Current Obesity Reports (2025) 14:69
https://doi.org/10.1007/s13679-025-00659-3

REVIEW

Weighty Consequences: Pollution's Hidden Weight in the Obesity Epidemic: A Narrative Review

Seda Nur Köktürk[1] · **Hülya Yardımcı**[2]

Received: 30 January 2025 / Accepted: 18 August 2025
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Abstract

Purpose of Review This review aims to comprehensively examine the relationship between environmental pollution and obesity by integrating epidemiological evidence, biological mechanisms, and anthropometric measurements. It specifically focuses on how pollutants contribute to weight gain and metabolic disruptions, particularly in vulnerable populations.

Recent Findings Environmental pollution, including air, noise, light, persistent organic pollutants, and microplastics/plastic additives, is increasingly recognized as a contributor to the global obesity epidemic. Epidemiological studies highlight the associations between pollution exposure and obesity risk, while mechanistic research reveals how pollutants disrupt metabolic processes, alter hormonal balance, and induce systemic inflammation.

Summary This review synthesizes current findings on the role of environmental pollutants in obesity, integrating both epidemiological and mechanistic perspectives. It explores how exposure to pollutants disrupts metabolic pathways, alters endocrine functions, and promotes systemic inflammation, ultimately contributing to obesity. Vulnerable groups, such as pregnant women, children, adolescents, and the elderly, are particularly at risk due to their heightened sensitivity to environmental exposures. By highlighting critical research gaps, it underscores the need for further longitudinal studies and interventional strategies aimed at mitigating pollution-induced metabolic risks. Public health policies targeting environmental pollution could serve as an effective approach to obesity prevention and control.

Keywords Environmental pollution · Obesity · Overweight · Public health · Epidemiological studies

Abbreviations

BAT	Brown Adipose Tissue
BDE47	2,2',4,4'-Tetrabromodiphenyl Ether
BMI	Body Mass Index
Bmp7	Bone Morphogenetic Protein 7
BPA	Bisphenol A
cm	Centimeter
COx	Carbon oxides
dB	Decibel
DDE	Dichlorodiphenyldichloroethylene
DNA	Deoxyribonucleic acid
HCB	Hexachlorobenzene
IL	Interleukin
kg	Kilogram
m	Meter
Mcp-1	Monocyte Chemoattractant Protein-1
NO2	Nitrogen Dioxide
Nrf2	Nuclear factor erythroid 2–related factor 2
O3	Ozone
OCP	Organochlorine Pesticides
PAH	Polycyclic Aromatic Hydrocarbons
PBDE	Polybrominated Diphenyl Ethers
PCB	Polychlorinated Biphenyls
PFOA	Perfluorooctanoic Acid
pfos	Perfluorooctane Sulfonate
PGC-1	Peroxisome Proliferator-Activated Receptor Gamma Coactivator 1-Alpha
PM	Particulate Matter
PPAR	Proliferator-Activated Receptor

✉ Seda Nur Köktürk
snkokturk@ankara.edu.tr

[1] Department of Nutrition and Dietetics, Ankara University Graduate School of Health Sciences, Ankara, Türkiye, Turkey

[2] Department of Nutrition and Dietetics, Ankara University Faculty of Health Sciences, Ankara, Türkiye, Turkey

Published online: 23 September 2025

Prdm16	PR domain containing 16
PS	Polystyrene
PVAT	Perivascular Adipose Tissue
REV-ERB-	Nuclear Receptor Subfamily 1 Group D Member 1
SREBP-1	Sterol Regulatory Element-Binding Protein-1
TNF-	Tumor Necrosis Factor
UCP-1	Uncoupling Protein
USA	United States of America
W/H	Waist To Hip Ratio
WAT	White Adipose Tissue
g	Microgram
m	Micrometer

Introduction

Obesity, defined as excessive and unhealthy accumulation of fat, adversely affect the body's physiological functions and pose a public health problem with numerous negative impacts on individuals' lives [1]. Currently, obesity has become a global concern, with its prevalence in adults having increased by 1.5 times from year 2000 to the present. Moreover, the prevalence of obesity among children under five years of age has risen in nearly all countries worldwide [2]. Obesity is predominantly a multifactorial disease that typically develops as a result of chronic positive energy balance, wherein energy intake through diet exceeds energy expenditure. Once established, obesity can be maintained even in a state of energy balance. Increasing prevalence of obesity, particularly in many Asian countries alongside rapid industrialization and urbanization in recent years, suggests that shifting lifestyles and environmental factors may also contribute to this rise [3]. Increasing environmental pollution accompanying industrialization has emerged as a significant threat to public health and has been reported to be associated with obesity in epidemiological research. This review aims to elucidate the impact of various types of environmental pollution on the risk of obesity development in pregnant women, children, adolescents, and adults, as well as to explain potential mechanisms contributing to obesity pathophysiology. In addition, this review examines obesity-related anthropometric measurements alongside the risk of obesity development. Furthermore, the review discusses the role of nutrition and provides recommendations for dietary strategies that may help mitigate the adverse effects of environmental pollution on obesity development.

Methods

This narrative review involved a literature search of the PubMed, Google Scholar, Web of Science, Scopus, Oxford University Press, Wiley, and Taylor & Francis databases, accessed through the libraries of both Bayburt and Ankara Universities, up until July 2024. Only studies published in English were included. The review focused on literature from 2015 onwards, with approximately 70% of the citations in this article being published within the last 5 years.

The following keywords were used for the literature search: Obesity" OR "Body Mass Index" OR "Weight Gain" OR "Adiposity" AND "Environmental Pollution" OR "Air Pollution" OR "Noise Pollution" OR "Light Pollution" OR "Persistent Organic Pollutants" OR "Microplastics" OR "Plastic Additives".

Air Pollution

Outdoor air pollution is a mixture of various pollutants originating from both natural sources (such as wildfires and volcanic eruptions) and anthropogenic activities, including transportation, energy production, industrial processes, and residential heating [4]. Exposure to tobacco smoke, fumes from cooking oils used in frying methods, smoke from grilling, and odors from painting contribute to indoor air pollution [5].

Air pollution consists of a heterogeneous mixture of particulate matter (PM), gases, and other pollutants that can be found in particulate phase. Nearly 99% of world's population is exposed to air with high levels of pollutants exceeding recommended limits, breathing unhealthy air, with exposure levels being highest in low and middle-income countries [6].

