POLISH HEART JOURNAL

Kardiologia Polska
The Official Peer-reviewed Journal of the Polish Cardiac Society since 1957

Online first
This is a provisional PDF only. Copyedited and fully formatted version will be made available soon

ISSN 0022–9032 e-ISSN 1897–4279

Environmental degradation. An under-recognized secondary risk factor of hypertension

Authors: Elias Sanidas, Maria Velliou, Dimitrios Papadopoulos, Costas Thomopoulos, Charalampos Grassos

Article type: Review

Received: January 3, 2025

Accepted: January 29, 2025

Early publication date: February 14, 2025

This article is available in open access under the Creative Common Attribution International (CC BY) license, which allows copying, distributing, and transmitting work, adapting work, and making commercial use of the work under the condition that the user must attribute the work in the manner specified by the author or licensor (but not in any way that suggests they endorse the user or their use of the work).

Environmental degradation. An under-recognized secondary risk factor of hypertension

Short title: Environmental degradation, a modern risk factor of hypertension

Elias Sanidas[1], Maria Velliou[2], Dimitrios Papadopoulos[1], Costas Thomopoulos[1], Charalampos Grassos[3]

[1]Department of Cardiology, Laiko General Hospital, Athens, Greece
[2]Emergency Medicine Department, Attikon University Hospital, Athens, Greece
[3]Department of Cardiology, General Oncology Hospital "Oi Agioi Anargiroi", Kifisia, Greece

Correspondence to:
Elias Sanidas, MD, PhD,
Staff Attending Interventional Cardiologist,
Department of Cardiology, Laiko General Hospital
Agiou Thoma 17, 11522, Athens, Greece,
e-mail: easanidas@yahoo.gr

ABSTRACT

Environmental degradation is an increasingly urgent issue caused by anthropogenic activities, such as urbanization, industrialization and overpopulation growth, and natural disasters, such as flood, typhoons, droughts and rising temperatures. The most common causes of environmental degradation include air, noise and light pollution and climate change, also referred as global warming, with extreme weather patterns. Available evidence undoubtedly supports that the aforementioned environmental changes have been recognized as contributing to cardiovascular morbidity and mortality. The aim of this review was to discuss the impact of various environmental exposures, including air pollutants, noise and artificially light at night, and changes in ambient temperature and humidity on blood pressure values and the burden of hypertension.

Key words: climate change, environment, hypertension, noise, pollution

INTRODUCTION

Human health and wellbeing are inextricably linked to environment. Evidence suggests that climate change, ambient and household air quality and environmental parameters such as noise, light, temperature and humidity impact human health. According to a World Health Organization (WHO) report, it is estimated that approximately 12.6 million deaths every year are attributed to unhealthy environments [1]. Exposure to environmental stressors has also been increasingly recognized as a secondary causative factor of cardiovascular disease, including hypertension (HTN) [2].

HTN is considered an important risk factor for global disability and mortality despite medical advances in treatment and prevention. Elevated blood pressure (BP) has been recognized as contributor of cardiovascular disease including heart failure, coronary artery disease, atrial fibrillation, heart valve diseases, aortic syndromes, stroke and chronic kidney disease [3]. Nowadays, 1.39 billion adults globally have HTN and this number is predicted to increase to 1.5 billion by 2025 [4]. The recently published guidelines on HTN by the European Society of Cardiology acknowledged the role of environmental factors in the risk, progression and severity of the disease in addition to established pathophysiologic processes [5]. Recognizing the urgent need for measurements to reduce the harmful environmental exposures and create healthier environments, the WHO developed a repository of systematic review on interventions in environment, climate change and health [6, 7].

Unhealthy environments and climate change related HTN represents one of the biggest challenges. Even though the exact impact of environmental stressors in BP is not fully established yet, it is clear that they have negative effects on the risk, the development and the treatment of the disease. The aim of this article is to review and summarize current evidence regarding the effect of environment and specifically noise, air and light pollution, ambient temperature and outdoor humidity on BP levels and the burden of HTN.

ENVIRONMENTAL FACTORS AND RISK OF HYPERTENSION

Noise pollution

Exposure to environmental noise has emerged as a serious public issue worldwide due to the rapid increase in urbanization, industry and transportation. Available data supports that road traffic noise is the most substantial source of annoyance, followed by neighbour and aircraft noise. Railway

noise, industrial noise and novel sources of noise, such as wind turbines, are enumerated less frequently [8]. It is estimated that approximately 40% of European citizens is exposed to road traffic noise levels >55 dB over a 24-hour period while 30% is exposed to noise levels >55 dB during the night (between 11 P.M. and 7 A.M.) [9]. People in their mid-40s (peaking at the age of 44) report higher levels of annoyance from traffic noise highlighting an inverted U-shaped effect of age [10]. The recommended levels for specific sources of noise are presented in Table 1 [11].

