buildings

MDPI

Review

Effects of Indoor Air Quality on Human Physiological Impact: A Review

Tong Nie [1], Guofu Zhang [2,*], Yinan Sun [1], Wenhao Wang [1], Tianai Wang [1] and Haoyan Duan [1]

[1] College of Architecture and Urban Planning, Qingdao University of Technology, Qingdao 266033, China; nietong@qut.edu.cn (T.N.); s_yinan1999@163.com (Y.S.); wangmunho@163.com (W.W.); wta19980818@163.com (T.W.); draymondd@163.com (H.D.)

[2] Weifang Product Quality Inspection Research Institute (Weifang Fiber Inspection Institute), Weifang 261036, China

* Correspondence: weifenghexu@163.com

Abstract: As urbanization accelerates, indoor air quality has emerged as a critical determinant of population health. To systematically evaluate the relationship between indoor air quality (IAQ) and human physiological responses, we conducted a comprehensive review of 63 experimental studies retrieved from three major databases (ScienceDirect, Google Scholar, Web of Science) spanning the years 2000–2023. This systematic review synthesizes evidence from experimental studies examining the physiological impacts of indoor air contaminants, including gaseous pollutants, particulate matter (PM), and volatile organic compounds (VOCs). Through an analysis of cardiovascular biomarkers (heart rate variability, blood pressure), respiratory parameters, and neurological indicators (electroencephalogram patterns), we identify the mechanisms linking air quality degradation to impaired physiological functioning. Our findings demonstrate that optimized ventilation systems and high-efficiency particulate filtration can mitigate exposure risks, potentially enhancing cardiovascular efficiency, pulmonary capacity, and cognitive performance. The evidence further suggests that sustained improvements for indoor environments may decrease incidence rates of respiratory pathologies and neurological disorders.

Keywords: indoor air quality; physiological index; human health; indoor exposure

Received: 25 February 2025
Revised: 30 March 2025
Accepted: 8 April 2025
Published: 15 April 2025

Citation: Nie, T.; Zhang, G.; Sun, Y.; Wang, W.; Wang, T.; Duan, H. Effects of Indoor Air Quality on Human Physiological Impact: A Review. *Buildings* **2025**, *15*, 1296. https://doi.org/10.3390/buildings15081296

1. Introduction

In recent years, air pollution has placed an enormous burden on global public health [1]. Globally, fewer than 1% of low-income and middle-income countries have urban air quality that meets the thresholds recommended by the World Health Organization (WHO). Data from the most recent Global Air Quality Guidelines released by the WHO show that about 7 million people die each year globally from ambient air pollution, accounting for about 11.6% of all deaths worldwide. Nearly 3.8 million of these premature deaths are due to indoor air pollution [2]. Studies have found that pollutant levels for indoor air are sometimes even higher than for outdoor air [3,4]. Individuals who spend extended periods indoors may experience health problems due to poorer indoor air quality (IAQ) [5]. Therefore, there is an urgent need to improve IAQ.

Indoor air pollutants are heterogeneous mixtures of particulate matter (PM), gases, and liquids, including CO_2, O_3, $PM_{2.5}$, PM_{10}, and substances such as formaldehyde and benzene. Indoor air pollution triggers physiological problems in the human body caused by either a single factor or a combination of factors, involving various response mechanisms. Many experimental studies have confirmed the existence of an effect of IAQ on human mental

and psychological states, primarily in terms of subjective comfort and work efficiency [6]. It has been found that, as the IAQ decreases, children's academic performance [7] as well as adults' performance in the office [3,7] decreases, along with an increase in fatigue. More than 20% of the exposed population experiences exacerbated symptoms of sick building syndrome (SBS). Poor IAQ negatively impacts the human psyche and can lead to irreversible physiological health problems, especially in the cardiovascular and respiratory systems [8]. Many studies have found that elderly, obese, or sick individuals are more susceptible to the effects of air pollutants than healthy people [9–13].

This review systematically evaluates five cardiorespiratory and neurological biomarkers: arterial blood pressure, heart rate (HR), heart rate variability (HRV), forced vital capacity (FVC), and electroencephalogram (EEG). These parameters quantitatively assess autonomic regulation, pulmonary function, and cortical activation patterns in response to IAQ fluctuations, providing the multisystem physiological profiling of environmental exposure impacts. Numerous studies have shown that, in terms of cardiovascular indicators, elevated levels of pollutants, such as CO_2, $PM_{2.5}$, and VOCs for indoor air, lead to an increased HR [8,14–16], blood pressure [17–19], and a decrease in the HRV index [20,21]. Respiratory studies demonstrate dose-dependent CO_2 effects, with hypercapnic exposure reducing forced vital capacity (FVC) [22], and elevating minute ventilation rates [23]. Neurophysiological investigations reveal CO_2-mediated cortical oscillation alterations, specifically α-band (8–13 Hz) suppression with concomitant β-band (14–30 Hz) and θ-band (4–7 Hz) amplification [24,25]. Current reviews exhibit limited scope in addressing IAQ biomarker interactions, predominantly focusing on singular pollutants like formaldehyde and CO_2 [26,27]. Therefore, this paper provides a comprehensive and objective review of the relevant literature, summarizes the effects of indoor air quality on human physiological indicators, and answers the following questions:

1. How do distinct categories of indoor air pollutants (e.g., gaseous pollutants, particulate matter, VOCs) differentially impact cardiovascular, respiratory, and neurological physiological indicators?
2. What synergistic effects emerge from combined exposure to multiple pollutants, and how do these interactions modulate health risks?
3. To what extent can current intervention strategies (e.g., ventilation optimization, air filtration) mitigate pollutant-induced physiological disturbances across diverse populations?

It also identifies research trends and their status, outlines optimization strategies, and provides an outlook for future research.

2. Materials and Methods

To investigate recent developments in the IAQ's physiological impacts and assess pollutant-exposure relationships, we systematically reviewed the literature from three multidisciplinary databases (ScienceDirect, Google Scholar, Web of Science). This methodology enables comprehensive tracking of emerging trends while establishing mechanistic links between indoor air contaminants and measurable physiological responses. Searching terms included IAQ or air pollutants (e.g., "carbon dioxide", "particulate matter", "ozone", or "volatile organic compounds") and physiological indicators (e.g., "blood pressure", "electroencephalogram", "heart rate variability", or "oximetry"). The keywords used in this study are shown in Table 1. The inclusion criteria for this study are as follows: (1) only studies with authentic or quasi-experimental designs were reviewed in this study; (2) studies on the effects of inhalation of indoor air pollutants on human physiological indices were excluded, with a focus on individual-level, maskless, IAQ intervention studies; and (3) experimental studies in which subjects engaged in physical activity were excluded.

The testing state was dominated by a sedentary state to ensure the smooth breathing of the subjects. This study systematically reviewed the nearly 20 years of research literature on IAQ, with 63 articles retained for review according to inclusion and exclusion criteria. Figure 1 illustrates the literature on the effects of IAQ on human physiological indices and the number and type of publications per year since 2000, with the majority of the studies occurring after 2010 (N = 43). The search process is shown in Figure 2.

Table 1. Keywords used in this study.

Category	Keywords
Pollutants	"Indoor air quality", "carbon dioxide", "particulate matter", "ozone", or "volatile organic compounds"
Physiological Indicators	"Blood pressure", "electroencephalogram", "heart rate variability", or "oximetry"
Exposure Scenarios	"Controlled experiment", "human subjects", "residential buildings", or "office"

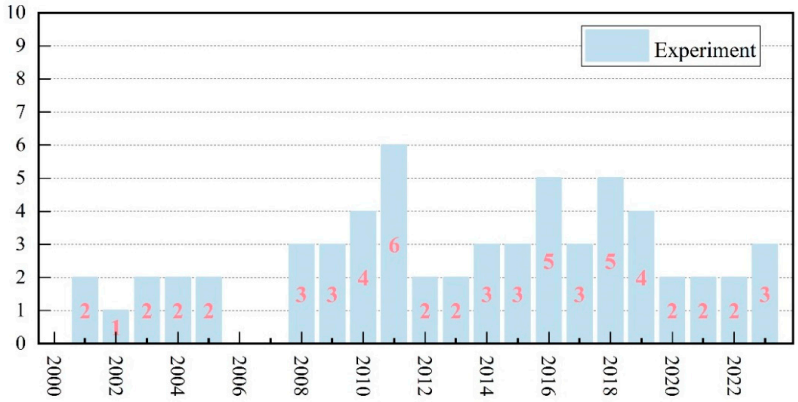

Figure 1. Number and typology of published papers on IAQ.

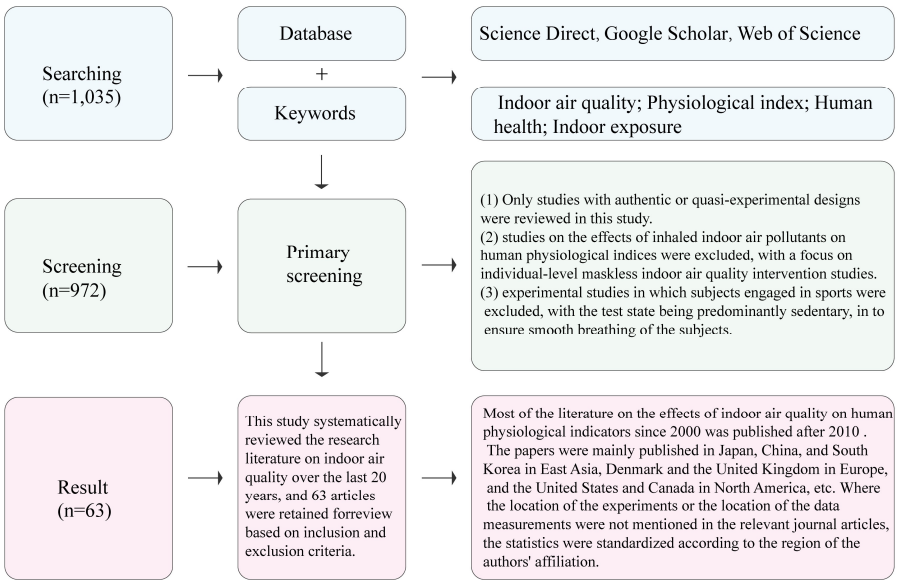

Figure 2. Search process.