Oxidative stress and inflammation resulting from air pollution have been associated with DNA damage that may contribute to obesity. According to a hypothesis, this DNA damage has been associated with alterations in purine/pyrimidine metabolism and pathways such as gluconeogenesis and glycogenolysis [7]. Also, $PM_{2.5}$ exposure may cause metabolic disorders of lipid synthases and fatty acid transporter proteins in adipose tissue and liver through the Nuclear factor erythroid 2–related factor 2 (Nrf2)/peroxisome proliferator-activated receptor (PPAR) pathway, leading to adipose tissue overgrowth. This exposure has also been shown to alter expression of key genes involved in lipid metabolism, downregulating PPARα and PPARγ while upregulating SREBP1, as well as increasing levels of inflammatory markers tumor necrosis factor alpha (TNFα) and interleukin-6 (IL-6) [8]. DNA fragmentation has been observed following NO_2 exposure in a dose- and time-dependent manner [9]. In Fig. 1 effects of air pollution

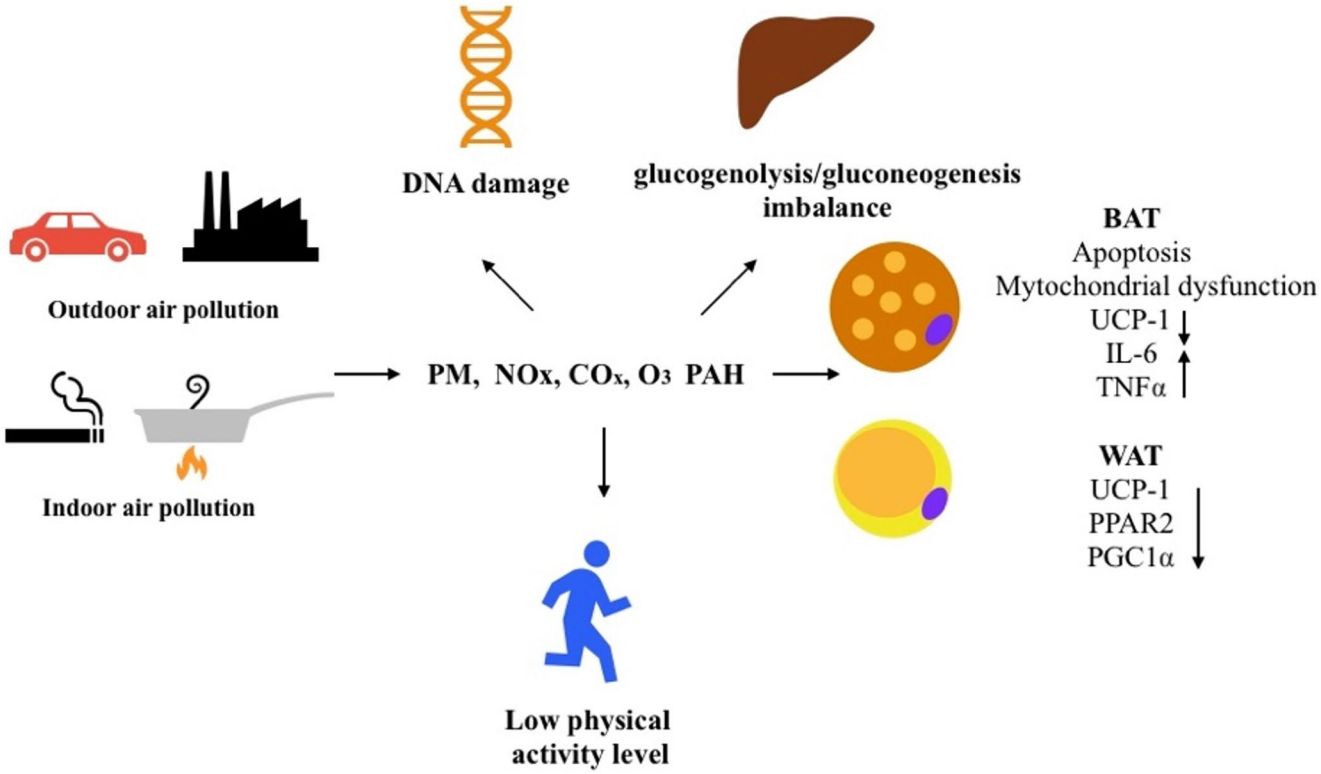


Fig. 1 Mechanisms through which indoor and outdoor air pollution may contribute to the development of obesity

are illustrated. Also, an increase in air pollution levels is related to reduced physical activity, which can contribute to a more sedentary lifestyle [10]. Exposure to air pollutants has been associated with decreased lung function, increased blood pressure, and other cardiovascular and respiratory symptoms, which may be linked to impairments in exercise capacity and performance. Air pollution has also been linked to changes in the expression of Uncoupling protein-1 (UCP-1), a gene involved in thermogenic activity in brown adipose tissue [11]. In addition, air pollution has been linked to changes in brown adipose tissue, including reduced mass, apoptosis, and mitochondrial dysfunction. It has likewise been associated with lower expression levels of PR domain containing 16 (Prdm16), peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α), PPARγ, and UCP-1 genes in white adipose tissue (WAT). Furthermore, $PM_{2.5}$ has been associated with increased expression of IL-6 and TNFα, as well as with markers of apoptosis [12] and impaired brown adipose tissue development [13]. Brown adipose tissue (BAT) promotes thermogenesis by burning energy through uncoupled respiration in mitochondria, a process associated with increased energy expenditure and reduced fat storage [13]. Although BAT may contribute to overall energy expenditure in adults due to its thermogenic potential, its activation in humans is a complex and highly variable process.

Research on the association between air pollution and anthropometric measurements and the risk of obesity development is presented in Table 1. In the relevant literature, studies conducted since 2015 have shown that exposure to $PM_{2.5}$ during pregnancy is associated with parameters such as body mass index (BMI), waist-to-hip ratio (W/H ratio), and obesity in children later in life [14–16]. Fleisch et al. [17], in their study examining the risk of obesity based on the distance of women's homes from main roads during pregnancy, reported a significant increase in children's total body fat mass.

Another study investigating the effects of traffic-related air pollution components on children and adolescents was conducted by Blosemna et al. [18]. This study found that an increase in NO_2 levels was associated with people with overweight. Additionally, a study conducted in Spain reported a link between PM_{10} exposure and increased risk of having obesity [19].

Studies conducted on adults have similarly explored the relationship between air pollution and obesity. In the studies by Wang et al. [20] and Zhang et al. [21] a relationship was found between the increase in BMI and air pollutants, especially in women, while lower BMI values were observed in men. Han et al. [22] identified a higher likelihood of individuals having overweight or obesity among men. However, Kim et al. [23] reported that among air pollution

Table 1 Effect of air pollution on anthropometric measurements and obesity in various groups