Nowadays, noise has been identified as an essential, though under-recognized, risk factor of HTN with dose-response relationship. People living or working in environments with noise exposure are significantly correlated with greater risk of HTN [12]. Patients with cardiovascular disease and males are more prone to noise related development of HTN [13]. From pathophysiologic point of view, stress caused by sound increases the sympathetic nervous activity and the release of catecholamines (adrenalin and noradrenaline) from the adrenal medulla, resulting in peripheral vasoconstriction and increased heart rate and BP. In parallel, sound promotes the release of cortisol that further enhances the effect of catecholamines rising BP [14]. Exposure to aircraft noise has been associated with an increase in arterial stiffness and carotid-femoral pulse wave velocity and even short-term decline in sound, as caused during the COVID-19 lockdown, might reserve this unfavorable effect (Table 2) [15, 16].

A meta-analysis of 26 studies conducted by the WHO showed that a rise in traffic noise by 10 dB was correlated with a 1.05-fold greater risk of incident HTN [17]. In addition, chronic exposure to aircraft noise has been associated with HTN among populations living near airports. A French study of 1244 subjects, part of the DEBATS (Discussion on the health effect of aircraft noise) research programme, showed that a 10 dB rise in aircraft noise levels increased the incidence of HTN by 1.36 times [18].

A positive dose-response relationship also exists between noise exposure in workplace environments and HTN risk. A meta-analysis of 23 studies published between 1977 and 2019 showed that workers chronically exposed to noise levels 80–85 dB had a 1.77-fold significantly higher risk of HTN relative to the comparison group (workers with average daily noise exposure levels ≤80 dB), whereas the hazard was more than three times greater for those with a noise exposure of >85 dB. Of note, employees with an occupational noise exposure of 90 dB for five years, 85 dB for 15–20 years or 83 dB for 20–30 years had about twice risk of developing HTN. The vast majority of participants were employees in noise-exposed factories, oil or minerals mining

industry or gas industry, air force pilots, power plant workers and shipyard workers. As it concerns sex differences, HTN risk did not differ between men and women, but data is limited in this field given that the aforementioned working populations are predominately male [19].

Nocturnal loud noise might be even more detrimental than daytime one. Noise at night leads to sleep disturbances and release of stress hormones, that can activate the sympathetic nervous system preventing dipping [9, 13]. The HYENA (HTN and Exposure to Noise near Airport) study included 4861 patients surrounding at least 5 years near one of 6 major European airports and found that a 10 dB increase in nighttime aircraft noise was correlated with a 1.14-fold higher risk of HTN. However, there was no significant exposure–response relationship for daytime noise. Men and those exposed to >65 dB level of sound were more susceptible to BP elevation [20]. The positive association between nighttime aircraft noise and HTN risk was also supported by 2 recently published nationwide prospective studies of female nurses free of HTN at study baseline. Among 63 229 participants in the first study and 98 880 participants in the second one, 33 190 and 28 255 new HTN cases were recorded, respectively [21].

The correlation between environmental noise and BP has also been studied in pediatric populations. A meta-analysis of 13 studies including approximately 8700 children found that a 5 dB increase in road traffic noise levels at kindergarten/school increased the systolic and diastolic BP by 0.48 mm Hg and 0.22 mm Hg, respectively. However, there was high heterogeneity and methodological issues in the primary studies that further flattened this association [22].

Road traffic noise seems to increase the risk of pre-eclampsia and hypertensive disorders in pregnancy as well. A Danish study of about 73 000 pregnant women without a known history of HTN showed that a 10 dB greater road traffic noise was correlated with a 10% higher risk of pre-eclampsia and 8% higher risk of pregnancy-related hypertensive disorders [23]. Likewise, exposure to noise levels 80-85 dB at workplace during pregnancy was associated with a 1.10-fold superior risk of hypertensive disorders and a 1.11-fold greater risk of preeclampsia [24].

Data regarding the effect of antihypertensive treatment in noise induced HTN is limited. An experimental study including mice that were receiving the beta blocker propranolol or the alpha blocker phenoxybenzamine and exposed to loud noise during the last four days of the drug administration showed that pharmacological interventions could prevent the noise induced endothelial dysfunction and oxidative stress. However, no BP improvement was recorded [25]. Alpha and/or beta blockers antagonizing the α and β adrenergic receptors to which catecholamines

link might be promising drugs for the mitigation of the adverse noise effects, but further research is required in this field.