The journals to which the papers included in the review belonged were analyzed and the results are shown in Table 2. The papers were primarily published in Japan, China, and South Korea, in East Asia; Denmark and the United Kingdom, in Europe, as well as

the United States and Canada, in North America. The cited studies are primarily from these seven countries for to the following reasons: East Asia's industrialization drives air pollution research, with Japan/South Korea excelling in IAQ tech, and China prioritizing smog control. In Europe, Denmark leads in green building standards, while the UK emphasizes pollution epidemiology. North America leverages interdisciplinary approaches and diverse demographics. Shared drivers include strong funding, policy–science synergy, and global academic networks. The relevant journal articles that did not involve the experimental locations or data measurement locations were standardized according to the regions to which the authors belonged.

Table 2. Journals belonging to the 63 papers included in the review.

Journal Title	Number of Citations
Building and Environment	12
Environmental Health Perspectives	11
Indoor Air	8
International Journal of Environmental Research and Public Health	4
Science of The Total Environment	3
Atmospheric Environment	3
Environment International	3
Environmental Research	2
Other (1 each of 21 journals)	21

3. Effects of Gaseous Pollutants on Human Physiological Indicators

3.1. Effect of CO_2 Content on Human Physiological Indicators

Carbon dioxide is one of the more common gaseous pollutants affecting IAQ and has a significant effect on human physiological indicators. Table A1 shows some of the studies on the effects of CO_2 on human physiological indicators. Elevated CO_2 levels influence cardiovascular functioning through blood pressure regulation, heart rate (HR), and heart rate variability (HRV). Research documents dose-dependent SBP reductions (4.8–7.3 mmHg/1000 ppm CO_2 decrease) [28], concurrent with improved IAQ assessments [29], suggesting systemic cardiovascular–environment interactions. The diastolic blood pressure (DBP) of the human body significantly increased after the CO_2 concentration reached 3000 ppm [19] or 5000 ppm [23] when subjects completed the task test. Regarding HR, Zhang et al. [30] found that HR remained unchanged when subjects completed the task test at a CO_2 concentration of 5000 ppm by adding CO_2. Several studies have documented modest HR reductions occurring at specific CO_2 concentration thresholds [19,23]. Emerging evidence demonstrates a dose-dependent elevation in HR under elevated CO_2 exposure. Controlled experiments by Chen and Hsiao [16] revealed a 7.2% HR increase in hypercapnic conditions (>1000 ppm) compared to normocapnic environments (<800 ppm), while Snow et al. [14] documented acute 2.4 bpm HR surges during rapid CO_2 escalation (830–2700 ppm). This compensatory mechanism enhances cardiac output to accelerate pulmonary perfusion, optimizing gas exchange efficiency for blood oxygen saturation maintenance. The experimental evidence demonstrates bidirectional cardiovascular responses to CO_2 exposure, with paradoxical heart rate modulation observed across concentration gradients. Elevated CO_2 levels consistently compromise HRV, indicating autonomic nervous system dysregulation. Kajtár and Herczeg [23] documented dose-dependent autonomic responses, with mid-frequency (MF) power and MF/HF ratio reductions ($\leq$4000 ppm CO_2) coinciding with high-frequency (HF) power elevation at 5000 ppm, suggesting coordinated sympathetic activation with parasympathetic withdrawal. These patterns reflect impaired autonomic regulation that elevates cardiovascular morbidity risks [8,13,31,32]. Conversely, investigations under acute hypercapnic exposure (unventilated environments) revealed

paradoxical increases in time-domain HRV indices—SDNN [33] and pNN50 [25]. This paradoxical response potentially reflects compensatory autonomic modulation [34] to optimize metabolic efficiency during CO_2 challenges.

High indoor CO_2 concentrations can negatively affect the human respiratory system. Mechanistically, an increase in CO_2 concentration results in insufficient alveolar ventilation, leading to CO_2 retention in the human body. The blood's CO_2 concentration increases, and SpO_2 decreases accordingly [16]. This situation elevates the partial pressure of CO_2 (PCO_2) delivered to the lungs, resulting in hypercapnia. Hypercapnia falls under the classification of type II respiratory failure. Additionally, an increased CO_2 concentration causes a reduction in forced vital capacity (FVC) [22] and an elevation in respiratory volume [23] among subjects. Notably, respiratory rate [23,26,35] and end-tidal CO_2 ($ETCO_2$) [22] remain statistically unaffected under acute hypercapnic conditions.

Increased CO_2 concentrations affect human brain activity. It was found that subjects exposed to low levels of CO_2 concentration (1244 ± 70 ppm) exhibited effects on EEG signals when completing a task test. Both alpha and theta wave relative power increased, indicating decreased attention and increased drowsiness, respectively [35]. Subjects exposed to high levels of CO_2 concentration (5087 ± 318 ppm) demonstrated an increase in beta wave relative power [25]. Individuals exposed to ultra-high CO_2 concentrations (40,000 ppm) exhibited a decrease in alpha waves and an increase in theta waves [24]. However, this finding contrasts with the conclusion of Shan et al. [35], who observed an increase in the relative power of theta waves, as mentioned above. This difference may be attributed to variations in experimental procedures, both with and without the task test setup, leading to differing levels of psychological stress among subjects, which consequently affects the EEG signal. Furthermore, individuals with inadequate sleep are more susceptible to the effects of CO_2 in an enclosed space [14]. The correlation between EEG and CO_2 concentration was notably linked to the hours of sleep the subjects had the previous night.

In addition, CO_2 concentrations have an effect on other physiological indicators in the human body. For example, exposure to medium-to-high CO_2 concentrations of 1000 ppm, 3000 ppm, and 5000 ppm increased the concentration of α-amylase in human saliva [19,30]. However, no significant changes in salivary cortisol were observed when exposed to high CO_2 concentrations of 5000 ppm [30]. Chen and Hsiao [16] noted that facial temperature was positively correlated with CO_2 concentration. Zhang et al. [25] concluded no significant changes in urinary pH after subjects slept at high CO_2 levels. All the above experiments were conducted under thermo-neutral conditions. However, under high-temperature conditions with increasing CO_2 concentration, there was no substantial change in physiological indices, such as tympanic membrane temperature of the right ear, skin temperature, heart rate (HR), blood pressure (BP), arterial oxygen saturation, etc. [36]. Kang et al. [37] studied the effects of differences in carbon dioxide concentrations on sleep quality. The study found that sleep quality was significantly reduced at ventilation rates of 1000 ppm and 1300 ppm CO_2 compared to ventilation with an average CO_2 concentration of 750 ppm [37].

In summary, elevated CO_2 exposure adversely impacts multiple physiological systems. Cardiovascular effects include increased blood pressure, reduced HRV, elevated HR, and heightened cardiovascular disease risk. Respiratory consequences involve increased respiratory volume, decreased FVC, and hypercapnia, disrupting acid-base balance and cellular metabolism [27]. Neurophysiological studies report altered EEG patterns with reduced alpha waves and increased beta/theta wave activity. Current guidelines recommend maintaining indoor $CO_2 \leq 1000$ ppm for acceptable IAQ [38]. This is in line with the results of Kim et al. that indoor CO_2 levels should be maintained below 1000 ppm to safeguard human physiological health [28]. However, recent ASHRAE guidelines emphasize CO_2

as an indicator of ventilation effectiveness rather than a direct health-based limit [39]. As Persily notes, the previous 1000 ppm benchmark was often misinterpreted as a safety threshold, whereas contemporary standards prioritize context-specific ventilation rates [39]. The main summary of the study is shown in Figure 3.

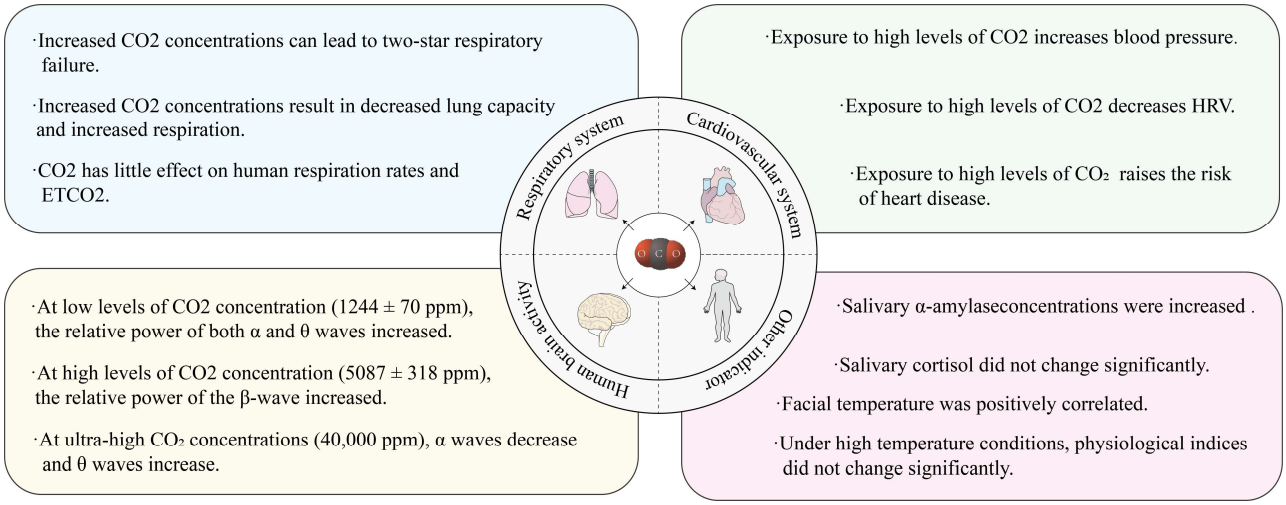

Figure 3. Effect of CO_2 content on human physiological indicators.

3.2. Effect of O_3 Content on Human Physiological Indicators

Table A2 shows some of the studies on the effects of O_3 on human physiological indicators. Current research consensus standardizes indoor ozone exposure at 120 ppb, demonstrating the null effects on cardiovascular parameters (SBP, MAP, HR) [40] and vascular endothelial markers (FMD, NMD) [41]. Hoffmann et al. [42] quantified marginal blood pressure reductions (SBP: −4.0%, DBP: −2.0%, MAP: −2.8%) per IQR increase in ozone exposure, though statistically non-significant. Contrasting evidence from Urch et al. [43] identified significant brachial artery diameter constriction (−0.09 mm BAD) at this exposure level [41], revealing potential exposure–response divergences across vascular metrics.