	Author, year	Country	Study Design	Pollutants	Pollutant That Has Effects	Confounding Factors	Findings
Pregnant Women							
n:2115	Fleisch et al. [14]	U.S.A.	Prospective cohort	$PM_{2.5}$	$PM_{2.5}$	Pregnancy age, baby birth weight z-score, and gestational age	Air pollution from traffic during the third trimester was associated with: Fetal growth is negatively affected ↓ Obesity risk increased at age 6 ↑
n:1418	Fleisch et al. [17]	U.S.A.	Prospective cohort	$PM_{2.5}$	$PM_{2.5}$	Child's age, gender, and race/ethnicity, mother's age, education level, smoking status during pregnancy, neighborhood income level, and season	Mothers living < 50 m from a main road compared to those living ≥ 200 m away had children with a higher total fat mass of 2.1 kg ↑
n:239	Chiu et al. [24]	U.S.A.	Prospective cohort	$PM_{2.5}$	$PM_{2.5}$	Race/ethnicity, mother's age, education level, mother's pre-pregnancy weight, prenatal smoking, gestational age, and child's birth weight	Exposure to higher doses of $PM_{2.5}$ during weeks 10–29 of pregnancy was associated with higher waist/hip ratio (W/H) in girls. ↑
n:1446	Mao et al. [15]	U.S.A.	Prospective cohort	$PM_{2.5}$	$PM_{2.5}$	Mother's age, education level, smoking status, race/ethnicity, diabetes, marital status, household annual income, birth season, premature birth, breastfeeding status, and birth weight	Compared to the lowest quartile of $PM_{2.5}$ exposure, children in the highest quartile had an increased risk of having overweight/obesity 1.1 times higher for 3rd trimester ↑ 1.3 times higher for age 2↑
n:72,745	Pedersen et al. [25]	Denmark	Prospective cohort	NO_2	NO_2	Mother's age, pre-pregnancy BMI (Body Mass Index), height, season of conception, income, and education level	With every 10 μg/m^3 increase in traffic-related NO_2 levels during the first trimester gestational diabetes mellitus risk increases by 24% ↑ 2nd and 3rd trimester ↔
n:535	Rundle et al. [16]	U.S.A.	Longitudinal	PAH	PAH	Maternal weight gain during pregnancy, pre-pregnancy BMI, gender, ethnicity/race, birth weight, anthropometric measurements, square of the child's age in months.	Exposure to prenatal PAH was associated with higher BMI ↑ at ages 5, 7, and 10.
Children and Adolescents							
n:3680	Bloemsma et al. [18]	Holland	Prospective cohort	Traffic NO_2, $PM_{2.5}$ PM_{10}	NO_2	Age, gender, education level, parents' education level, smoking during pregnancy, father's smoking status at home, socioeconomic status	With every 8.90 μg/m³ increase in NO_2 concentration, an increased risk of overweight by 1.4 times has been reported ↑
n:2660	de Bont et al. [19]	Spain	Cross-sectional	NO_2, $PM_{0.1}$ $PM_{2.5}$ PM_{10}	PM_{10}	Mother and father's education level and country of birth, father's employment status, number of siblings, household status, and mother's smoking status during pregnancy	A 5.6 μg/m³ increase in PM_{10} levels around the home environment rises overweight or obesity risk by 10% ↑ In the school environment, it rises the risk of overweight or obesity by 30%.↑
Adults							
n:13,757	Wang et al. [20]	China	Longitudinal	$PM_{2.5}$, NO_2, O_3	$PM_{2.5}$, NO_2, O_3	Age, gender, education level, marital status, employment status, place of residence, urban gross domestic product, smoking status, alcohol consumption, sleep duration, physical activity, presence of diabetes and hypertension, annual household income, type of cooking fuel, annual average temperature, and relative humidity	Every 10 μg/m³ increase in $PM_{2.5}$, NO_2, and O_3 concentration has been associated with an increase in body weight 0.83, 0.92 and 1.38 kg respectively ↑ waist circumference 0.69, 1.19 and 0.74 cm respectively ↑ Weight gain is significantly higher in females↑
n:7424	Han et al. [22]	China	Longitudinal	$PM_{2.5}$, NO_2, O_3	$PM_{2.5}$, NO_2	Age, gender, smoking status, marital status, alcohol consumption, region (urban or rural), household income, physical activity, presence of chronic diseases, and education level	A 35% higher risk of overweight/obesity was observed ↑ It has been determined that males are at higher risk.
n:7826 (20–60 year)	Zhang et al. [21]	U.S.A.	Cross-sectional	Traffic $PM_{2.5}$	Traffic $PM_{2.5}$	Age, gender, smoking status, country of birth, household income, health insurance coverage, and education level	Males ↓ BMI Females ↑BMI
n:158 (17–22 year)	Kim et al. [23]	U.S.A.	Prospective cohort	NO_2, O_3, $PM_{2.5}$, PM_{10}	O_3	Age, gender, race/ethnicity, occupation, parental education, exercise, smoking status, e-cigarette use, total body fat percentage, dietary energy intake	Short-term exposure to O_3 and long-term exposure to NO_2 have been found to be associated with dyslipidemia Anthropometric measurements have not been affected.

components, O_3 was not associated with changes in anthropometric measurements in young adults, although it was linked to dyslipidemia.

The different findings in studies on adults may be due to various factors such as participant characteristics (e.g., age, gender, socioeconomic status), geographical and temporal differences in air pollution levels, the diversity of measurement methods used, and variations in sample sizes. These differences suggest that the relationship between air pollution and obesity may be more complex and exhibit individual variability.

Noise Pollution

One of the impacts of urbanization on the environment and health is noise pollution. Noise is defined as "unwanted sounds that threaten human health [26]. World Health Organization (WHO) has reported that after air pollution, noise pollution is the type of pollution that poses the greatest threat to human health. According to the European Environment Agency, the predominant cause of noise pollution is road traffic [27].

Noise has been linked to stress and sleep disorders, which are also recognized as risk factors for metabolic diseases [28]. High-level exposure to noise is generally considered to have more direct effects on physiological stress responses, while low-level exposure tends to have more indirect effects, often through disrupting sleep quality over time. Exposure to loud noise is associated with increased stress responses and sleep disorders. Stress involves activation of the autonomic nervous system and endocrine system. Cognitive perception of noise is linked to the release of the stress hormone cortisol [29]. Noise-related activation of the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system has been associated with increased levels of stress hormones such as cortisol and catecholamines. These changes have also been related to higher circulating levels of IL-1ß, IL-6, and pro-inflammatory monocytes, which are involved in inflammation and oxidative stress [30, 31]. Noise pollution has been found to disrupt sleep stages, with increased frequency of nighttime awakenings. Additionally, exposure to traffic noise during sleep has been reported to affect heart rate through autonomic arousal. These changes in heart rate variability have been found to be associated with endothelial dysfunction, which may contribute to the development of metabolic diseases [32].

Research on the relationship between noise pollution and anthropometric measurements and the risk of obesity development is presented in Table 2. Studies examining noise pollution have reported varying effects depending on the type of sound source. Road traffic noise has been shown to affect gestational body weight [33]. Furthermore, exposure to traffic noise during pregnancy has been associated with increased BMI values, as well as higher risks of children having overweight or obesity by the time they reach eight years of age [34, 35]. de Bont et al. [36] reported that exposure to both air and road traffic noise pollution in children and adolescents was associated with a 36% higher likelihood of having overweight or obesity. In a study by Liang et al. [37], which compared children aged 6–9 years living in quiet and noisy environments, the risk of obesity was found to be 50% lower in children living in quiet areas.

Some studies in the literature have also identified associations with anthropometric measurements [38–40]. Foraster et al. [38] reported that each 10 dB increase in road traffic noise was associated with a 20% higher likelihood of individuals having overweight, a 17% higher likelihood of having obesity, and a 16% higher likelihood of having central obesity. An increase in railway noise was associated with a 12% increase in the risk of having overweight. Veber et al. [40] reported that women exposed to bedroom noise had a higher risk of central obesity, while this relationship was not observed in men. In contrast, Cramer et al. [41] in a study conducted in Denmark, found no association between traffic-related noise and BMI or waist circumference. These differences may arise due to variations in study designs, sample differences, the country in which the research was conducted, and other factors.