Air pollution

Air pollution is now considered a key triggering factor for cardiovascular morbidity and mortality. According to the WHO, ambient air contamination has been identified as the 13th leading cause of mortality worldwide [13]. Ranked 9th among widely recognized modifiable risk factors, above low physical activity, dyslipidemia, dietary factors and drug use, it accounts for 6% of all-cause and all-age deaths every year [26]. In this context, the World Heart Federation, the American College of Cardiology, the American Heart Association and the European Society of Cardiology released official statements on the impact of air pollution on cardiovascular health [26, 27].

Air pollution is defined as the contamination of the ambient or indoor environment by any biological, chemical or physical agent that changes the natural composition of the atmosphere. The main outdoor air pollution sources include road traffic, emissions from industries, garbage, large destructive fires that spread quickly and indoor sources of air pollution. Household air pollution is generated by the burning of solid fuel sources, such as wood, crop waste, charcoal, coal and dung, for cooking and heating, the chemicals and solvents contained in many cleaning products, the mold, the air-conditioning systems and the second-hand smoke [13, 28].

Important pollutants that are implicated in cardiovascular risk are particulate matters (PM), ozone (O_3), nitrogen dioxide (NO_2), carbon monoxide (CO), sulfur dioxide (SO_2) and volatile organic compounds (including benzene). PM consists of particles that differ in size and composition and are classified according to the molecular size in three groups: coarse PM (PM10, diameter ranging from 2.5 to 10 μm), fine PM (PM2.5, diameter ranging from 0.1 to 2.5 μm) and ultrafine PM (PM0.1, diameter below 0.1 μm) [26]. In 2021 the WHO published specific recommendations on the air quality guideline levels for the pollutants PM2.5, PM10, ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide [29] (Table 1).

There is mounting evidence supporting that exposure to PM2.5, black carbon and other pollutants is capable of altering arterial BP [26]. A rise in outdoor PM2.5 by 10 mg/m^3 has been correlated with an increase in BP by 1 to 3 mm Hg. Interestingly, longer-term exposure might contribute to the development of chronic HTN *per se* [26, 30]. The three main pathophysiological mechanisms of BP elevation due to air pollution are: (1) autonomic nervous system imbalance and

sympathetic overactivity, (2) oxidative stress and endothelial dysfunction, (3) release of pro-inflammatory mediators and inflammation and (4) activation of prothrombotic pathways [31] (Table 2).

A large meta-analysis of 100 studies including about 0.7 million participants from 16 countries showed the positive association between four ambient air pollutants (PM2.5, PM10, SO_2, NO_2) with HTN and BP levels and this correlation was stronger in Asians, North Americans, males and regions with higher pollutant levels. Subgroup analysis by age showed that while the long-term effects were greater in the elderly population, the short-term effects were larger in the young population [32]. The PURE study, including data from 21 low-, middle- and high-income countries, showed that 3-year outdoor PM2.5 exposure was somewhat associated with a greater incidence of HTN and for every 10 $\mu g/m^3$ rise in PM2.5 the HTN hazard increased by 4%. Notably, PM2.5 concentration >62 $\mu g/m^3$ was correlated with 36% greater HTN risk compared to PM2.5 concentration <14 $\mu g/m^3$ [33]. A prospective study in India supported the temporal nature of the exposure-response relationship showing that 1-, 1.5- and 2-year exposure increased the risk of HTN by 53%, 59%, and 16%, respectively. The observed effects were larger in subjects with waist-hip ratio >0.95 [34]. The positive correlation between long-term exposure to outdoor PM2.5 and its chemical components (sulfate, nitrate, ammonium, organic matter, black carbon) with HTN incidence was also confirmed in a recent study of 8258 subjects from the WHO Study on Global Ageing and Adult Health that were followed up for a mean period of 7.13 years. According to this study, each increase in PM2.5 by 17.60 $\mu g/m^3$ was correlated with a 17% greater risk of HTN. This association was stronger in hypertensives, males, rural dwellers, smokers and users of unclean cooking fuel. Similarly, every rise in sulfate by 2.99 $\mu g/m^3$, nitrate by 4.23 $\mu g/m^3$, ammonium by 3.01 $\mu g/m^3$, organic matter by 3.62 $\mu g/m^3$ and black carbon by 0.77 $\mu g/m^3$ further increased the risk of HTN by 14%, 12%, 15%, 21% and 19%, respectively [35].