Environmental studies demonstrate synergistic interactions between ozone (O_3) and $PM_{2.5}$ through atmospheric multipollutant mechanisms [44–46]. For example, in two randomized double-blind crossover trials, Brook et al. [46] demonstrated that DBP increased by 2.9 mmHg under $PM_{2.5}$-only exposure and 3.6 mmHg during combined $PM_{2.5}$–O_3 exposure. This effect might be attributed to O_3 reducing vagal tone, subsequently leading to autonomic nervous system changes. In the crossover design experiment conducted by Fakhri et al. [44], which included ten mild asthmatics, it was observed that DBP increased by 2.0 mmHg, and the SDNN intervals increased in the subjects following the combined effect of $PM_{2.5}$ and O_3. Notably, no heterogeneity in asthma status was identified.

In summary, a single O_3 concentration does not significantly affect cardiovascular function. However, a more substantial synergistic effect may be induced when older groups or vulnerable individuals with cardiovascular and respiratory diseases are exposed to both $PM_{2.5}$ and O_3. These individual differences need to be addressed in future studies.

3.3. Effects of Other Gaseous Pollutant Contents on Human Physiological Indicators

Beyond CO_2 and O_3, indoor air quality analysis must consider carbon monoxide (CO) and oxygen (O_2) dynamics. Ambient CO concentrations demonstrate null cardiovascular effects, with preserved heart rate stability during controlled exposures [47]. Covariance analysis reveals no HRV–CO correlations at subthreshold concentrations [48], though elevated CO (>2.7 ppm) induces autonomic modulation evidenced by increased

r-MSSD metrics [47]. Regarding oxygen homeostasis, Wang et al. [49] observed negligible EEG signal variation in hypercapnic subjects under hyperoxic (150 mmHg) and hypoxic (50 mmHg) challenges, suggesting neural adaptation mechanisms. In summary, fewer studies exist on the effects of gaseous pollutants other than CO_2 and O_3 for indoor air on human physiological indices. Only a few studies have focused on CO and O_2, and they do not significantly affect human physiological indices, such as HR and EEG, at low concentration levels.

4. Effects of Particulate Matter on Human Physiological Indicators

Indoor air contains a significant amount of atomized PM with individual particle sizes ranging from a few nanometers to 10 microns. This PM primarily originates from direct emissions from various indoor sources, but can also result from the infiltration of PM from outdoor air. In this paper, the primary PM studied for indoor air is dominated by $PM_{2.5}$, while also encompassing submicron particles, PM_1, PM_5, PM_{10}, and so forth. The main summary of the study is shown in Figure 3.

4.1. Effects of $PM_{2.5}$ on Human Physiological Indicators

Table A3 shows some of the studies on the effects of $PM_{2.5}$ on human physiological indicators. It was found that short-term indoor exposure to $PM_{2.5}$ decreased the frequency domain indexes HF [21,45,50] and LF [50] of HRV and SDNN of time domain indexes [45,50,51]. Additionally, emerging evidence demonstrates CO_2-induced cardiovascular activation, triggering significant elevations in heart rate [8,17] and blood pressure [18,40,52,53]. These effects are not conducive to the relaxation of the autonomic nervous system. Moreover, $PM_{2.5}$ exposure heightens the risk of infection or inflammation in the body, as evidenced by elevated malondialdehyde concentrations in plasma [54], and indirectly through increased ultrasensitive C-reactive protein and 8-hydroxydeoxyguanosine levels in the blood [53].

Currently, most studies on the effects of $PM_{2.5}$ for indoor air on human physiological indicators primarily focus on the human cardiovascular system. It has been found that exposure to indoor $PM_{2.5}$ in healthy populations leads to an increase in blood pressure [18]. Moreover, for each increase in the interquartile range (IQR) of $PM_{2.5}$, the HR of subjects increases by an average of 4–6 bpm [8]. Studies investigating the effects on specific sensitive groups, such as the elderly, overweight individuals, and mildly or critically ill patients with heart or lung diseases, have been intensified. The morbidity and mortality of cardiovascular diseases associated with acute $PM_{2.5}$ exposure are higher in sensitive populations than in healthy populations. Pan et al. recruited 43 patients with physician-diagnosed stable COPD and measured their 24-h HRV and HR [21]. They observed that HF decreased by only 2.01% in COPD patients with normal body weight after exposure to $PM_{2.5}$, whereas it decreased by as much as 34.85% in overweight COPD patients. The effect on parasympathetic activity associated with indoor particulate matter was more pronounced. Hoffmann et al. [42] conducted a panel study of 70 subjects with T2DM, with blood pressure measured every 2 weeks by automated oscillometric sphygmomanometer and pulse wave analysis, and hourly central site measurements of fine particulate matter, ozone, and meteorology. They noted that indoor $PM_{2.5}$ raised arterial blood pressure in patients with type 2 diabetes. Regarding age, the elderly are more sensitive to $PM_{2.5}$ than the young [44,55]. Jia et al. [56] found that a five-minute exposure to $PM_{2.5}$ increased HF and LF by 1.30% and 1.34%, respectively, in healthy elderly subjects. Baumgartner et al. [18] reported that a 1-log $\mu g/m^3$ increase in $PM_{2.5}$ concentration elevated blood pressure in adult females, with middle-aged and older females above 50 years of age showing a stronger correlation with increased blood pressure. Furthermore, pronounced sex differences have been noted in changes

in hematological parameters and protein oxidation products induced by $PM_{2.5}$ exposure. Physiological responses generally appear weaker in men than in women. Sørensen et al. [54] discovered that, for every 10 $\mu g/m^3$ increase in indoor $PM_{2.5}$ concentrations, plasma proteins increased by an average of 1.6% in both sexes, and malondialdehyde concentrations increased by 3.7% in females. However, there was no significant change observed for males.

In recent years, with the increasing awareness of health protection, people are increasingly focusing on purifying indoor air from $PM_{2.5}$. This is typically achieved through air conditioning filtration [48]. Studies have demonstrated that, when individuals are exposed to unfiltered indoor air, the SBP, DBP, and HR of student subjects increase by 4.11 mmHg, 2.78 mmHg, and 3.11 bpm per IQR, respectively, averaged over 4 h [57]. After the filter is activated, the subjects' BP and HR do not change significantly with rising $PM_{2.5}$ levels [57]. However, microvascular functioning experiences a significant enhancement after the second day [58]. Zhou et al. [59] assessed the relationship between short-term exposure to indoor $PM_{2.5}$ in the workplace and cognitive ability in adults. Even during short-term indoor exposure, elevated concentrations of $PM_{2.5}$ can have adverse effects on cognitive ability [59]. Furthermore, air conditioning filtration does not improve lung function, systemic inflammation, nor does it significantly reduce monocyte activation or lung cell damage in the elderly [58], which suggests that the ameliorative effect of air conditioning filtration is limited. The effects of other filtration methods exhibit similar variability.

Liu et al. [60] utilized active filtration (with activated carbon filters) in their experiments. Their study demonstrated that each 10 $\mu g/m^3$ increment in the 8-h moving average $PM_{2.5}$ concentration during sham filtration (without filter) correlated with a statistically significant 1.34% reduction in SDNN. By contrast, active filtration attenuated this effect, resulting in a non-significant 0.81% decrease in SDNN. Additionally, Liu et al. [61] used an ionization air purifier to reduce $PM_{2.5}$ levels indoors, which enhanced the six major pathways of HRV in children (1. galactose metabolism is upregulated, 2. downregulation of purine metabolism, 3. upregulation of vitamin B metabolism, 4. lysine degradation upregulation, 5. improved β-oxidation of fatty acids, And 6. upregulation of progesterone biosynthesis), leading to increased energy production and improved anti-inflammatory capacity. However, the NAI produced worsened effects on the five major pathways of HRV (1. UTP synthesis down-regulation, 2. TCA cycle down, 3. dysregulation of tyrosine and phenylalanine metabolism, 4. reduced antioxidant scavenging activity, And 5. histidine degradation increased), decreased energy production, and inhibited antioxidant capacity.

Moreover, studies have been conducted using indoor plants to reduce and purify $PM_{2.5}$ for indoor air. Chen et al. [15] conducted six 24-h home visits with each elderly subject and measured $PM_{2.5}$, TVOCs, HR, and BP consecutively at each home visit. They discovered that placing plants indoors resulted in a reduction of about 9.1 $\mu g/m^3$ of $PM_{2.5}$ in the air, along with corresponding reductions in SBP (without plants: 128.4 mmHg vs. with plants: 114.1 mmHg) and HR (without plants: 76.4 bpm vs. with plants: 70.3 bpm) [15]. However, the precise mechanisms underlying the observed reductions in SBP and HR remain unresolved. These effects may arise from BVOC emissions by indoor plants, their psychophysiological stress-reducing properties, or interactions with $PM_{2.5}$ attenuation dynamics. Future investigations should delineate the relative contributions of phytochemical, perceptual, and particulate-modulating pathways to indoor plant-mediated physiological adaptations.

In summary, $PM_{2.5}$ for indoor air causes subjects to experience increased sympathetic activation, decreased parasympathetic tone, or both, which reduces the level of autonomic control over the human heart [58,62], resulting in acute health damage to the cardiovascular system [50]. Notably, studies have shown that the degree of harm caused by $PM_{2.5}$ is

likely to be correlated with the carbon level in $PM_{2.5}$ [40], leading to arterial constriction and subsequently increasing blood pressure [43]. Therefore, in future studies, further consideration should be given to the physiological effects of the elemental composition and levels of particulate matter. The main summary of the study is shown in Figure 4.