Light Pollution

Light pollution is a new form of environmental pollution resulting from urbanization and industrialization. International Dark-Sky Association defines light pollution as the artificial alteration of outdoor light levels by human activity [43]. On the other hand, according to the International Commission on Illumination, light pollution is the sum of all adverse effects of artificial light [44]. Today, it is estimated that more than 80% of the world's population is affected by light pollution and its various forms [45]. Additionally, light pollution is increasing at a rate of 2.2% annually due to rapid population growth and expanding urbanization in artificially illuminated areas, exacerbating the problem of light pollution [46].

Light pollution has been associated with sleep disturbances, which may play a role in the development of obesity, mental health conditions, and certain types of cancer [47]. Additionally, certain aspects of the endocrine system, such as hormonal secretion and metabolic regulation, are influenced by the circadian rhythm. The CLOCK gene, a core component of the circadian clock, has been reported to be sensitive to light exposure. It regulates the expression of

Table 2 Effect of noise pollution on anthropometric measurements and obesity in various groups

	Author, year	Country	Study Design	Pollutants	Relevant Pollutants	Confounding Factors	Findings
Pregnant Women							
n:74,065 n:52,661 (postpartum)	Sørensen et al. [33]	Denmark	Longitudinal	Road and railway noise	Road and railway noise	Age, education, household income, regional income, season, smoking and alcohol consumption	With every 10 dB increase in road traffic sound level was associated with Gestational body weight gain increases by 3.8 g/week↑ The body weight that cannot be lost after pregnancy increases by 0.09 kg. Railway noise higher than 65 dB, compared to lower than 55 dB, increases gestational body weight gain by 4.5 g/week ↑
n:6403	Weyde et al. [34]	Norway	Longitudinal	Residential facade traffic noise	residential facade traffic noise	BMI at birth, pre-pregnancy BMI, mother's education level, age, gender of the child, diet (Nordic Diet score), physical activity	It has been reported that from birth to 8 years of age, the group with the highest traffic noise exposure was associated with a 0.35 kg/m² greater increase in BMI compared to the group with the lowest exposure.
n:40,974	Christensen et al. [35]	Denmark	Longitudinal	Residential facade traffic noise	traffic noise	Mother's age at birth, smoking status, BMI, education and level, degree of urbanization	Every 10 dB increase in traffic noise, both during the intrauterine period and during childhood, increases the risk of overweight or obesity by 6%.↑
Children and Adolescents							
n:2213	de Bont et al. [36]	Spain	Cross-sectional	Air pollution, road noise	Air pollution, road noise	Parent's education level, occupation, economic level, mother's smoking status, BMI, number of siblings of the child and country of birth	All types of pollution combined are correlated with an increase BMI by 0.17 kg/m² and the risk of overweight or obesity by 36% ↑ behaviors such as fast food consumption, sedentary life, screen time and sleep ↔
n:3412	Liang et al. [37]	China	Cross-sectional	Residential noise	Residential noise	Age, gender, exercise level, father's education and region of residence, sleep quality, study stress and vegetable/fruit consumption	The risk of obesity for children living in quiet areas is reduced by 50% compared to children living in noisy areas ↓
Adults							
n:484 College students	Argalášová et al. [42]	Slovakia	Cross-sectional	Noise level around the dormitory	Noise level around the dormitory	Gender, nutritional status, behavioral and psychosocial factors used in calculating the Cardiovascular Risk Score	Exposure to traffic noise in men was associated with a 2.19-fold higher body fat percentage.↑
n:3796	Foraster et al. [38]	Sweden	Cross-sectional	Aircraft, highway, railway noise	Highway, railway noise	Lifestyle, physical activity and air pollution	per 10 dB increase in road noise was associated with an increase of: BMI 0.39 kg/m^2 ↑ Body fat mass 0.45% ↑ Waist circumference (WC) 0.93 cm ↑ Overweight risk 20% ↑ Obesity risk 17% ↑ Central obesity risk 16% ↑ Railway noise is associated with a 12% higher risk of overweight. ↑

Table 2 (continued)

	Author, year	Country	Study Design	Pollutants	Relevant Pollutants	Confounding Factors	Findings
n: 5184	Pyko et al. [39]	Sweden	longitudinal	Aircraft, highway, railway noise	Aircraft, highway	Lifestyle, physical activity level and air pollution	Per 10 dB increase in the noise level associated with road and aircraft noise, respectively, WC increases by 0.40 cm and 0.16 cm ↑ Body weight gain was associated only with aircraft noise. Exposure to aircraft and road traffic noise increases the incidence of central obesity by 19% and 7%, respectively. ↑ All three modes of transportation noise increase the risk of obesity by 2.26 times
n:15,501	Cramer et al. [41]	Denmark	cross-sectional	Traffic noise	-	Degree of urbanization, air pollution levels, shift work, use of sedatives, work tension, use of sleeping pills and presence of obesity in the family	It has been reported that traffic noise level is not related to BMI and waist circumference.
n:13,578	Veber et al. [40]	Estonia, Iceland, Norway, Switzerland and Denmark	cross-sectional	Traffic noise on bedroom front	Traffic noise on bedroom front	Age, smoking status, marital status, employment status, education, time spent at home, having children, family history of obesity and physical activity level	Women exposed to traffic noise in the bedroom had a 1.3 kg/m² higher BMI and a 3.3 cm larger waist circumference compared to others, indicating a risk of central obesity. No statistically significant results were found in men.

key enzymes involved in lipid metabolism, including fatty acid synthase, acetyl-CoA carboxylase, and sterol regulatory element-binding protein-1 (SREBP-1) [48].

The down-regulation of the CLOCK gene, which is influenced by PPARα activity, has been linked to impaired lipid metabolism in hepatocytes [49]. The CLOCK gene affects the transcriptional activity of PPARα, thereby modulating circadian rhythm and influencing the expression of genes involved in fatty acid oxidation and ketogenesis in the liver. Exposure to artificial light has been associated with increased microRNA levels, reduced Nuclear receptor subfamily 1 group D member 1 (REV-ERB-α) expression, and higher expression of fatty acid synthase and ATP-citrate lyase. These alterations may support adipogenic processes and have been linked to an increased risk of obesity [50, 51].

A study on outdoor night-time light exposure found that children in the third and fourth quartiles had a higher risk of having overweight or obesity compared to those in the lowest quartile. Specifically, those in the third quartile had a 40% increased risk of having overweight and a 46% increased risk of having obesity, while those in the fourth quartile had a 12% and 20% increased risk, respectively. The associations were stronger in males [52]. In another study, both the presence of artificial light at night and light intensity were found to be associated with the risk of obesity in children and adolescents. Furthermore, it has been found that exposure to light pollution is more strongly associated with obesity in children from regions with low socioeconomic status [53].

A study by Xu et al. [54] reported a significant association between higher night-time light exposure and obesity, with obesity rates increasing by 22% in the second quartile, 35% in the third, and 26% in the fourth quartile compared to the first. The study also found that age and regular physical activity influenced the strength of this association [54]. In another study, night-time light levels were found to be associated with obesity, with this association being stronger in older adults and males. Each incremental increase in nighttime light levels was linked to a 14% higher obesity risk in males and a 24% higher risk in individuals aged 60 and above. Additionally, nighttime light levels related to abdominal obesity risk [55].