Available evidence suggests a link between exposure to increased ambient O_3 levels and BP values. A 10 $\mu g/m^3$ increase in O_3 levels in short term has been correlated with increases in diastolic and mean BP by 9.2 mm Hg and 7.2 mm Hg, respectively [36]. The Black Women's Health Study including 59 000 African American women showed that long-term exposure to higher O_3 levels increased the incidence of HTN by 9% [37]. The Beijing-Tianjin-Hebei Medical Examination-based Cohort demonstrated a non-linear correlation between O_3 exposure and incident HTN. Among 16 630 participants without a known history of HTN, about 10% developed

HTN during a follow-up period of 21 946 person-years. This correlation was stronger among males, individuals aged >44 years and those with body mass index $\geq$25kg/m^2 [38].

It can be assumed that approximately three billion people are exposed to household air pollution globally. A study that included subjects from the 2016 Albania Demographic Health and Survey showed that those exposed to household polluting fuels (e.g., kerosene, charcoal, wood etc.) were 17% more likely to develop HTN compared to those exposed to clean fuels (e.g., electricity, natural gas, biogas and liquid petroleum gas). Females, rural participants and those aged >24 years were more prone to pollution induced HTN [39]. Likewise, another study published the same year showed that household air pollution from cooking and heating with solid fuel (e.g., coal and biomass) was correlated with higher systolic and diastolic BP values. Also, a rise in PM2.5 exposure further increased the intima–media thickness and the total area of plaques in the carotid arteries underlining that air pollution might contribute to vascular damage as well. However, no association with arterial stiffness was recorded [40].

Exposure to air pollution before and during pregnancy also increases the risk of hypertensive disorders in pregnancy. A study of 130470 singleton pregnancies in Rome during 2014–2019 showed that exposure to PM2.5, PM10 and NO_2 was associated with a greater risk of preeclampsia [41]. A recent meta-analysis of 12 studies found that a 10 μg/m^3 rise in PM2.5 during pregnancy increased the risk of preeclampsia by 7% [42].

It seems that keeping well-controlled BP status and receiving angiotensin receptor blocker (ARB) treatment might attenuate the adverse impact of air pollution on BP. A study of 277 hypertensives at intermediate-to-high cardiovascular risk from China showed that for each 43.78 μg/m^3 increase in PM2.5 levels systolic BP increased by 0.85 mm Hg among those with uncontrolled HTN whereas no correlation was recorded in the group of subjects with controlled HTN. In addition, for every 43.78 μg/m^3 increase in PM2.5 levels systolic BP increased by 0.32 among those taking ARB and 1.53 among those not taking ARB [43].

However, given that air pollution increases the risk of dementia, antihypertensive medication adherence might be affected [44, 45]. A systematic review and meta-analysis of 15 studies showed that air pollution was correlated with a 13% greater hazard of dementia. This association was most robust after exposure to specific air pollutants, such as PM10, PM2.5, NO_2, CO and nitrogen oxides, but not O_3 [44].

Light pollution

Replacing traditional lighting by light-emitting diode (LED) lighting significantly reduced energy consumption, carbon emissions and energy costs. However, the outdoor artificially light at night (ALAN) increased, altering the night sky and contributing to light pollution. Light pollution increased by 49% from 1992 to 2017 and has been currently recognized as a key environmental problem. Almost 85% of global population lives under light-polluted environments [46]. Very common sources include the excessive or inappropriate outdoor lighting that impacts the naturally occurring darkness and results in extreme brightness and the light emitted from smartphones, monitors/laptops and TV screens [47].

From pathophysiologic point of view, nocturnal light exposure impairs the production of melatonin, a sleep-inducing hormone, disrupting the circadian rhythm and, thus, increasing the risk of HTN [48]. Moreover, the nighttime artificial light activates the sympathetic nervous system, leading to vasoconstriction, increased peripheral vascular resistance and high BP [49] (Table 2).

The first study that evaluated the connection between nighttime light exposure and HTN was published in 2014 and showed that nocturnal light exposure in home settings was associated with 3.3–4.7 mm Hg higher nighttime systolic BP and 2.3–2.9 mm Hg higher nighttime diastolic BP [50]. Likewise, a nationwide cross-sectional study from China found that ALAN intensity levels and HTN risk followed a non-linear pattern, resembling a reverse “L” shape. Indeed, participants exposed to the highest quartile of outdoor ALAN were correlated with a 1.31-fold greater risk of HTN. Moreover, after adjusting for potential confounding factors, greater outdoor ALAN intensity levels were correlated with a rise in systolic, diastolic and mean BP by 0.6 mm Hg, 0.9 mm Hg and 0.8 mm Hg, respectively [51]. Exercise seems to alleviates the negative effects of indoor nocturnal light exposure on BP and HTN and moderate to vigorous physical activity could prevent HTN risk in night shift workers [52]. Nonetheless, available evidence is limited and further research is needed in this field to establish the potential correlation of ALAN exposure with BP values and HTN incidence.