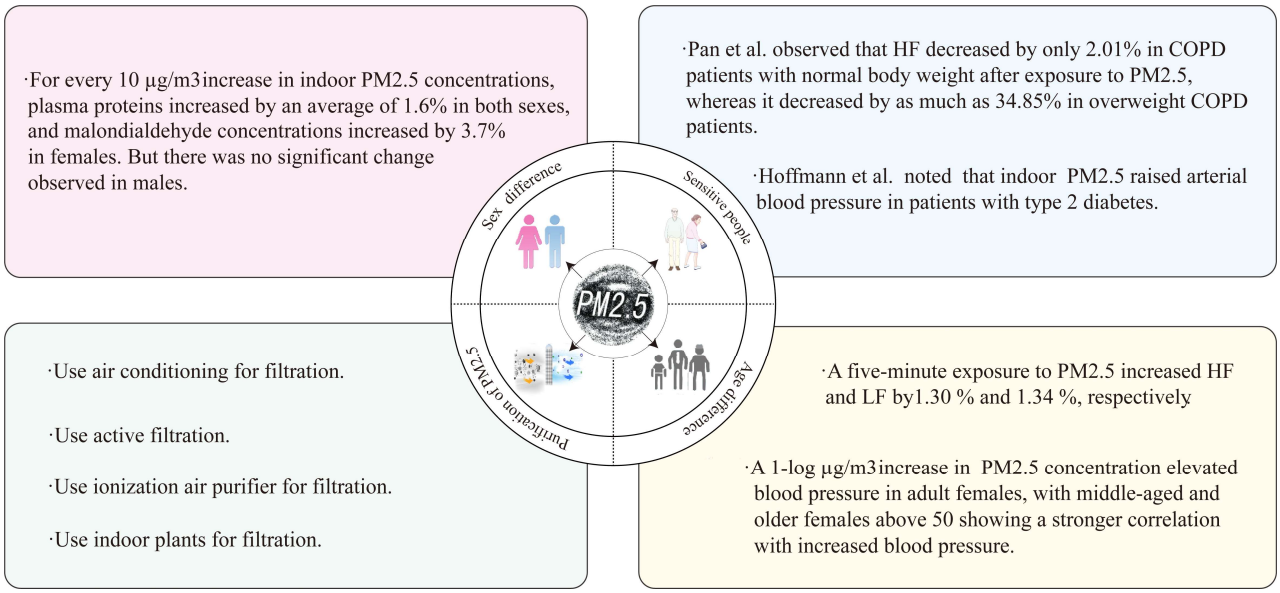

Figure 4. Effect of $PM_{2.5}$ on human physiological indicators.

4.2. Effects of Other Diameters of PM on Human Physiological Indicators

Current research on the physiological effects of particulate matter beyond $PM_{2.5}$ remains focused on cardiovascular outcomes, including blood pressure elevation [8] and reduced HRV indices (SDNN, r-MSSD) [48]. These alterations are associated with autonomic dysfunction, contributing to cardiac conditions (e.g., myocardial infarction, hypertension) and non-cardiac disorders (e.g., diabetes mellitus, COPD). Smaller particulate fractions exhibit stronger associations with HRV suppression and heart rate elevation [21,50], particularly posing risks for myocardial ischemia or arrhythmia in high-risk cardiovascular populations [63]. Chuang et al. [62] observed that $PM_{0.3–1.0}$ exposure specifically reduced HRV in patients with hypertension and coronary heart disease, whereas larger particulates ($PM_{0.3–1.0}$) showed no significant effects.

In summary, there is a correlation between particle size and physiological indicators, such as HRV. The pathophysiological impact of particulate matter intensifies inversely with aerodynamic diameter, demonstrating heightened cardiopulmonary toxicity at smaller particle sizes. Consequently, indoor air purification systems should prioritize size-selective filtration strategies targeting ultrafine particulate fractions to optimize particulate removal efficacy and mitigate cardiopulmonary health risks.

5. Effects of VOCs on Human Physiological Indicators

Volatile Organic Compounds represent a class of ubiquitous chemical contaminants that significantly compromise IAQ. This group encompasses formaldehyde, benzene, toluene, xylene, ethylbenzene, styrene, trichloroethylene, and chlorinated derivatives including p-dichlorobenzene and perchloroethylene—all classified as established neurotoxicants. Current research indicates a knowledge gap regarding their systematic toxicological classification, with limited experimental data elucidating exposure–response relationships. Notably, formaldehyde has emerged as a prototypical VOC in IAQ research, serving as the primary subject of focused investigation in controlled IAQ studies. Table A4 shows some of

the studies on the effects of VOCs on human physiological indicators. The main summary of the study is shown in Figure 5.

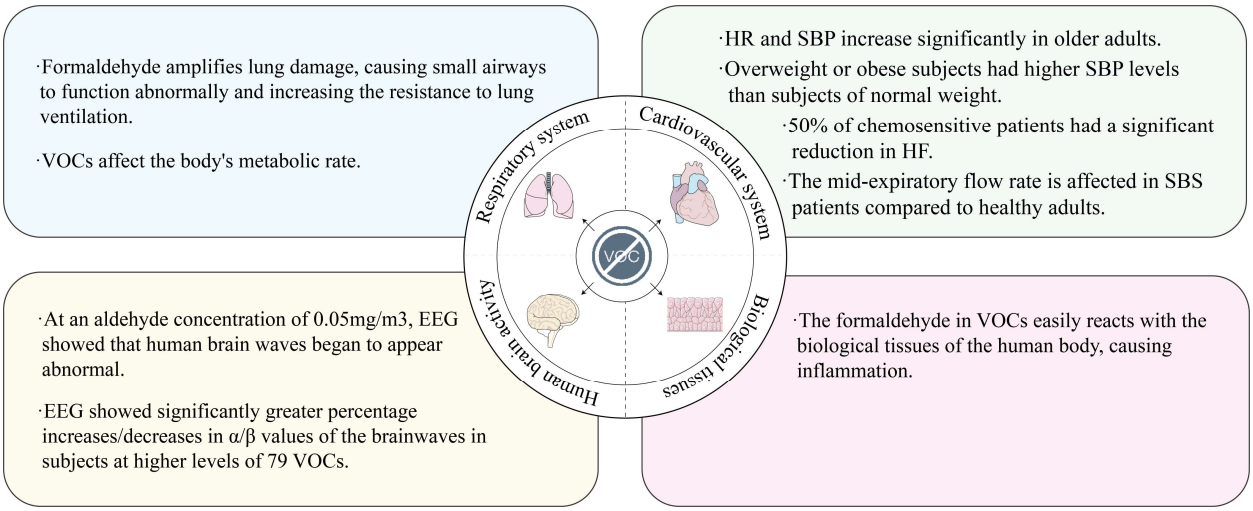

Figure 5. Effect of VOCs on human physiological indicators.

VOCs can affect the human cardiovascular system [53], especially for the physiological health of individuals with poorer or compromised health. Studies have shown that HR and SBP significantly increase in the elderly as the concentration of VOCs for indoor air rises [15]. Overweight or obese subjects exhibit higher SBP levels compared to normal-weight subjects [17]. Fifty percent of chemically sensitized patients exhibit significantly lower HF and suppressed parasympathetic activity in humans [20]. The mid-expiratory flow rate (FEF50), an indicator of human lung function, is affected in SBS patients compared to healthy adults, indicating reduced respiratory function. Skin measurements of sebum and perspiration are only responsive in SBS patients [64]. Changes in the concentration of VOCs themselves can have varying effects on the same physiological indicators. Mizukoshi et al. [65] measured seven healthy subjects for 24 h under usual daily living conditions. They found that, when the VOCs, primarily toluene and xylene, increased, the HF of HRV in subjects was negatively correlated with them and positively correlated with LF/HF [65]. The opposite result was found when the VOCs decreased.

VOCs affect human brain activity. Chen et al. [66] found that, at a formaldehyde concentration of 0.05 mg/m^3, an EEG showed that human brain waves began to show abnormalities. Nakayama et al. [67] found that an EEG showed a significantly greater percentage of increases/decreases in α/β values of the brainwaves in subjects at higher levels of 79 VOCs. However, subjects did not experience changes in cerebral blood flow [68], but they exerted more effort when performing tasks at maximum speed. Subjectively, they reported increased fatigue during exposure.

VOCs can affect the human respiratory system. Formaldehyde amplifies lung damage, causing small airways to function abnormally and increasing the resistance to lung ventilation [58,69,70], which is not conducive to physiological relaxation. VOCs also affect the metabolic rate of the human body in the working conditions of office workers. For example, Bakó-Biró et al. [71] found a significant decrease in the CO_2 production rate of about 5% for subjects in the office environment, which could be caused by a change in breathing pattern (shallow breathing) or a slower work rate, leading to a decrease in metabolic rate. However, Wargocki et al. [72] reported that subjects exposed to 41 VOCs, mainly acetone and acetic acid, had a metabolic rate in the office state of 1.3, much higher than the relaxed sedentary normal value of 1.2. This is due to the unconscious tension of the muscles during mental labor.

In addition, formaldehyde in VOCs readily reacts with the biological tissues of the body, especially the mucous tissues of the respiratory tract and eyes [73], causing the potential for inflammation [64]. Formaldehyde-exposed women have an increased risk of spontaneous abortion and adverse pregnancy outcomes, including chromosome and DNA damage (genotoxicity), oxidative stress, altered levels and/or function of biological enzymes, hormones and proteins, and DNA methylation [74]. Most importantly, exposure to airborne formaldehyde dramatically increases non-carcinogenic health risks and carcinogenic risks in various human systems [26]. Kim et al. [75] noted that chronic exposure to formaldehyde reduces the number of human leukocytes and may lower platelet and hemoglobin counts [64,69], which can lead to cancers of the lymphopoietic system in humans [76], and can increase the risk of acute myeloid leukemia (AML) and other blood cancers [77]. Shim et al. [78] objectively measured indoor concentrations of aldehydes and VOCs in 128 stores in 10 underground commercial areas in Korea. The results showed that, on the environmental side, eye irritation symptoms were related to n-butanol, n-heptane, and xylene; respiratory symptoms were related to n-heptane; and systemic symptoms were related to benzene, n-heptane, and decanal concentrations [78].

In summary, exposure to VOCs can have a more serious negative impact on human physiological health. This effect is even more severe than exposure to some gaseous pollutants or particulate matter. However, it was found that the concentration of VOCs for indoor air could be effectively reduced by opening windows, ventilation, or air conditioning filtration. Therefore, in future research, the consideration of purification methods for VOCs for indoor air should be strengthened to provide methods with universal applicability to enhance the comfort and health of building occupants.

6. Effects of Other IAQ Factors on Human Physiological Indicators

In addition to indoor air pollutants, such as gaseous pollutants, particulate matter, and VOCs, some studies have investigated the effects of environmental tobacco smoke (ETS) and carbon black (BC) on human physiological health. ETS is a class of substances produced by people smoking tobacco products indoors, containing more than 7000 compounds including nicotine and tar. By measuring vascular tone and function and blood pressure in 65 nonsmoking subjects, Brook et al. [52] found that, for every 10 $\mu g/m^3$ increase in the average daily concentration of ETS, the SBP of ETS-exposed subjects increased by 3.94 mmHg and 6.57 mmHg on lagged days 1 and 2 after exposure, respectively. Pope et al. [69] noted that ETS exposure was negatively correlated with all HRV indices. Among them, SDNN decreased by about 12%, independent of mean HR and oxygen saturation. The effects of BC were similar, even more pronounced for overweight individuals [50], and the changes in parasympathetic activity were more noticeable [21]. For every 1 $\mu g/m^3$ increase in BC concentration for indoor air in the experiments by Liu et al. [60], SBP was not significantly reduced by 1.09% after active filtration, and the SBP was not significantly reduced after control for pseudo filtration. The SBP after control for sham filtration was significantly increased by 2.41%. This indicates that activated charcoal filtration is very effective in the absorption of BC, thus significantly reducing the SBP of the subjects and inducing physiological relaxation. Taken together, both ETS and BC are capable of affecting the autonomic function of the human heart, which in severe cases can lead to acute health damage to the cardiovascular system.