A study on individuals aged 50–71 reported that night-time exposure to artificial light was associated with a 12% higher odds of developing obesity in males and 19% in females over a 10-year period [56]. In a study

conducted in Korea, high-intensity artificial light at night was reported to be linked with a 20% higher obesity rate. Subgroup analyses revealed that the prevalence of obesity was 45% higher in elderly individuals, but no such trend was observed in young and adult individuals [57]. The research on the relationship between light pollution, anthropometric measurements, and the risk of obesity is presented in Table 3.

Persistent Organic Pollutants (POPs)

POPs have high lipid solubility. Some pollutants like PFOS can also dissolve in water. These pollutants tend to bioaccumulate in organisms with slow metabolism and therefore persist in the body for extended periods [59]. In addition, these pollutants persist in the environment for extended periods and can be transported internationally. Reports indicate

Table 3 Effect of light pollution on anthropometric measurements and obesity in various groups

	Author, Year	Country	Study Design	Confounding Factors	Findings
Children and Adolescents					
n:47,990	Lin et al. [52]	China	Cross-sectional	Age, gender, parents' education level and annual household income	Compared with children in the lowest quartile of outdoor illumination levels at night, children in the third quartile are at greater risk of overweight 40% ↑ obesity 46% ↑ Additionally, this relationship was found to be stronger in men.
n:129,500	Dang et al. [53]	China	Cross-sectional	Age, gender and residence address, sleep time, exercise time, breakfast habits, egg and milk consumption, screen time	The risk of obesity was associated with both the area illuminated by artificial light at night and the intensity of the light.↑
Adults					
n:43,722	Park et al. [58]	U.S.A.	Cross-sectional longitudinal	Age, race/ethnicity, location, educational attainment, household income, marital status, smoking and alcohol consumption, caffeine consumption, menopausal status at baseline, depression, and perceived stress	Women exposed to artificial lighting while sleeping at night are at higher risk of becoming overweight or obese 19% ↑ A comparison between those not exposed to artificial lighting and those who slept with the light on at night showed that the latter group had a body weight ≥5 kg ↑ BMI 10% ↑ overweight risk 22% ↑ obesity risk 33% ↑
n:24,845	Xu et al. [54]	China	Cross-sectional	Age, gender, race, household income level, education level, low-energy weight loss diet, sugary drink consumption, regular exercise, alcohol and cigarette consumption	Higher light levels were found to be significantly associated with obesity. It has been reported that the obesity rate increased by 22% in the second quarter, 35% in the third quarter, and 26% in the fourth quarter compared to the first quarter. In addition, this study determined that age and regular exercise may influence the relationship between light pollution and obesity risk.
n:98,658	Zhang et al. [55]	China	Cross-sectional	Age, gender, education level, smoking and alcohol consumption, physical activity, healthy nutrition score, region of residence (rural/urban), household income, HOMA-IR	Although night light level is associated with obesity, this relationship has been found to be stronger in the elderly and men. Each gradual increase in night light levels was correlated with a 14% higher risk of obesity in men and a 24% higher risk in individuals aged 60 and over. It has also been found that the risk of abdominal obesity is associated with night light levels.
n:8525	Koo et al. [57]	S. Korea	Cross-sectional	Age, gender, education level, building type, household income, alcohol consumption, smoking, caffeine or alcohol consumption before sleep, delayed sleep pattern, short sleep duration and snoring habit.	High-intensity artificial light at night is associated with a 20% higher obesity prevalence. Subgroup analysis showed a 45% higher obesity risk specifically in older individuals.
n:239,781 (50–71 age)	Zhang et al. [56]	U.S.A.	Prospective cohort	Age, gender, BMI race/ethnicity, education, baseline status, marital status, poverty rate, and population density	Exposure to artificial light is associated with a 12% higher risk of obesity in men and a 19% higher risk in women.

that such pollutants are found even in regions where signs of life are not observed [60]. Additionally, these mentioned pollutants bioaccumulate in the food chain and raise health concerns [59]. These pollutants pose a significant health problem, particularly in developing countries. Approximately 90% of human exposure to POPs comes from contaminated food or animal feed sources [61].

These compounds, found ubiquitously, have been implicated in metabolic disturbances mediated through adipose tissue, which functions not only as a site for energy storage but also as an active endocrine organ by secreting adipokines and other signaling molecules. These metabolic disruptions have been linked to obesity, diabetes, nonalcoholic fatty liver disease, and hyperlipidemia [62]. With weight loss, persistent organic pollutants stored in adipose tissue are released into the circulation, potentially exerting additional systemic effects [63].

Preclinical research suggests that dioxin may disrupt mitochondrial function and is associated with obesity and metabolic syndrome through the activation of aryl hydrocarbon receptor ligands [64]. Evidence suggests that perfluoroalkyl compounds may play a role in adipogenesis, possibly by interacting with the PPARγ signaling pathway [65]. Similarly, PFOS has been linked to adipogenic effects, possibly through interference with human mesenchymal stem cell function [66]. In addition, pollutants can accumulate in adipose tissue and have been associated with endoplasmic reticulum stress, adipokine dysregulation, and increased inflammatory markers in hypertrophic, dysfunctional adipose tissue [67].

In a study, participants' serum POP concentrations were measured, revealing that low doses of POPs were associated with DNA hypomethylation, which may be linked to increased expression of normally silenced repetitive elements or proto-oncogenes, potentially contributing to genomic instability and increased disease risk [68]. In addition, another study reported that chronic exposure to atrazine was associated with a decrease in basal metabolic rate, along with increased body weight, intra-abdominal fat accumulation, and insulin resistance [64].

A preclinical study investigated the role of DDE and microbiota dysbiosis in obesity. The research observed that DDE exposure in rats was associated with increased body weight and fat percentage, ass well as impaired glucose homeostasis. Additionally, microbiota analysis further indicated an elevated *Firmicutes/Bacteroidetes* ratio, which is commonly linked to dysbiosis. In addition to these findings, organic pollutants have been shown to alter the lipid metabolome profile. Specifically, phosphatidylethanolamine and triacylglycerol have been reported to strongly correlate with dysbiosis within the changing lipid metabolome profiles [69].

PCBs, PBDEs, and OCPs have been reported to be associated with alterations in bile acid metabolism, potentially through effects on the bile salt hydrolase activity of *Lactobacillus*, *Clostridium*, and *Bacteroides*. Bile acids are not only essential for the emulsification and absorption of dietary lipids but also act as signaling molecules regulating metabolic pathways involved in glucose and lipid homeostasis. Additionally, chemicals like DDE have been associated with changes in the ratio of *Proteobacteria* to *Firmicutes/Bacteroidetes*, and may influence short-chain fatty acid synthesis. These microbial alterations have been linked to disruptions in energy metabolism that are observed in obesity and prediabetic conditions [70]. The effects of persistent organic pollutants on anthropometric measurements and obesity are presented in Table 4.

Microplastics and Plastic Additives

There are currently at least 45 different types of plastic commercially available in the world, including polypropylene, polyethylene, polyethylene terephthalate, polystyrene (PS), polyurethane, polyvinylchloride and polycarbonate. Plastics are widely preferred because they are resistant to chemical and biological degradation and low cost [77]. It is estimated that approximately 75.000-300.000 tons of microplastics are released annually in European Union countries [78]. As plastics continue to break down in the environment, microplastics are projected to make up 13% of the total plastic mass by 2060, with this proportion expected to keep rising [79].