Environment and extreme weather conditions

Temperature. Extreme weather and events, such as heat waves, storms, hurricanes, blizzards, cyclones and floods, are an expression of climate change. The major characteristic of climate change is the variations in ambient temperature. BP shows a seasonal variation with higher levels

at lower environmental temperatures and lower levels at higher temperatures [53, 54]. Pathophysiologically, exposure to low temperatures rises sympathetic nervous activity resulting in vasoconstriction and endothelial dysfunction that increases BP values (Table 2) [55]. Acute change in weather conditions seems also to affect the hospital admissions due to hypertensive disease. A reduction in outdoor temperature by 5℃ can increase the HTN risk by 3% and *vice versa* [56].

The seasonal variation of BP might increase the incidence of HTN and reduce the control of the disease, potentially further increasing the cardiovascular risk [57]. An analysis of data from the Kadoorie Biobank study including 23 000 subjects from 10 diverse regions in China with a prior history of cardiovascular disease showed that mean systolic BP was 9 mm Hg greater in winter versus summer. In addition, at outdoor temperature >5℃ every 10℃ rise in environmental temperature was correlated with a 6.2 mm Hg higher systolic BP and every 10 mm Hg rise in systolic BP was correlated with 21% higher cardiovascular mortality. Interestingly, cardiovascular mortality followed the seasonal variation of BP, with 41% greater risk in winter [58]. The HOMED-BP (HTN Objective Treatment Based on Measurement by Electrical Devices of BP) trial demonstrated that among 1649 hypertensives the highest home BP measurements were recorded in mid-to-late January and the lower home BP measurements in mid-to-late July. Males, elderly and lightweight patients were more susceptible to large seasonal differences to home BP [59]. A retrospective cross-sectional study from Brazil showed a stronger inverse correlation between outdoor temperature and home BP measurements compared to office measurements. Moreover, regions with greater mean ambient temperature were characterized by higher incidence of normotension and white-coat HTN while regions with lower mean air temperature presented higher incidence of masked and sustained HTN [60].

A meta-analysis of 23 studies showed that a 1℃ decrease in outdoor temperature might increase systolic and diastolic BP by 0.26 mm Hg and 0.13 mm Hg, respectively. These changes in BP were more apparent in patients with cardiovascular disease [61]. In addition, hypertensives, and particularly those with higher BP values, seem to be more likely to BP alterations following ambient temperature changes compared to non-hypertensive counterparts. Interestingly, patients with a known history of HTN tend to respond more acutely to environmental temperature changes than subjects without HTN [62].

Even hourly and daily temperature variability is inversely correlated with BP [63]. A study of about 1900 patients from an outpatient unit in China showed that each 10℃ reduction in outdoor

temperature increased systolic BP by 0.84 mm Hg, diastolic BP by 0.56 mm Hg, mean BP by 1.38 mm Hg and pulse pressure by 0.66 mm Hg. These correlations were more apparent in women, individuals with higher body mass index, less education or aged 18-65 years, hypertensives and those with coronary artery disease [64].

BP can also be affected by weather patterns characterized by day-to-day or week-to-week changes in temperature, irrespective of seasonal changes. A study of approximately 16 000 patients with a known history of HTN from the Glasgow Blood Pressure Clinic in Scotland demonstrated that stable temperature was correlated with a 2% drop in BP while an extreme reduction in ambient temperature was correlated with 2.1% and 1.6% increase in systolic BP and diastolic BP, respectively [65].

Also, an increase in air temperature decreases the mean daytime BP and the morning surge in BP, but increases the nighttime BP. A study of almost 900 patients found that nocturnal BP was 2.4% greater in summer and 1.8% lower in winter whereas morning surge was 1.7 mm Hg lower in summer and 1.1 mm Hg greater in winter [66]. Likewise, the Nagahama study showed that the nocturnal BP dipping was greater in winter compared to summer (summer: −5.8 ± 7.8%, winter: −11.0 ± 7.7%) resulting in more risers (summer: 19.9%, winter: 7.8%) and non-dippers (summer: 51.4%, winter: 37.0%) in summer and more dippers (summer: 26.3%, winter: 43.1%) and extreme-dippers (summer: 2.4%, winter: 12.1%) in winter [67].