7. Conclusions

This comprehensive review examines the physiological impacts of indoor air pollutants on human health through systematic analysis of IAQ research. We evaluate current evidence linking IAQ variations to measurable changes in cardiovascular and respiratory

biomarkers, focusing on blood pressure, HR, HRV, and EEG as critical physiological indicators. Epidemiological data confirm that compromised IAQ under low ventilation conditions adversely affects systemic physiological regulation. Dose-dependent IAQ alterations correlate linearly with cardiovascular stress, respiratory compensation, and autonomic dysfunction. Also, in response to the article, the following conclusions were drawn:

1. Gaseous pollutants for indoor air have a wide range of effects on human physiological indicators. The effects of CO_2 on the human body cover indicators of the cardiovascular system, the respiratory system, and brain activity, while studies of O_3 have focused on the effects on indicators of the human cardiovascular system. Since elevated aerosol concentrations for indoor air are often accompanied by the accumulation of other types of indoor pollutants, the simultaneous exposure of subjects to O_3 and $PM_{2.5}$ may cause synergistic effects in terms of changes in physiological indicators. Therefore, potential interactions between multiple indoor air pollutant exposures deserve further exploration in future studies.
2. PM for indoor environments predominantly impacts the cardiovascular system, with adverse effects intensifying as particle size decreases and carbon content increases. Empirical evidence indicates that air filtration systems and ionization technologies can mitigate these effects by improving cardiovascular and respiratory biomarker profiles in settings with suboptimal IAQ. Future research should prioritize evaluating the efficacy of particulate purification devices across PM size fractions and compositional variations, while exploring integrated applications of IoT-based monitoring and AI-driven environmental control systems for enhanced indoor air management. Improvements in the performance of purification devices, IoT, and AI should be considered as a future challenge, which will improve the IAQ and promote the physiological health of people.
3. VOCs for indoor air can also affect physiological indicators in a number of human systems. If the trend of VOCs for indoor air changes in the opposite direction, the effect on human physiological indicators may also have the opposite effect. The negative effects of VOCs on IAQ are more severe than those of other factors that affect IAQ. However, current research on indoor VOCs lacks standardized classification frameworks for specific chemical subgroups. Addressing these knowledge gaps requires large-scale controlled intervention trials employing longitudinal study designs to identify dominant VOC species and characterize their temporal concentration patterns. Such methodological refinements will enable comprehensive assessment of public health risks associated with targeted indoor air remediation strategies.

Author Contributions: Conceptualization, T.N. and G.Z.; methodology, T.N.; software, T.N.; validation, T.N., G.Z. and Y.S.; formal analysis, W.W.; investigation, T.W. and Y.S.; resources, H.D.; data curation, W.W.; writing—original draft preparation, T.N.; writing—review and editing, T.N.; visualization, T.N.; supervision, T.N.; project administration, T.N.; funding acquisition, H.D. All authors have read and agreed to the published version of the manuscript.

Funding: This study was funded by the Natural Science Foundation of Qingdao Municipality (23-2-1-96-zyyd-jch).

Data Availability Statement: The data presented in this study are available on request from the corresponding author.

Conflicts of Interest: The authors declare no conflicts of interest.

Appendix A

Table A1. The effect of CO_2 concentration for indoor air on human physiology.

Author	Participants	Ages	Range	Task	Intervention	Physiological Index	Effect
Zhang et al. (2021) [25]	Adults N = 4 (4 males)	24.20 ± 2.48	1626 ± 306 ppm, 3562 ± 259 ppm, 5087 ± 318 ppm	√	—	HRV, EEG	Decreased SDNN standard deviation and increased PNN50 for HRV (1500 ppm: 8% vs. 3500 ppm: 9.6%); Increase in β relative power of EEG.
Kim et al. (2020) [29]	Adults N = 22 (16 males, 6 females)	27.57 ± 0.09	1000–2000 ppm	—	—	Blood pressure	SBP increased (2000 ppm: 126.388 mmHg vs. 1000 ppm: 122.337 mmHg).
Zhang et al. (2017) [19]	University students N = 25 (10 males, 15 females)	23 ± 2	500 ppm, 1000 ppm, 3000 ppm	√	—	Respiration rate, HR, α-amylase, DBP	No change in respiration rate; HR reduced; significant increase in α-amylase concentration; DBP increased significantly.
Zhang et al. (2016) [30]	University students N = 10 (5 males, 5 females)	25 ± 2	500 ppm, 5000 ppm	√	—	Respiration rate, HR, α-amylase, cortisol	Respiratory rate, HR unchanged; α-amylase concentration increased; no difference in cortisol.
Snow et al. (2019) [14]	Employees or Students of the University N = 31	22.5 ± 4.8	830 ppm, 2700ppm	√	—	EEG, HR, skin temperature, respiratory rate	EEG unchanged; HR increased.
Chen and Hsiao (2014) [16]	Youngster N = 10 (6 males, 4 females)	26 ± 5	More than 800 ppm, 800–1000 ppm, More than 1000 ppm	—	—	HR, SpO_2, facial temperature	Increased HR; increased facial temperature; decreased SpO_2.
Mishra et al. (2021) [22]	Adults N = 15 (8 males, 7 females)	21~55	900 ppm, 1450ppm	√	—	Respiration rate, $ETCO_2$, FVC	Respiratory rate, $ETCO_2$ unchanged; lung volume FVC decreased.
Shan et al. (2022) [35]	University students N = 25 (15 males, 15 females)	—	1244 ± 70 ppm, 618 ± 45 ppm	√	—	EEG	The relative power of both alpha and theta waves of EEG increased.
Jin et al. (2022) [24]	Adults N = 15 (8 males, 7 females)	26.6 ± 3.4	4000 ppm, 40,000 ppm	—	—	EEG	EEG was affected.
Liu et al. (2017) [36]	University students N = 12 (6 males, 6 females)	24.8 ± 2.6	380 ppm, 3000 ppm	—	—	Right ear tympanic membrane temperature, skin temperature, heart rate, blood pressure, SpO_2, $ETCO_2$	All unchanged.
Kang et al. (2023) [37]	Adults N = 36 (16 males, 16 females)	23.7 ± 3.6	0–9999 ppm	√	√	Wrist skin temperature, blood pressure and pulse, oxygen saturation of blood, salivary biomarker	There was no significant difference.

Table A2. Effects of O_3 concentration for indoor air on human physiology.

Author	Participants	Ages	Range	Task	Interven-tion	Physiological Index	Effect
Urch et al. [40]	Healthy adults N = 23 (13 males, 10 females)	32 ± 10	121 ± 3 ppb	—	—	DBP, HR	DBP increased from 1 mmHg to 6 mmHg when exposed in combination with $PM_{2.5}$.
Brook et al. (2002) [41]	Healthy adults N = 25 (15 males, 10 females)	34.9 ± 10	120 ppb	—	—	Blood pressure, FMD, NMD, BAD	BAD (−0.09 ± 0.15 mm vs. 0.01 ± 0.18 mm) was significantly lower, and there were no significant differences in FMD (0.29 ± 4.11% vs. −0.03 ± 6.63%), NMD (3.87 ± 5.43% vs. 3.46 ± 7.92%), and blood pressure.
Hoffmann et al. (2012) [42]	Diabetes mellitus type 2 (T2DM) N = 70 (53 males, 37 females)	Mean 64.4	13.3 ppb	—	—	Blood pressure	For each additional interquartile spacing, SBP, DBP, and central mean arterial blood pressure decreased by 4.0%, 2.0%, and 2.8%, respectively.
Urch et al. (2004) [43]	Healthy adults N = 24 (14 males, 10 females)	35 ± 10	120 ppb	—	—	BAD	BAD reduced by 0.09 mm. Unable to assess.
Brook et al. (2009) [46]	Healthy adults N = 50 (19 males, 31 females)	27 ± 8	120 ppb	—	—	DBP	There was little impact on DBP.
Fakhri et al. (2009) [44]	Adults N = 50 (24 males, 26 females)	27.08 ± 7.13	113.9 ± 6.6 ppb	—	—	HRV, blood pressure, respiratory rate, HR	In synergistic exposure with O_3 DBP increased by 2 mmHg and SDNN increased.
Power et al. (2008) [45]	Asthmatic N = 5 (1 males, 4 females)	Mean 37	200 ppb	—	Filtration	HRV	Combined particle and ozone exposure reduces SDNN in asthmatics.

Table A3. Effects of $PM_{2.5}$ concentration for indoor air on human physiology.

Author	Participants	Ages	Range	Task	Interven-tion	Physiological Index	Effect
Dong et al. (2018) [50]	Older people N = 29 (29 females)	Mean 68.2	55.7 ± 55.4 μg/m^3	—	—	HRV	HRV's HF, LF, and SDNN declined.
Lu et al. (2018) [21]	COPD patients N = 43 (40 males, 3 females)	71.49 ± 6.40	58.01 ± 52.82 μg/m^3	—	—	HRV, HR	HF decreased by 34.85% in overweight patients compared to 2.01% in normal weight patients.
Power et al. (2008) [45]	Asthmatic N = 5 (1 males, 4 females)	Mean 37	313 ± 19.5 μg/m^3	—	Filtration	HRV	The SDNN standard deviation of HRV was significantly lower when exposed synergistically with O_3; there was no significant change in HRV when exposed to particles only.
Jung et al. (2016) [17]	Office staff N = 115 (83 males, 32 females)	34.2 ± 5.7	40.7 ± 29.1 μg/m^3	—	—	Blood pressure, HR	HR increased.
Rumchev et al. (2018) [8]	Adults N = 63 (28 males, 35 females)	Mean 61	18.74 μg/m^3	—	—	Blood pressure, HR	For each IQR increase in $PM_{2.5}$, heart rate increases by 4–6 bpm.
Brook et al. (2015) [52]	Adults N = 65 (50 males, 15 females)	Mean 44.6	11.6 ± 8.5 μg/m^3	—	—	Blood pressure, HR, BAD and FMD	A 10 μg/m^3 increase in $PM_{2.5}$ was associated with a 1.41 mmHg increase in SBP after 1 day.
Fakhri et al. (2009) [44]	Adults N = 50 (24 males, 26 females)	27.08 ± 7.13	121.6 ± 48.0 μg/m^3	—	—	HRV, blood pressure, respiratory rate, HR	DBP does not have a significant effect and SDNN increases when younger subjects are exposed only to $PM_{2.5}$.