Although the exact amount of microplastics ingested through diet is not definitively determined, the presence of microplastics has been detected in various foods and beverages such as seafood, honey, milk, beer, tea, soft drinks, salt, and sugar [80–86]. Moreover, exposure to microplastics through the placenta during intrauterine life has been reported. This situation reveals the danger posed by microplastics [87]. Due to their tiny size, microplastics inhaled through air or ingested with food can be absorbed by lung and intestinal epithelial cells. Their presence has been associated with increased reactive oxygen species formation and epithelial permeability, potentially triggering immune system responses [88, 89]. Moreover, microplastics have been detected in the liver and kidney and have been associated with alterations in lipid metabolism, possibly by supporting adipocyte differentiation [90]. A preclinical study reported that microplastics were associated with altered adipogenesis and lipid metabolism, potentially through modulation of PPAR signaling, which regulates genes involved in lipid uptake, storage, and adipocyte differentiation [91]. In another preclinical study, exposure to PS microplastics was linked

Table 4 Effect of persistent organic pollutants on anthropometric measurements and obesity in various groups

	Author, Year	Country	Pollutants	Study Design	Confounding Factors	Findings
Pregnant Women						
n:412	Lauritzen et al. [71]	Sweden	PFOS, PFOA, PCB-153	Prospective cohort	Mother's age, education level, smoking during pregnancy, pre-pregnancy BMI, body weight gain at week 17, interpregnancy interval and previous breastfeeding duration.	Maternal serum POP levels during the 17-20th gestational weeks were associated with an increase in the triceps skinfold thickness z-score ↑ and BMI z-score ↑ With every 1-unit increase in PFOS levels, the risk of overweight or obesity in children increases 2-fold. ↑
n:447	Cabrera-Rodríguez et al. [72]	Spain	p, p'-DDE, PCB-28, PCB-52, BDE-47	Cross-sectional	-	p, p′-DDE was associated with an increase in birth weight in female infants ↑ Birth weight decreased ↓ in male infants exposed to more than three types of OCPs during the prenatal period. Increased levels of PCB-28, PCB-52, and BDE-47 were associated with lower birth weight ↓ PAH exposure had no significant effect on fetal development ↔
n:520	Tang-Péronard et al. [73]	Faroe Islands	PCB, DDE, HCB	Prospective cohort	Mother's pre-pregnancy BMI, education level, socioeconomic status, smoking and gestational age	PCB levels in maternal serum at the 34th week of pregnancy and in transitional milk during the postpartum period were examined. Postprandial insulin levels increased 3.71-fold ↑ only in female infants.↑
n:1667	Rouxel et al. [74]	Spain	Mixed POP	Longitudinal	Mother's smoking during pregnancy, child's fish consumption and child's sedentary behavior	POP levels in maternal cord serum were examined. BMI z-score and body fat mass increased ↑ at age 12 In particular, HCB and beta-hexachlorocyclohexane have been reported to have a significant effect on adiposity.
n:379	Güil-Oumrait et al. [75]	Spain	p, p'-DDT, p,p'-DDE, HCB, PCB	Longitudinal	Mother's pre-pregnancy BMI, education level, socioeconomic status, smoking and gestational age	HCB is associated with an increase in BMI and Waist/Hip ratio z-score ↑ Sustained increases in HCB were associated with increases in body fat percentage.
Adults						
n:8365	LaVerda et al. [76]	Spain	Triazine, atrazine	Longitudinal	BMI, age, smoking, energy intake/expenditure	BMI per 100 cumulative days of pesticide exposure: Triazine 0.07–0.11 kg/m² increase ↑ Atrazine 0.10–0.19 kg/m² increase ↑

to alterations in the gut microbiota, including reduced beta-diversity and changes in the relative abundance of bacterial populations previously linked to obesity. Additionally, PS exposure was associated with dysregulation of gene expression in perivascular adipose tissue (PVAT), including upregulation of pro-inflammatory markers (IL-6 and Mcp-1) and adipogenesis-related genes such as Irs2 and Bmp7, which may contribute to cardiovascular dysfunction [92] Polystyrene microplastics of 5–20 µm in size may accumulate in the liver, kidneys, and intestines, where such accumulation is related to alterations in ATP synthesis and changes in lipid metabolism. Deng et al. (2017) [90] reported that 5–20 µm PS exposure was associated with decreased triglyceride and total cholesterol levels in the livers of mice. Another study found that PS exposure was related to reduced the mRNA levels of key genes involved in lipogenesis and triglyceride synthesis in the epididymal fat of mice [93]. Jin et al. [94] observed changes in the transcriptional levels of CYP7a1 and ABCb11, two proteins involved in bile acid synthesis and transport, in the livers of mice exposed to PS microplastic.

Lee et al. (2023) [95] found that PS microplastics exhibited high affinity for macrophages in mice that developed obesity due to a high-fat diet. This was accompanied by increased systemic inflammation and insulin resistance, suggesting a potential link between microplastic exposure and disrupted glucose metabolism. In a systematic review of preclinical studies, microplastic exposure was reported to be associated with intestinal dysbiosis, characterized by an increased abundance of *Firmicutes*, *Proteobacteria*, and *Chlamydia*, and a reduction in *Bacteroidetes*. These microbial alterations were linked to increased intestinal permeability and elevated levels of inflammatory markers such as IL-1α, IL-1ß, TNFα, interferon gamma, and IL-6 [96].

Figure 2 shows the sources of air pollution and its effects on the gut.

Butyltins have been shown to bind to retinoic acid receptors and PPARγ, and their exposure has been associated with increased adipogenesis and lipid accumulation [97]. Similarly, monoethylhexyl phthalate and diethylhexyl phthalate may exert their effects through PPARγ activation. Due to structural similarities, Bisphenol A (BPA) and 17-β-estradiol can bind to estrogen receptors, and such interactions have been linked to obesogenic effects. Additionally, both 17-β-estradiol and BPA have been observed to stimulate pancreatic β-cells. Although the short half-life of BPA in blood complicates its measurement, studies have reported associations with altered glucose metabolism and insulin resistance in exposed individuals [98]. In a review, prenatal exposure to endocrine-disrupting plastic additives was reported to alter embryonic metabolism via epigenetic mechanisms and may increase the risk of obesity in subsequent generations [99].

The level of exposure to microplastics varies depending on geographical location and individual factors, and it has not yet been clearly determined. Compared to other pollutants, microplastics represent a relatively new area of research, and the number of studies investigating their impact on obesity in humans is limited. Therefore, a table summarizing this topic could not be provided in this section.

Discussion

The rising prevalence of obesity has become a focal point in scientific literature, particularly in relation to industrialization and its associated environmental consequences. Environmental pollution, an inevitable byproduct of industrial development, poses significant and widespread threats to public health, transcending geographical and developmental boundaries. While a substantial body of literature has examined the epidemiological associations between obesity and various forms of environmental pollutants—such as air pollution, noise, persistent organic pollutants light, and microplastics—this study distinguishes itself by integrating a comprehensive analysis of multiple pollution types, alongside a detailed exploration of the underlying biological mechanisms involved in the development of obesity.