Temperature variability during preconception and in early pregnancy might affect BP levels and impair the incidence of severe hypertensive disorders in this sensitive subgroup of population [68, 69]. A large cohort of approximately 2 million pregnant women from China showed that very cold exposure before conception was correlated with a 1.22-fold greater risk of preeclampsia or eclampsia. Nonetheless, in the first half of pregnancy very cold exposure seemed to have the opposite effect [68]. Similarly, a study of almost 8000 pregnant women from Johannesburg in South Africa found that increased temperature in early pregnancy (gestational age: 2–5 week) heightened the risk of pre-eclampsia, eclampsia and HELLP (hemolysis, elevated liver enzymes, low platelets) while increased temperature in mid-late pregnancy tended to protect against severe hypertensive disorders. This alternative pattern in the early pregnancy might be due to several mechanisms including vascular disruption and placental insufficiency [69].

Humidity. The tropospheric specific humidity has raised since 1976 and this increase is due to human-induced climate change and global warming. It is expected that for each 1°C of

warming, the amount of water vapor in the saturated air increases by 7% [70]. Available evidence suggests that humidity has a detrimental effect on health, but data regarding cardiovascular system and particularly BP is limited.

A cohort study of almost 40 000 Chinese aged >65 years (51.3% being hypertensives) showed a non-linear inverse association between the average annual relative humidity and HTN. In particular, an increase in humidity in the past year by 1% decreased the likelihood of HTN by 0.4%, but these effects were more apparent in higher humidity levels (>70%) [71]. On the other hand, data from Project Viva, a prebirth cohort in Boston, showed that exposure to relative humidity in the early second trimester (weeks: 14–20) was correlated with a considerably greater risk of preeclampsia [72].

Figure 1 illustrates the main environmental factors which existing evidence supports that have a direct or an indirect effect on BP levels impairing the burden of HTN.

INTERVENTIONS STRATEGIES

The implementation of efficient mitigation strategies to control several environmental stressors and reduce their impact on BP is of paramount importance. It is supported that reducing air pollution exposure could prevent increases in BP levels. A large longitudinal study of approximately 134 000 Taiwanese adults found that every 5 $\mu g/m^3$ decline in PM2.5 was correlated with a lower hazard of HTN by 16% [73]. A randomized crossover study of 77 participants living close to major roadways showed that decreasing traffic air pollution exposure by using stand-alone high-efficiency particulate arrestance filtration was effective in reducing increases in systolic BP [74]. Moreover, an experimental study demonstrated that acute aircraft noise induced elevated BP was somewhat corrected within a 4-day period of noise cessation underlining the reversibility of noise induced effects during a noise-off period [75]. Hence, it is essential each individual adopts a friendlier environmental behavior, such as using public transports as much as possible and/or shift to active transport (walking and cycling). In addition, using renewable energy sources for cooking and heating, indoor portable air cleaners, air-conditioners with aeration system and ventilation systems would reduce air pollution in indoor spaces [76]. Also, minimizing nighttime artificial light exposure might help lower BP [77].

CONCLUSION

Environmental degradation is a modern, yet often under-recognized, risk factor of HTN increasing the incidence of the disease and complicating its treatment. Exposure to air, noise or light pollution is linked to a greater incidence of HTN while extreme weather patterns characterized by high temperature and/or high humidity are correlated with lower BP values. In light of this evidence, strategies to reduce exposure to environmental factors and prevent environmental degradation are required.

Article information

Conflict of interest: None declared.

Funding: None.

Open access: This article is available in open access under the Creative Common Attribution International (CC BY) license, which allows copying, distributing, and transmitting work, adapting work, and making commercial use of the work under the condition that the user must attribute the work in the manner specified by the author or licensor (but not in any way that suggests they endorse the user or their use of the work).

REFERENCES

1. Prüss-Ustün A, Wolf J, Corvalán C, et al. Preventing Disease through Healthy Environments, a global assessment of the burden of disease from environmental risks. Geneva, Switzerland: World Health Organization, 2016.
2. Blaustein JR, Quisel MJ, Hamburg NM, et al. Environmental impacts on cardiovascular health and biology: An overview. Circ Res. 2024; 134(9): 1048–1060, doi: 10.1161/CIRCRESAHA.123.323613, indexed in Pubmed: 38662864.
3. Fuchs FD, Whelton PK. High blood pressure and cardiovascular disease. Hypertension. 2020; 75(2): 285–292, doi: 10.1161/HYPERTENSIONAHA.119.14240, indexed in Pubmed: 31865786.
4. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. Nat Rev Nephrol. 2020; 16(4): 223–237, doi: 10.1038/s41581-019-0244-2, indexed in Pubmed: 32024986.
5. McEvoy JW, McCarthy CP, Bruno RM, et al. 2024 ESC Guidelines for the management of elevated blood pressure and hypertension. Eur Heart J. 2024; 45(38): 3912–4018, doi: 10.1093/eurheartj/ehae178, indexed in Pubmed: 39210715.