Table A3. *Cont.*

Author	Participants	Ages	Range	Task	Interven-tion	Physiological Index	Effect
Jia et al. (2012) [56]	Healthy older people N = 30 (12 males, 18 females)	57.9 ± 5.4	45.58 μg/m^3	—	—	HRV	HF and LF increased by 1.30% and 1.34%, respectively.
Lin et al. (2011) [57]	Healthy students N = 60 (30 males, 30 females)	Median age 25.0	23.65 ± 12.6 μg/m^3 18.05 ± 8.45 μg/m^3	—	Filtration	Blood pressure, HR	Without filter, SBP, DBP, and HR increased by 4.11 mmHg, 2.78 mmHg, and 3.11 bpm, respectively; after filtering the air, BP, and HR did not change significantly with the rise of $PM_{2.5}$.
Karottki et al. (2013) [58]	Older people N = 48 (22 males, 26 females)	67 ± 6.5	4 μg/m^3 8 μg/m^3	—	Filtration	Blood pressure, microvascular function, pulmonary function	There was no improvement in microvascular, functional lung function, and no significant reduction in systemic inflammation, monocyte activation, or lung cell injury.
Liu et al. (2018) [60]	Healthy older people, COPD patients N = 35 (20 males, 15 females)	66.26 ± 7.71	58.24 μg/m^3 37.99 μg/m^3	—	Activated carbon filtration	Blood pressure, HRV	For every 10 μg/m^3 increase in $PM_{2.5}$, there was a significant reduction of 1.34% for sham-filtered SDNN and a non-significant reduction of 0.81% for activated carbon filtration.
Liao et al. (1999) [70]	Older people N = 26 (7 males, 19 females)	Mean 81	9.8 ± 3.7 μg/m^3	—	—	HRV	HRV decreased.
Zhou et al. (2023) [59]	Adults N = 66	18–65	0–30 μg/m^3; ±3 μg/m^3. 30–1000 μg/m^3; ± 10%	—	—	electrodermal activity, EDA and heart rate variability, HRV	Reducing significantly indoor elevated $PM_{2.5}$ levels can improve some cognitive abilities in office workers.

Table A4. Effects of VOCs concentration for indoor air on human physiology.

Author	Participants	Ages	Range	Task	Intervention	Physiological Index	Effect
Kjærgaard et al. (1991) [64]	Adults N = 35 (15 males, 20 females)	Mean 41.25	25,000 $\mu g/m^3$	√	—	PML, sebaceous sweat, mid-expiratory flow	PML increased; skin indicators regarding sebum and sweat, and mid-expiratory flow (FEF50) responded only in the SBS group.
Chuang et al. (2017) [53]	Healthy adults N = 200 (100 males, 100 females)	Mean 43.4	0.98 ± 0.56 ppm, 0.43 ± 0.21 ppm, 1.22 ± 0.81 ppm	—	Refrigeration	Blood pressure, Hs-CRP, 8-OHdG	BP, Hs-CRP, and 8-OHdG increased.
Chen et al. (2019) [15]	Older people N = 100 (50 males, 50 females)	Mean 67.3	347.5 ± 78.2 ppb, 748.4 ± 163.4 ppb	—	—	Blood pressure, HR	SBP and HR increased.
Mizukoshi et al. (2015) [20]	Chemically sensitized patients N = 8 (3 males, 5 females)	44 ± 11	306 ± 148 $\mu g/m^3$	—	—	HRV	HF was significantly negatively correlated with it.
Jung et al. (2016) [17]	Office staff N = 115 (83 males, 32 females)	34.2 ± 5.7	528.7 ± 315.4 $\mu g/m^3$	—	—	Blood pressure, HR	SBP levels were higher in overweight or obese subjects.
Mizukoshi et al. (2010) [65]	Adults N = 7 (4 males, 3 females)	32 ± 13	63–1447 $\mu g/m^3$	—	—	HRV	ΔTVOC is negatively correlated with HF and positively correlated with LF/HF.
Bakó-Biró et al. (2005) [71]	Adults N = 60 (60 females)	Not mentioned	Not mentioned	√	—	CO2 exhalation rate	The CO_2 exhalation rate was significantly reduced by approximately 5%.
Wargocki et al. (1999) [72]	Healthy adults N = 30 (30 females)	Mean 24	165 ± 50 $\mu g/m^3$, 195 ± 10 $\mu g/m^3$, 220 ± 20 $\mu g/m^3$	√	—	CO2 exhalation rate	The metabolic rate was 1.3, much higher than the relaxed sedentary normal of 1.2.
Nakayama et al. (2021) [67]	Healthy adults N = 168	20~50s	3629 $\mu g/m^3$, 55 $\mu g/m^3$	√	—	EEG	The rate of increase/decrease in α/β values was significantly higher in the group with higher levels of VOCs than in the group with lower concentrations.
Lin et al. (2013) [48]	Healthy adults N = 300 (136 males, 164 females)	Mean 43.2	77.2 ± 27.3 ppb, 68.6 ± 23.1 ppb, 52.3 ± 20.4 ppb	—	Air conditioner	HRV, Hs-CRP, 8-OHdG, plasma fibrinogen	Hs-CRP, 8-OHdG and plasma fibrinogen were elevated.
Shim et al. (2023) [78]	Underground stores N = 454	Over 20	Not mentioned	—	—	eye irritation, respiratory, general symptoms	The concentrations of n-butanol, n-heptane, and xylene were associated with eye irritation symptoms, while those of n-heptane were associated with respiratory symptoms, and those of benzene, n-heptane, and decanal were associated with general symptoms.

References

1. Matson, P.A. Environmental Challenges for the Twenty-First Century: Interacting Challenges and Integrative Solutions. *Ecol. Law Q.* **2001**, *27*, 1179.
2. Patial, S.; Nazim, M.; Khan, A.A.P.; Raizada, P.; Singh, P.; Hussain, C.M.; Asiri, A.M. Sustainable Solutions for Indoor Pollution Abatement During COVID Phase: A critical study on current technologies & challenges. *J. Hazard. Mater. Adv.* **2022**, *7*, 100097. [CrossRef]
3. Madruga, D.G. A Comprehensive Review on the Indoor Air Pollution Problem, Challenges, and Critical Viewpoints. In *Integrating IoT and AI for Indoor Air Quality Assessment*; Saini, J., Dutta, M., Marques, G., Halgamuge, M.N., Eds.; Springer International Publishing: Cham, Switzerland, 2022; pp. 9–26.
4. Rudel, R.A.; Perovich, L.J. Endocrine Disrupting Chemicals in Indoor and Outdoor Air. *Atmos. Environ.* **2009**, *43*, 170–181. [CrossRef]
5. Saini, J.; Dutta, M.; Marques, G. Chapter 6—Indoor Air Pollution: A Comprehensive Review of Public Health Challenges and Prevention Policies. In *Current Trends and Advances in Computer-Aided Intelligent Environmental Data Engineering*; Marques, G., Ighalo, J.O., Eds.; Academic Press: Cambridge, MA, USA, 2022; pp. 105–126.
6. Bako-Biro, Z.; Wargocki, P.; Weschler, C.J.; Fanger, P.O. Effects of Pollution from Personal Computers on Perceived Air Quality, SBS Symptoms and Productivity in Offices. *Indoor Air* **2004**, *14*, 178–187. [CrossRef]
7. Wargocki, P.; Wyon, D.P. Providing Better Thermal and Air Quality Conditions in School Classrooms Would Be Cost-Effective. *Build. Environ.* **2013**, *59*, 581–589. [CrossRef]
8. Rumchev, K.; Soares, M.; Zhao, Y.; Reid, C.; Huxley, R. The Association between Indoor Air Quality and Adult Blood Pressure Levels in a High-Income Setting. *Int. J. Environ. Res. Public Health* **2018**, *15*, 2026. [CrossRef]
9. Huang, W.; Zhu, T.; Pan, X.; Hu, M.; Lu, S.E.; Lin, Y.; Wang, T.; Zhang, Y.; Tang, X. Air Pollution and Autonomic and Vascular Dysfunction in Patients With Cardiovascular Disease: Interactions of Systemic Inflammation, Overweight, and Gender. *Am. J. Epidemiol.* **2012**, *176*, 117–126. [CrossRef] [PubMed]
10. Puett, R.C.; Hart, J.E.; Yanosky, J.D.; Paciorek, C.; Schwartz, J.; Suh, H.; Speizer, F.E.; Laden, F. Chronic Fine and Coarse Particulate Exposure, Mortality, and Coronary Heart Disease in the Nurses' Health Study. *Environ. Health Perspect.* **2009**, *117*, 1697–1701. [CrossRef]
11. Miller, K.A.; Siscovick, D.S.; Sheppard, L.; Shepherd, K.; Sullivan, J.H.; Anderson, G.L.; Kaufman, J.D. Long-Term Exposure to Air Pollution and Incidence of Cardiovascular Events in Women. *New Engl. J. Med.* **2007**, *356*, 447–458. [CrossRef]
12. Weichenthal, S.; Villeneuve, P.J.; Burnett, R.T.; van Donkelaar, A.; Martin, R.V.; Jones, R.R.; DellaValle, C.T.; Sandler, D.P.; Ward, M.H.; Hoppin, J.A. Long-Term Exposure to Fine Particulate Matter: Association with Nonaccidental and Cardiovascular Mortality in the Agricultural Health Study Cohort. *Environ. Health Perspect.* **2014**, *122*, 609–615. [CrossRef]
13. Chen, J.-C.; Cavallari, J.M.; Stone, P.H.; Christiani, D.C. Obesity Is A Modifier of Autonomic Cardiac Responses to Fine Metal Particulates. *Environ. Health Perspect.* **2007**, *115*, 1002–1006. [CrossRef] [PubMed]
14. Snow, S.; Boyson, A.S.; Paas, K.H.W.; Gough, H.; King, M.-F.; Barlow, J.; Noakes, C.J.; Schraefel, M.C. Exploring the Physiological, Neurophysiological and Cognitive Performance Effects of Elevated Carbon Dioxide Concentrations Indoors. *Build. Environ.* **2019**, *156*, 243–252. [CrossRef]
15. Chen, R.-Y.; Ho, K.-F.; Hong, G.-B.; Chuang, K.-J. Houseplant, Indoor Air Pollution, and Cardiovascular Effects Among Elderly Subjects in Taipei, Taiwan. *Sci. Total Environ.* **2020**, *705*, 135770. [CrossRef] [PubMed]
16. Chen, Y.-C.; Hsiao, T.-C. Physiological Responses to Different CO_2 Levels in Poor Ventilation Room. In Proceedings of the 6th European Conference of the International Federation for Medical and Biological Engineering, Dubrovnik, Croatia, 7–11 September 2014; Springer: Cham, Switzerland, 2015; pp. 423–426.
17. Jung, C.-C.; Su, H.-J.; Liang, H.-H. Association between Indoor Air Pollutant Exposure and Blood Pressure and Heart Rate in Subjects According to Body Mass Index. *Sci. Total Environ.* **2016**, *539*, 271–276. [CrossRef]
18. Baumgartner, J.; Schauer, J.J.; Ezzati, M.; Lu, L.; Cheng, C.; Patz, J.A.; Bautista, L.E. Indoor Air Pollution and Blood Pressure in Adult Women Living in Rural China. *Environ. Health Perspect.* **2011**, *119*, 1390–1395. [CrossRef]
19. Zhang, X.; Wargocki, P.; Lian, Z. Physiological Responses During Exposure to Carbon Dioxide and Bioeffluents at Levels Typically Occurring Indoors. *Indoor Air* **2017**, *27*, 65–77. [CrossRef]
20. Mizukoshi, A.; Kumagai, K.; Yamamoto, N.; Noguchi, M.; Yoshiuchi, K.; Kumano, H.; Sakabe, K.; Yanagisawa, Y. In-situ Real-Time Monitoring of Volatile Organic Compound Exposure and Heart Rate Variability for Patients with Multiple Chemical Sensitivity. *Int. J. Environ. Res. Public Health* **2015**, *12*, 12446–12465. [CrossRef]
21. Pan, L.; Wu, S.; Li, H.; Xu, J.; Dong, W.; Shan, J.; Yang, X.; Chen, Y.; Shima, M.; Deng, F.; et al. The Short-Term Effects of Indoor Size-Fractioned Particulate Matter and Black Carbon on Cardiac Autonomic Function in COPD Patients. *Environ. Int.* **2018**, *112*, 261–268. [CrossRef] [PubMed]
22. Mishra, A.K.; Schiavon, S.; Wargocki, P.; Tham, K.W. Respiratory Performance of Humans Exposed to Moderate Levels of Carbon Dioxide. *Indoor Air* **2021**, *31*, 1540–1552. [CrossRef]

