YX. The impact of PM_{2.5} on the human respiratory system. *J Thorac Dis*. 2016;8(1):E69-74.
- 13.Samoli E, Analitis A, Touloumi G, Schwartz J, Anderson HR, Sunyer J, et al. Estimating the exposure-response relationships between particulate matter and mortality within the APHEA multicity project. *Environmental Health Perspectives*. 2004;113(1):88-95.
- 14.Ostro B, Broadwin R, Green S, Feng WY, Lipssett M. Fine particulate air pollution and mortality in nine California counties: results from CALFINE. *Environmental Health Perspectives*. 2005;114(1):29-33.
- 15.Lewis TC, Robins TG, Dvonch JT, Keeler GJ, Yip FY, Mentz GB, et al. Air pollution-associated changes in lung function among asthmatic children in Detroit. *Environmental Health Perspectives*. 2005;113(8):1068-75.
- 16.Zanobetti A, Franklin M, Koutrakis P, Schwartz J. Fine particulate air pollution and its components in association with cause-specific emergency admissions. *Environ Health*. 2009;8(1):58.
- 17.Dominici F, Peng RD, Bell ML, Pham L, McDermott A. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *JAMA*. 2006;295(10):1127-34.
- 18.Huynh M, Woodruff TJ, Parker JD, Schoendorf KC. Relationships between air pollution and preterm birth in California. 2006;20(6):454-61.
- 19.Martinelli N, Girelli D, Cigolini D, Sandri M, Ricci G, Rocca G, et al. Access Rate to the Emergency Department for Venous Thromboembolism in Relationship with Coarse and Fine Particulate Matter Air Pollution. *PLoS ONE*. 2011;7(4):e34831.
- 20.de Oliveira BFA, Ignotti E, Artaxo P, do Nascimento Saldiva PH, Junger WL, Hacon S. Risk assessment of PM_{2.5} to child residents in Brazilian Amazon region with biofuel production. *Environ Health*. 2012;11(1):64.
- 21.Correia AW, Pope CA, Dockery DW, Wang Y, Ezzati M, Dominici F. The effect of air pollution control on life expectancy in the United States: an analysis of 545 US counties for the period 2000 to 2007. *Epidemiology*. 2013 Jan;24(1):23-31.
- 22.Indoor Air Quality (IAQ). <https://www.epa.gov/indoor-air-quality-iaq>. Accessed 7 December 2018.
- 23.Güler Ç. Kapalı Ortam Hava Kirliliği. *Mesleki Sağlık ve Güvenlik Dergisi (MSG)*. 2015; 3(12):25-32.
- 24.Gold D. Indoor air pollution. *Clinics in Chest Medicine*. 1992;13(2):215-29.
- 25.Jenkins RA, Tomkins B, Guerin MR. The chemistry of environmental tobacco smoke: composition and measurement. CRC Press; 2000.
- 26.Schlitt H, Knoppel H. Carbonyl compounds in mainstream and sidestream tobacco smoke. Present and Future of Indoor Air Quality. 1989:197-206.
- 27.Spengler JD, Dockery DW, Turner WA, Wolfson JM, Ferris BG. Long-term measurements of respirable sulfates and particles inside and outside homes. *Atmospheric Environment (1967)*. 1981;15(1):23-30.
- 28.Jones A. Asthma and domestic air quality. *Social Science & Medicine*. 1998;47(6):755-64.
- 29.Forastiere F, Corbo GM, Michelozzi P, Pistelli R, Agabiti N, Brancato G, et al. Effects of environment and passive smoking on the respiratory health of children. *International journal of epidemiology*. 1992;21(1):66-73.
- 30.Arshad SH, Matthews S, Gant J, Hide DW. Effect of allergen avoidance on development of allergic disorders in infancy. *The Lancet*. 1992;339(8808):1493-7.
- 31.National Research Council. Environmental tobacco smoke: measuring exposures and assessing health effects. National Academies Press; 1986.
- 32.Janerich DT, Thompson WD, Varela LR, Greenwald P, Chorost S, Tucci C, et al. Lung cancer and exposure to tobacco smoke in the household. *New England Journal of Medicine*. 1990;323(10):632-6.
- 33.Formaldehyde. <https://www.epa.gov/formaldehyde>. Accessed 19 March 2019.
- 34.Indoor Air Quality (IAQ). <https://www.epa.gov/indoor-air-quality-iaq>. Accessed 19 March 2019.
- 35.Wallace LA. Human exposure to volatile organic pollutants: implications for indoor air studies. *Annual Review of Energy and the Environment*. 2001;26(1):269-301.
- 36.Shima M, Adachi M. Effect of outdoor and indoor nitrogen dioxide on respiratory symptoms in schoolchildren. 2000;29(5):862-70.
- 37.Jones AP. Indoor air quality and health. *Atmospheric Environment*. 1999;33(28):4535-64.
- 38.McDonald JC. An epidemiological view of asbestos in buildings. *Toxicology and Industrial Health*. 1991;7(5-6):187-93.
- 39.Ohar J, Sterling DA, Bleecker E, Donohue J. Changing patterns in asbestos-induced lung disease. *Chest*. 2004;125(2):744-53.
- 40.United Nations Scientific Committee on the Effects of Atomic Radiation, Sources and Effects of Ionizing Radiation. New York, NY United Nations; 2006.
- 41.World Health Organization WHO Guidelines for indoor air quality: selected pollutants, 2010. http://www.euro.who.int/__data/assets/pdf_file/0009/128169/e94535.pdf. Accessed 15 December 2018.
- 42.Hussain SP, Kennedy CH, Amstad P, Lui H, Lechner JF, Harris CC. Radon and lung carcinogenesis: mutability of p53 codons 249 and 250 to 238 Pu alpha-particles in human bronchial epithelial cells. *Carcinogenesis*. 1997;18(1):121-5.
- 43.Weaver DA, Hei TK, Hukku B, McRaven JA, Willey JC. Cytogenetic and molecular genetic analysis of tumorigenic human bronchial epithelial cells induced by radon alpha particles. *Carcinogenesis*. 1997;18(6):1251-7.
- 44.Collie CG, Strong JC, Humpherys JA, Timpson N, Baker ST, Elderred T, et al. Carcinogenicity of radon/radon decay product inhalation in rats—effect of dose, dose rate and unattached fraction. *International Journal of Radiation Biology*. 2005;81(9):631-47.
- 45.National Research Council, Committee to Assess Health Risks from Exposure to Low Levels of Ionizing Radiation. Health risks from exposure to low levels of ionizing radiation: BEIR VII phase 2. National Academies Press; 2006.
- 46.Household air pollution and health. <https://www.who.int/news-room/fact-sheets/detail/household-air-pollution-and-health>. Accessed 7 December 2018.

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