6. World Health Organization (WHO). Repository of systematic reviews on interventions in environment, climate change and health. 2024.
7. Shrikhande S, Wolf J, Vert C, et al. World Health Organization repository of systematic reviews on interventions in environment, climate change and health: A new resource for decision makers, intervention implementers, and researchers. Environ Health. 2024; 23(1): 88, doi: 10.1186/s12940-024-01105-y, indexed in Pubmed: 39434138.
8. Jarosińska D, Héroux MÈ, Wilkhu P, et al. Development of the WHO environmental noise guidelines for the European region: An introduction. Int J Environ Res Public Health. 2018; 15(4), doi: 10.3390/ijerph15040813, indexed in Pubmed: 29677170.
9. Münzel T, Gori T, Babisch W, et al. Cardiovascular effects of environmental noise exposure. Eur Heart J. 2014; 35(13): 829–836, doi: 10.1093/eurheartj/ehu030, indexed in Pubmed: 24616334.
10. Van Gerven PWM, Vos H, Van Boxtel MPJ, et al. Annoyance from environmental noise across the lifespan. J Acoust Soc Am. 2009; 126(1): 187–194, doi: 10.1121/1.3147510, indexed in Pubmed: 19603876.
11. World Health Organization. Compendium of WHO and other UN guidance on health and environment, 2022 update. https://www.who.int/publications/i/item/WHO-HEP-ECH-EHD-22.01 (January 2, 2025).
12. Chen F, Fu W, Shi O, et al. Impact of exposure to noise on the risk of hypertension: A systematic review and meta-analysis of cohort studies. Environ Res. 2021; 195: 110813, doi: 10.1016/j.envres.2021.110813, indexed in Pubmed: 33545125.
13. Sanidas E, Papadopoulos DP, Grassos H, et al. Air pollution and arterial hypertension. A new risk factor is in the air. J Am Soc Hypertens. 2017; 11(11): 709–715, doi: 10.1016/j.jash.2017.09.008, indexed in Pubmed: 28989071.
14. Münzel T, Sørensen M. Noise pollution and arterial hypertension. Eur Cardiol. 2017; 12(1): 26–29, doi: 10.15420/ecr.2016:31:2, indexed in Pubmed: 30416547.
15. Rojek M, Rajzer MW, Wojciechowska W, et al. Relationship among long-term aircraft noise exposure, blood pressure profile, and arterial stiffness. J Hypertens. 2019; 37(7): 1350–1358, doi: 10.1097/HJH.0000000000002060, indexed in Pubmed: 30747795.
16. Wojciechowska W, Januszewicz A, Drożdż T, et al. Blood pressure and arterial stiffness in association with aircraft noise exposure: Long-term observation and potential effect of

COVID-19 lockdown. Hypertension. 2022; 79(2): 325–334, doi: 10.1161/HYPERTENSIONAHA.121.17704, indexed in Pubmed: 34657440.

17. Kempen Ev, Casas M, Pershagen G, et al. WHO environmental noise guidelines for the European region: A systematic review on environmental noise and cardiovascular and metabolic effects: A summary. Int J Environ Res Public Health. 2018; 15(2): 379, doi: 10.3390/ijerph15020379, indexed in Pubmed: 29470452.
18. Kourieh A, Giorgis-Allemand L, Bouaoun L, et al. Incident hypertension in relation to aircraft noise exposure: Results of the DEBATS longitudinal study in France. Occup Environ Med. 2022; 79(4): 268–276, doi: 10.1136/oemed-2021-107921, indexed in Pubmed: 35046099.
19. Bolm-Audorff U, Hegewald J, Pretzsch A, et al. Occupational noise and hypertension risk: A systematic review and meta-analysis. Int J Environ Res Public Health. 2020; 17(17): 6281, doi: 10.3390/ijerph17176281, indexed in Pubmed: 32872306.
20. Jarup L, Babisch W, Houthuijs D, et al. Hypertension and exposure to noise near airports: the HYENA study. Environ Health Perspect. 2008; 116(3): 329–333, doi: 10.1289/ehp.10775, indexed in Pubmed: 18335099.
21. Peters JL, Grady ST, Laden F, et al. Long-term nighttime aircraft noise exposure and risk of hypertension in a prospective cohort of female nurses. Int J Hyg Environ Health. 2025; 263: 114457, doi: 10.1016/j.ijheh.2024.114457, indexed in Pubmed: 39270405.
22. Dzhambov AM, Dimitrova DD. Children's blood pressure and its association with road traffic noise exposure — A systematic review with meta-analysis. Environ Res. 2017; 152: 244–255, doi: 10.1016/j.envres.2016.10.024, indexed in Pubmed: 27810682.
23. Pedersen M, Halldorsson TI, Olsen SF, et al. Impact of road traffic pollution on pre-eclampsia and pregnancy-induced hypertensive disorders. Epidemiology. 2017; 28(1): 99–106, doi: 10.1097/EDE.0000000000000555, indexed in Pubmed: 27648591.
24. Lissåker CT, Gustavsson P, Albin M, et al. Occupational exposure to noise in relation to pregnancy-related hypertensive disorders and diabetes. Scand J Work Environ Health. 2021; 47(1): 33–41, doi: 10.5271/sjweh.3913, indexed in Pubmed: 32783066.
25. Kuntić M, Kuntić I, Zheng J, et al. Interventions by cardiovascular drugs against aircraft noise-induced cardiovascular oxidative stress and damage. Antioxidants (Basel). 2025; 14(1): 59, doi: 10.3390/antiox14010059, indexed in Pubmed: 39857393.