23. Kajtár, L.; Herczeg, L. Influence of Carbon-Dioxide Concentration on Human Well-Being and Intensity of Mental Work. *Időjárás* **2012**, *116*, 145–169.
24. Jin, R.N.; Inada, H.; Négyesi, J.; Ito, D.; Nagatomi, R. Carbon Dioxide Effects on Daytime Sleepiness and EEG Signal: A Combinational Approach Using Classical Frequentist and Bayesian Analyses. *Indoor Air* **2022**, *32*, e13055. [CrossRef] [PubMed]
25. Zhang, J.; Cao, X.; Wang, X.; Pang, L.; Liang, J.; Zhang, L. Physiological Responses to Elevated Carbon Dioxide Concentration and Mental Workload During Performing MATB Tasks. *Build. Environ.* **2021**, *195*, 107752. [CrossRef]
26. Soltanpour, Z.; Mohammadian, Y.; Fakhri, Y. The Exposure to Formaldehyde in Industries and Health Care Centers: A Systematic Review and Probabilistic Health Risk Assessment. *Environ. Res.* **2022**, *204*, 112094. [CrossRef]
27. Guais, A.; Brand, G.; Jacquot, L.; Karrer, M.; Dukan, S.; Grévillot, G.; Molina, T.J.; Bonte, J.; Regnier, M.; Schwartz, L. Toxicity of Carbon Dioxide: A Review. *Chem. Res. Toxicol.* **2011**, *24*, 2061–2070. [CrossRef] [PubMed]
28. Kim, J.; Kong, M.; Hong, T.; Jeong, K.; Lee, M. Physiological Response of Building Occupants Based on Their Activity and the Indoor Environmental Quality Condition Changes. *Build. Environ.* **2018**, *145*, 96–103. [CrossRef]
29. Kim, J.; Hong, T.; Kong, M.; Jeong, K. Building Occupants' Psycho-Physiological Response to Indoor Climate and CO_2 Concentration Changes in Office Buildings. *Build. Environ.* **2020**, *169*, 106596. [CrossRef]
30. Zhang, X.; Wargocki, P.; Lian, Z. Human Responses to Carbon Dioxide, a Follow-up Study at recommended exposure limits in non-industrial environments. *Build. Environ.* **2016**, *100*, 162–171. [CrossRef]
31. Wargocki, P.; Wyon, D.P.; Clausen, G.; Fanger, P.O. The Effects of Outdoor Air Supply Rate in an Office on Perceived Air Quality, Sick Building Syndrome (SBS) Symptoms and Productivity. *Indoor Air* **2000**, *10*, 222–236. [CrossRef]
32. Wojciech Zareba, E.p.; Maison-Blanche, P.; Locati, E.H.; Martínez Sande, J.L. Noninvasive Electrocardiology in Clinical Practice. *Rev. Española Cardiol.* **2001**, *54*, 1241. [CrossRef]
33. Vehviläinen, T.; Lindholm, H.; Rintamäki, H.; Pääkkönen, R.; Hirvonen, A.; Niemi, O.; Vinha, J. High Indoor CO_2 Concentrations in an Office Environment Increases the Transcutaneous CO_2 Level and Sleepiness During Cognitive Work. *J. Occup. Environ. Hyg.* **2016**, *13*, 19–29. [CrossRef]
34. Wilson, G.F. An Analysis of Mental Workload in Pilots During Flight Using Multiple Psychophysiological Measures. *Int. J. Aviat. Psychol.* **2009**, *12*, 3–18. [CrossRef]
35. Shan, X.; Yang, E.-H.; Zhou, J.; Chang, V.W.C. Neural-Signal Electroencephalogram (EEG) Methods to Improve Human-Building Interaction under Different Indoor Air Quality. *Energy Build.* **2019**, *197*, 188–195. [CrossRef]
36. Liu, W.; Zhong, W.; Wargocki, P. Performance, Acute Health Symptoms and Physiological Responses During Exposure to High Air Temperature and Carbon Dioxide Concentration. *Build. Environ.* **2017**, *114*, 96–105. [CrossRef]
37. Kang, M.; Yan, Y.; Guo, C.; Liu, Y.; Fan, X.; Wargocki, P.; Lan, L. Ventilation Causing an Average CO_2 Concentration of 1,000 Ppm Negatively Affects Sleep: A Field-Lab Study on Healthy Young People. *Build. Environ.* **2024**, *249*, 111118. [CrossRef]
38. Zhang, X.; Wargocki, P.; Lian, Z.; Thyregod, C. Effects of Exposure to Carbon Dioxide and Bioeffluents on Perceived Air Quality, Self-Assessed Acute Health Symptoms, and Cognitive Performance. *Indoor Air* **2017**, *27*, 47–64. [CrossRef] [PubMed]
39. Persily, A. Please Don't Blame Standard 62.1 for 1000 ppm CO_2. *Ashrae J.* **2021**, *63*, 1–2.
40. Urch, B.; Silverman, F.; Corey, P.; Brook, J.R.; Lukic, K.Z.; Rajagopalan, S.; Brook, R.D. Acute Blood Pressure Responses in Healthy Adults During Controlled Air Pollution Exposures. *Environ. Health Perspect.* **2005**, *113*, 1052–1055. [CrossRef]
41. Brook, R.D.; Brook, J.R.; Urch, B.; Vincent, R.; Rajagopalan, S.; Silverman, F. Inhalation of Fine Particulate Air Pollution and Ozone Causes Acute Arterial Vasoconstriction in Healthy Adults. *Circulation* **2002**, *105*, 1534–1536. [CrossRef]
42. Hoffmann, B.; Luttmann-Gibson, H.; Cohen, A.; Zanobetti, A.; de Souza, C.; Foley, C.; Suh, H.H.; Coull, B.A.; Schwartz, J.; Mittleman, M.; et al. Opposing Effects of Particle Pollution, Ozone, and Ambient Temperature on Arterial Blood Pressure. *Environ. Health Perspect.* **2012**, *120*, 241–246. [CrossRef]
43. Urch, B.; Brook, J.R.; Wasserstein, D.; Brook, R.D.; Rajagopalan, S.; Corey, P.; Silverman, F. Relative Contributions of $PM_{2.5}$ Chemical Constituents to Acute Arterial Vasoconstriction in Humans. *Inhal. Toxicol.* **2008**, *16*, 345–352. [CrossRef]
44. Fakhri, A.A.; Ilic, L.M.; Wellenius, G.A.; Urch, B.; Silverman, F.; Gold, D.R.; Mittleman, M.A. Autonomic Effects of Controlled Fine Particulate Exposure in Young Healthy Adults: Effect Modification by Ozone. *Environ. Health Perspect.* **2009**, *117*, 1287–1292. [CrossRef] [PubMed]
45. Power, K.L.; Balmes, J.; Solomon, C. Controlled Exposure to Combined Particles and Ozone Decreases Heart Rate Variability. *J. Occup. Environ. Med.* **2008**, *50*, 1253–1260. [CrossRef]
46. Brook, R.D.; Urch, B.; Dvonch, J.T.; Bard, R.L.; Speck, M.; Keeler, G.; Morishita, M.; Marsik, F.J.; Kamal, A.S.; Kaciroti, N.; et al. Insights Into the Mechanisms and Mediators of the Effects of Air Pollution Exposure on Blood Pressure and Vascular Function in Healthy Humans. *Hypertension* **2009**, *54*, 659–667. [CrossRef]
47. Tarkiainen, T.H.; Timonen, K.L.; Vanninen, E.J.; Alm, S.; Hartikainen, J.E.; Pekkanen, J. Effect of Acute Carbon Monoxide Exposure on Heart Rate Variability in Patients with Coronary Artery Disease. *Clin. Physiol. Funct. Imaging* **2003**, *23*, 98–102. [CrossRef]
48. Lin, L.-Y.; Chuang, H.-C.; Liu, I.J.; Chen, H.-W.; Chuang, K.-J. Reducing Indoor Air Pollution by Air Conditioning Is Associated with Improvements in Cardiovascular Health among the General Population. *Sci. Total Environ.* **2013**, *463–464*, 176–181. [CrossRef]