What sets this research apart is its dual focus: it not only examines the epidemiological correlations between pollution exposure and obesity but also aims to uncover the biological pathways that mediate this relationship. Moreover, it is the first and only review to explore this topic across different populations, making it a unique contribution to the field. This dual approach, integrating both epidemiological and biological aspects, strengthens the paper's significance by filling an important gap in the existing literature and offering a more comprehensive understanding of the connection between pollution and obesity.

By considering hormonal dysregulation, metabolic alterations, and inflammatory processes as key mediators, this study aims to provide a deeper understanding of the biological mechanisms through which environmental pollutants may be related to the development of obesity. This approach underscores the necessity of future research prioritizing the identification and characterization of the specific biological and chemical mechanisms through which pollutants exert their obesogenic effects, focusing on metabolic disruptions, endocrine imbalance, and immune system modulation.

The contributions of this research extend beyond the establishment of epidemiological links, offering critical insights into the causal mechanisms underlying obesity development in polluted environments. Longitudinal cohort studies, particularly those that are prospective in nature, are essential for firmly establishing causal relationships between pollutant exposure and the pathogenesis of obesity. Additionally, investigating the genetic and epigenetic effects of environmental pollutants could further illuminate individual susceptibility to obesity, enabling the development of personalized prevention and intervention strategies. The potential cumulative effects of simultaneous exposure to multiple pollutants also warrant rigorous investigation, as real-world exposure often involves complex mixtures of environmental stressors. Understanding these cumulative impacts will be pivotal in developing more comprehensive models of obesity risk in polluted environments.

Although the majority of studies in the literature, as well as those included in this article, do not establish causality, they consistently report associations between environmental pollution, exposure to various pollutants, and increased anthropometric measurements across different populations. However, this narrative review does not provide statistical results. Future systematic reviews and meta-analyses incorporating statistical methods are needed to strengthen the evidence base.

Ultimately, this research may contribute meaningfully to the field of nutrition science by enhancing our understanding of the potential environmental factors involved in obesity development. By exploring the biological and biochemical pathways that may underlie the relationship between pollution and obesity, this work can help inform dietary and lifestyle recommendations. By exploring potential biological and biochemical pathways through which pollution may influence metabolic health, this work can help guide the development of dietary and lifestyle strategies intended to reduce the possible impacts of environmental pollutants.

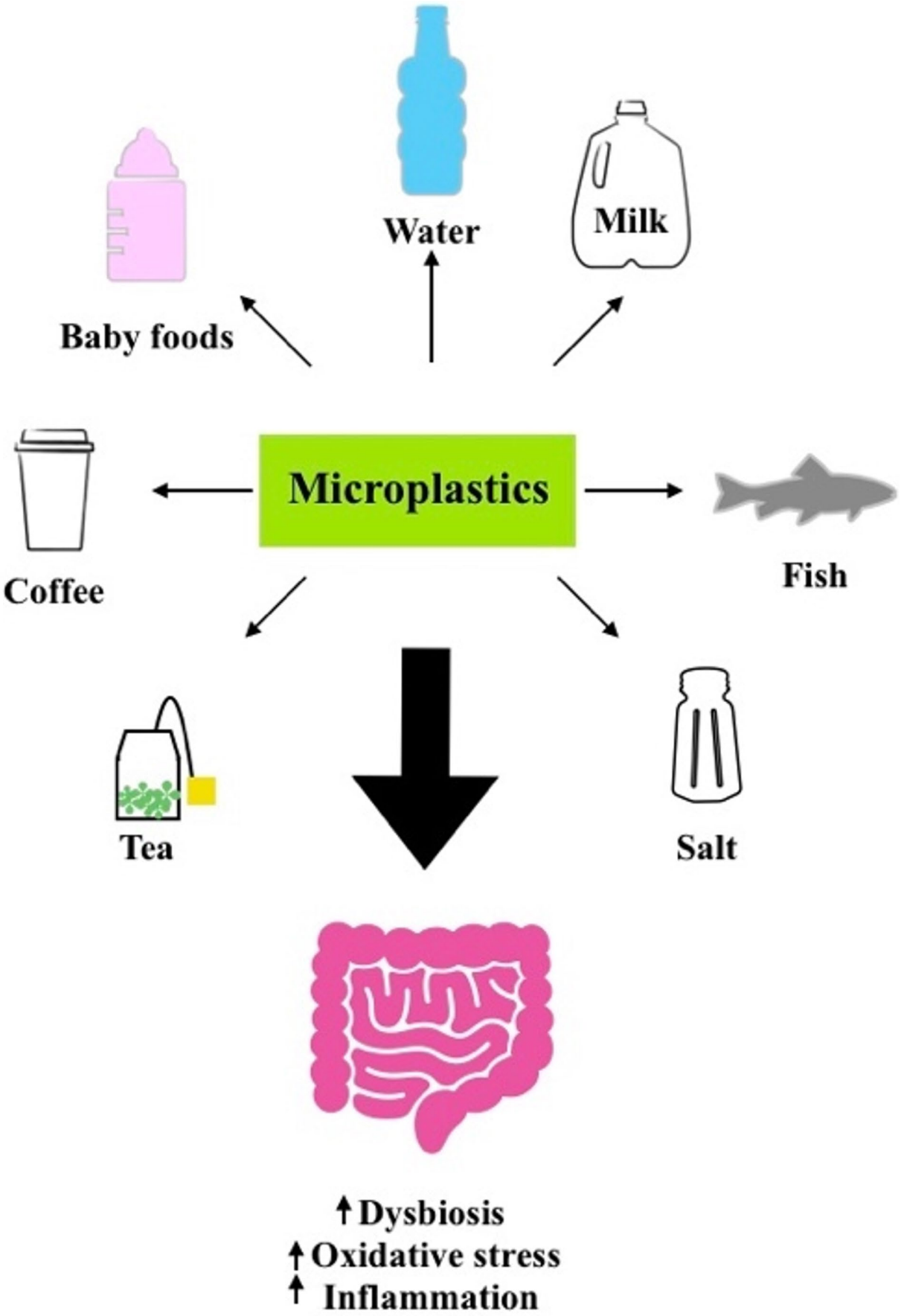

Water
Milk
Baby foods
Microplastics
Coffee
Fish
Tea
Salt
Dysbiosis
Oxidative stress
Inflammation

Fig. 2 Effects of microplastics on gut

These insights may be valuable for nutrition scientists, dietitians, and public health professionals in designing strategies to address obesity from an environmental and nutritional perspective.

Dietary strategies, such as consuming antioxidant-rich foods—including fruits, vegetables, nuts, and antioxidant beverages like green and black tea—may help alleviate oxidative stress potentially associated with exposure to environmental pollutants [100]. To minimize exposure, it is recommended to consume fruits and vegetables in season, ensuring they are washed thoroughly or treated with appropriate cleaning solutions. Agricultural activities should be conducted away from industrial and traffic-related areas, while organic farming practices should be promoted [101]. Measures to maintain indoor air quality include avoiding activities such as frying and smoking, which release pollutants into indoor environments. Reducing plastic usage, choosing glass containers over plastic for food storage, and supporting the purchase of organically certified foods with a lower environmental impact are also critical steps.