49. Wang, D.; Yee, B.J.; Wong, K.K.; Kim, J.W.; Dijk, D.-J.; Duffin, J.; Grunstein, R.R. Comparing the Effect of Hypercapnia and Hypoxia on the Electroencephalogram During Wakefulness. *Clin. Neurophysiol.* **2015**, *126*, 103–109. [CrossRef]
50. Dong, W.; Pan, L.; Li, H.; Miller, M.R.; Loh, M.; Wu, S.; Xu, J.; Yang, X.; Shan, J.; Chen, Y.; et al. Association of Size-Fractionated Indoor Particulate Matter and Black Carbon with Heart Rate Variability in Healthy Elderly Women in Beijing. *Indoor Air* **2018**, *28*, 373–382. [CrossRef] [PubMed]
51. Magari, S.R.; Hauser, R.; Schwartz, J.; Williams, P.L.; Smith, T.J.; Christiani, D.C. Association of Heart Rate Variability with Occupational and Environmental Exposure to Particulate Air Pollution. *Circulation* **2001**, *104*, 986–991. [CrossRef] [PubMed]
52. Brook, R.D.; Bard, R.L.; Burnett, R.T.; Shin, H.H.; Vette, A.; Croghan, C.; Phillips, M.; Rodes, C.; Thornburg, J.; Williams, R. Differences in Blood Pressure and Vascular Responses Associated with Ambient Fine Particulate Matter Exposures Measured at the Personal Versus Community Level. *Occup. Environ. Med.* **2010**, *68*, 224–230. [CrossRef]
53. Chuang, H.-C.; Ho, K.-F.; Lin, L.-Y.; Chang, T.-Y.; Hong, G.-B.; Ma, C.-M.; Liu, I.J.; Chuang, K.-J. Long-Term Indoor Air Conditioner Filtration and Cardiovascular Health: A Randomized Crossover Intervention Study. *Environ. Int.* **2017**, *106*, 91–96. [CrossRef]
54. Sørensen, M.; Daneshvar, B.; Hansen, M.; Dragsted, L.O.; Hertel, O.; Knudsen, L.; Loft, S. Personal PM2.5 Exposure and Markers of Oxidative Stress in Blood. *Environ. Health Perspect.* **2002**, *111*, 161–165. [CrossRef]
55. Devlin, R.B.; Ghio, A.J.; Kehrl, H.; Sanders, G.; Cascio, W. Elderly Humans Exposed to Concentrated Air Pollution Particles Have Decreased Heart Rate Variability. *Eur. Respir. J.* **2003**, *21*, 76s–80s. [CrossRef] [PubMed]
56. Jia, X.; Song, X.; Shima, M.; Tamura, K.; Deng, F.; Guo, X. Effects of Fine Particulate on Heart Rate Variability in Beijing: A Panel Study of Healthy Elderly Subjects. *Int. Arch. Occup. Environ. Health* **2011**, *85*, 97–107. [CrossRef]
57. Lin, L.-Y.; Chen, H.-W.; Su, T.-L.; Hong, G.-B.; Huang, L.-C.; Chuang, K.-J. The Effects of Indoor Particle Exposure on Blood Pressure and Heart Rate among Young Adults: An Air Filtration-Based Intervention Study. *Atmos. Environ.* **2011**, *45*, 5540–5544. [CrossRef]
58. Karottki, D.G.; Spilak, M.; Frederiksen, M.; Gunnarsen, L.; Brauner, E.V.; Kolarik, B.; Andersen, Z.J.; Sigsgaard, T.; Barregard, L.; Strandberg, B.; et al. An Indoor Air Filtration Study in Homes of Elderly: Cardiovascular and Respiratory Effects of Exposure to Particulate Matter. *Environ. Health* **2013**, *12*, 116. [CrossRef]
59. Zhou, J.; Wang, H.; Huebner, G.; Zeng, Y.; Pei, Z.; Ucci, M. Short-Term Exposure to Indoor PM2.5 in Office Buildings and Cognitive Performance in Adults: An Intervention Study. *Build. Environ.* **2023**, *233*, 110078. [CrossRef]
60. Liu, S.; Chen, J.; Zhao, Q.; Song, X.; Shao, D.; Meliefste, K.; Du, Y.; Wang, J.; Wang, M.; Wang, T.; et al. Cardiovascular Benefits of Short-Term Indoor Air Filtration Intervention in Elderly Living in Beijing: An Extended Analysis of BIAPSY Study. *Environ. Res.* **2018**, *167*, 632–638. [CrossRef] [PubMed]
61. Liu, S.; Huang, Q.; Wu, Y.; Song, Y.; Dong, W.; Chu, M.; Yang, D.; Zhang, X.; Zhang, J.; Chen, C.; et al. Metabolic Linkages between Indoor Negative Air Ions, Particulate Matter and Cardiorespiratory Function: A Randomized, Double-Blind Crossover Study among Children. *Environ. Int.* **2020**, *138*, 105663. [CrossRef]
62. Chuang, K.-J.; Chan, C.-C.; Chen, N.-T.; Su, T.-C.; Lin, L.-Y. Effects of Particle Size Fractions on Reducing Heart Rate Variability in Cardiac and Hypertensive Patients. *Environ. Health Perspect.* **2005**, *113*, 1693–1697. [CrossRef]
63. Xu, J.; Zhong, B. Review on Portable EEG Technology in Educational Research. *Comput. Hum. Behav.* **2018**, *81*, 340–349. [CrossRef]
64. Kjærgaard, S.K.; Mølhave, L.; Pedersen, O.F. Human Reactions to a Mixture of Indoor Air Volatile Organic Compounds. *Atmos. Environ. Part A. Gen. Top.* **1991**, *25*, 1417–1426. [CrossRef]
65. Mizukoshi, A.; Kumagai, K.; Yamamoto, N.; Noguchi, M.; Yoshiuchi, K.; Kumano, H.; Yanagisawa, Y. A Novel Methodology to Evaluate Health Impacts Caused by VOC Exposures Using Real-Time VOC and Holter Monitors. *Int. J. Environ. Res. Public Health* **2010**, *7*, 4127–4138. [CrossRef]
66. Chen, X.; Li, F.; Liu, C.; Yang, J.; Zhang, J.; Peng, C. Monitoring, Human Health Risk Assessment and Optimized Management for Typical Pollutants in Indoor Air from Random Families of University Staff, Wuhan City, China. *Sustainability* **2017**, *9*, 1115. [CrossRef]
67. Nakayama, Y.; Suzuki, N.; Nakaoka, H.; Tsumura, K.; Takaguchi, K.; Takaya, K.; Hanazato, M.; Todaka, E.; Mori, C. Assessment of Personal Relaxation in Indoor-Air Environments: Study in Real Full-Scale Laboratory Houses. *Int. J. Environ. Res. Public Health* **2021**, *18*, 10246. [CrossRef]
68. Nishihara, N.; Wargocki, P.; Tanabe, S.-I. Cerebral Blood Flow, Fatigue, Mental Effort, and Task Performance in Offices with Two Different Pollution Loads. *Build. Environ.* **2014**, *71*, 153–164. [CrossRef]
69. Pope, C.A., 3rd; Eatough, D.J.; Gold, D.R.; Pang, Y.; Nielsen, K.R.; Nath, P.; Verrier, R.L.; Kanner, R.E. Acute Exposure to Environmental Tobacco Smoke and Heart Rate Variability. *Environ. Health Perspect.* **2001**, *109*, 711–716. [CrossRef] [PubMed]
70. Liao, D.; Creason, J.; Shy, C.; Williams, R.; Watts, R.; Zweidinger, R. Daily Variation of Particulate Air Pollution and Poor Cardiac Autonomic Control in the Elderly. *Environ. Health Perspect.* **1999**, *107*, 521–525. [CrossRef]
71. Bakó-Bíró, Z.; Wargocki, P.; Wyon, D.P.; Fanger, P.O. Poor Indoor Air Quality Slows Down Metabolic Rate of Office Workers. *Proc. Indoor Air* **2005**, *1*, 76–80.

72. Wargocki, P.; Wyon, D.P.; Baik, Y.K.; Clausen, G.; Fanger, P.O. Perceived Air Quality, Sick Building Syndrome (SBS) Symptoms and Productivity in an Office with Two Different Pollution Loads. *Indoor Air* **1999**, *9*, 165–179. [CrossRef]
73. Rovira, J.; Roig, N.; Nadal, M.; Schuhmacher, M.; Domingo, J.L. Human Health Risks of Formaldehyde Indoor Levels: An Issue of Concern. *J. Environ. Sci. Health Part A* **2016**, *51*, 357–363. [CrossRef]
74. Duong, A.; Steinmaus, C.; McHale, C.M.; Vaughan, C.P.; Zhang, L. Reproductive and Developmental Toxicity of Formaldehyde: A Systematic Review. *Mutat. Res./Rev. Mutat. Res.* **2011**, *728*, 118–138. [CrossRef] [PubMed]
75. Kim, K.-H.; Jahan, S.A.; Lee, J.-T. Exposure to Formaldehyde and Its Potential Human Health Hazards. *J. Environ. Sci. Health Part C* **2011**, *29*, 277–299. [CrossRef] [PubMed]
76. Rhomberg, L.R.; Bailey, L.A.; Goodman, J.E.; Hamade, A.K.; Mayfield, D. Is Exposure to Formaldehyde in Air Causally Associated with Leukemia?—A Hypothesis-Based Weight-of-Evidence Analysis. *Crit. Rev. Toxicol.* **2011**, *41*, 555–621. [CrossRef]
77. Goldstein, B.D. Hematological and Toxicological Evaluation of Formaldehyde as a Potential Cause of Human Leukemia. *Hum. Exp. Toxicol.* **2010**, *30*, 725–735. [CrossRef] [PubMed]
78. Shim, I.-K.; Kim, J.; Won, S.R.; Hwang, E.S.; Lee, Y.; Park, S.; Ryu, J.; Lee, J. Prevalence of Sick Building Syndrome Symptoms and Subjective–Objective Indoor Air Quality of Stores in Underground Shopping Districts of Korea. *Build. Environ.* **2023**, *228*, 109882. [CrossRef]