Strategies to combat light pollution, such as using heavy curtains and promoting good sleep hygiene by avoiding screen exposure before bedtime, can complement dietary measures aimed at mitigating the effects of environmental pollution. Regular physical activity, at recommended levels and preferably in environments with minimal exposure to traffic-related air pollution, is essential for all age groups and genders. Obesity and environmental pollution appear to have a bidirectional and potentially reinforcing relationship. Pollutants that accumulate in adipose tissue may be retained for extended periods and could re-enter systemic circulation under conditions such as weight loss or impaired insulin sensitivity, potentially enhancing their biological activity [102]. Therefore, gradual weight loss strategies may be considered in individuals who have overweight or obesity, as these approaches could potentially minimize the risk of a rapid release of stored pollutants into the circulation during weight loss. Dietary energy intake should be calculated according to guidelines recommending weight loss of 0.5–1.0 kg/week [103], ensuring sufficient intake of antioxidant-rich foods and dietary diversity to compensate for nutrient losses during food processing. Embracing dietary patterns rich in antioxidants, such as the Mediterranean diet, which aligns with these recommendations, is advisable for mitigating the health impacts of environmental pollution in the context of obesity.

Conclusion

The findings from this review underscore the significant impact of environmental pollution on various age groups and sexes, particularly regarding its contribution to the obesity epidemic. Understanding the mechanisms by which pollutants—such as air, noise, light, persistent organic pollutants, and microplastics—impact metabolic health is essential for developing nutritional strategies to combat obesity. These pollutants disrupt metabolic processes, influence gut microbiota, alter hormonal balances, and trigger systemic inflammation, all of which contribute to weight gain and increased risks of diet-related chronic diseases. Given the global rise in obesity and environmental pollution, it is crucial for nutrition professionals to integrate this knowledge into dietary interventions and public awareness campaigns. Dietitians and nutritionists should advocate for the inclusion of pollution-mitigating foods, such as those rich in antioxidants and anti-inflammatory compounds, in dietary recommendations to support resilience against metabolic damage.

Reducing the impact of environmental pollution on obesity requires collaboration not only among dietitians but also among medical and public health professionals, environmental scientists, urban and regional planners, transportation engineers, agricultural and food scientists, as well as education and communication experts. This interdisciplinary cooperation will provide comprehensive and effective solutions to mitigate the effects of environmental pollution on obesity. Furthermore, public education initiatives must emphasize the connection between dietary patterns and environmental exposures, encouraging healthier food choices that minimize both personal risk and environmental footprint. Vulnerable populations—including pregnant women, infants, children, the elderly, and individuals with pre-existing metabolic conditions—require tailored nutritional strategies to mitigate the heightened risks posed by pollutants. Nutritional guidelines should consider protective dietary factors and the role of nutrient-dense, whole foods in reducing inflammation and supporting metabolic health under environmental stressors. Future research should prioritize long-term studies exploring the interactions between dietary intake, pollutant exposure, and metabolic health, as well as investigating genetic and epigenetic factors that may influence individual susceptibility. Additionally, exploring the cumulative effects of dietary contaminants and prenatal exposure can guide the development of targeted nutritional interventions. Addressing the intersection of environmental pollution and nutrition requires a multidisciplinary approach, integrating toxicology, biochemistry, epidemiology, dietetics, public health, and environmental sciences. With this multidisciplinary approach, the aim should be to

minimize the negative effects of environmental pollution, especially in vulnerable groups.

Limitations

This review has several limitations. First, the studies included are predominantly observational, which makes it difficult to establish causal relationships between environmental pollutants and obesity. As a result, the findings are based on correlations rather than causality, and further experimental research is required to explore these relationships in more depth. Second, due to the broad scope of environmental pollutants, some key pollutants associated with obesity were not addressed in this review. For example, substances such as smoking, phenols, phthalates, and arsenic, which are relevant to understanding the role of environmental pollutants in obesity, were not included. Additionally, due to the broad scope of environmental pollutants, not all mechanisms through which these pollutants may influence obesity could be thoroughly addressed in this review. A more detailed analysis of these mechanisms is necessary to better understand the complex interactions between pollutants and obesity. Finally, as a narrative review, this article presents an overview without the statistical rigor and objectivity of systematic reviews, which may limit the strength of the evidence and the ability to draw definitive conclusions. Future systematic reviews should quantitatively analyze existing studies to provide a more accurate assessment of the relationship between environmental pollutants and obesity.

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Han W, Xu Z, Hu X, Cao R, Wang Y, Jin J, et al. Air pollution, greenness and risk of overweight among middle-aged and older adults: A cohort study in China. Environ Res 2023; 216(Pt 1): 114372. https://doi.org/10.1016/j.envres.2022.114372This is one of the most recent longitudinal studies demonstrating that long-term exposure to air pollution increases the risk of being overweight or obese.

Wang Y, Tan H, Zheng H, Ma Z, Zhan Y, Hu K, et al. Exposure to air pollution and gains in body weight and waist circumference among middle-aged and older adults. Sci. Total Environ. 2023;869: 161895. https://doi.org/10.1016/j.scitotenv.2023.161895This study expands on previous findings by demonstrating that air pollution contributes not only to overall weight gain but also to abdominal obesity, a key risk factor for metabolic diseases.

Liang X, Tang X, Liu M, Liang X, Chen L, Chen X, et al. Associations of self-reported residential noise exposure with obesity and hypertension in children and adolescents. Front Pediatr. 2022; 10: 902868, doi: https://doi.org/10.3389/fped.2022.902868A study conducted with children reported that noise pollution increases the risk of both abdominal obesity and overall obesity.

Veber T, Pyko A, Carlsen HK, Holm M, Gislason T, Janson C, et al. Traffic noise in the bedroom in association with markers of obesity: a cross-sectional study and mediation analysis of the respiratory health in Northern Europe cohort. BMC Public Health 2023;23:1–12. https://doi.org/10.1186/s12889-023-16128-2This study underscores the significant impact of traffic noise on weight management and abdominal fat distribution in women, highlighting sleep disturbances as a key mediating factor.

Lin LZ, Zeng XW, Deb B, Tabet M, Xu SL, Wu QZ, et al. Outdoor light at night, overweight, and obesity in school-aged children and adolescents. Environ. Poll. 2022;305:119306. https://doi.org/10.1016/j.envpol.2022.119306By focusing on a large, diverse sample of children and adolescents, this study offers vital evidence on the gendered effects of light pollution, paving the way for future research and interventions aimed at reducing environmental influences on childhood obesity.

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Lee AG, Kang S, Yoon HJ, Im S, Oh SJ, Pak YK. Polystyrene microplastics exacerbate systemic inflammation in high-fat diet-induced obesity. Int. J. Mol. Sci. 2023; 24(15):12421. https://doi.org/10.3390/ijms241512421It has been reported that microplastic exposure exacerbates inflammation in obesity induced by a high-fat diet.

Author Contributions Conceptualization: Seda Nur Köktürk, Hülya Yardımcı; Methodology: Seda Nur Köktürk, Hülya Yardımcı; Writing - original draft preparation: Seda Nur Köktürk; Writing - review and editing: Seda Nur Köktürk, Hülya Yardımcı.

Funding No funding was provided for this study.

Data Availability No datasets were generated or analysed during the current study.

Declarations

Competing interests The authors declare no competing interests.

Conflict of interest The authors have no relevant interests to declare.

Ethical Approval Ethical approval was not required for this type of article as it does not involve original research with human neither animal subjects.

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