26. Newby DE, Mannucci PM, Tell GS, et al. Expert position paper on air pollution and cardiovascular disease. Eur Heart J. 2015; 36(2): 83–93b, doi: 10.1093/eurheartj/ehu458, indexed in Pubmed: 25492627.
27. Brauer M, Casadei B, Harrington RA, et al. Taking a stand against air pollution-the impact on cardiovascular disease: A joint opinion from the World Heart Federation, American College of Cardiology, American Heart Association, and the European Society of Cardiology. Circulation. 2021; 143(14): e800–e804, doi: 10.1161/CIRCULATIONAHA.120.052666, indexed in Pubmed: 33506685.
28. Caldeira D, Franco F, Bravo Baptista S, et al. Air pollution and cardiovascular diseases: A position paper. Rev Port Cardiol. 2022; 41(8): 709–717, doi: 10.1016/j.repc.2022.05.006, indexed in Pubmed: 36073271.
29. WHO global air quality guidelines: Particulate matter (PM(25) and PM(10)), ozone, nitrogen dioxide, sulfur dioxide and carbon monoxide. WHO Guidelines approved by the Guidelines Review Committee. Geneva, 2021.
30. Rajagopalan S, Al-Kindi SG, Brook RD. Air pollution and cardiovascular disease: JACC State-of-the-Art Review. J Am Coll Cardiol. 2018; 72(17): 2054–2070, doi: 10.1016/j.jacc.2018.07.099, indexed in Pubmed: 30336830.
31. Hahad O, Rajagopalan S, Lelieveld J, et al. Noise and air pollution as risk factors for hypertension: Part II — pathophysiologic insight. Hypertension. 2023; 80(7): 1384–1392, doi: 10.1161/HYPERTENSIONAHA.123.20617, indexed in Pubmed: 37073733.
32. Yang BY, Qian Z, Howard SW, et al. Global association between ambient air pollution and blood pressure: A systematic review and meta-analysis. Environ Pollut. 2018; 235: 576–588, doi: 10.1016/j.envpol.2018.01.001, indexed in Pubmed: 29331891.
33. Arku RE, Brauer M, Ahmed SH, et al. Long-term exposure to outdoor and household air pollution and blood pressure in the Prospective Urban and Rural Epidemiological (PURE) study. Environ Pollut. 2020; 262: 114197, doi: 10.1016/j.envpol.2020.114197, indexed in Pubmed: 32146361.
34. Prabhakaran D, Mandal S, Krishna B, et al. Exposure to particulate matter is associated with elevated blood pressure and incident hypertension in urban India. Hypertension. 2020; 76(4): 1289–1298, doi: 10.1161/HYPERTENSIONAHA.120.15373, indexed in Pubmed: 32816598.

35. Fu L, Guo Y, Zhu Q, et al. Effects of long-term exposure to ambient fine particulate matter and its specific components on blood pressure and hypertension incidence. Environ Int. 2024; 184: 108464, doi: 10.1016/j.envint.2024.108464, indexed in Pubmed: 38324927.
36. Song J, Zhu J, Tian G, et al. Short time exposure to ambient ozone and associated cardiovascular effects: A panel study of healthy young adults. Environ Int. 2020; 137: 105579, doi: 10.1016/j.envint.2020.105579, indexed in Pubmed: 32086080.
37. Coogan PF, White LF, Yu J, et al. Long-Term exposure to NO2 and ozone and hypertension incidence in the black women's health study. Am J Hypertens. 2017; 30(4): 367–372, doi: 10.1093/ajh/hpw168, indexed in Pubmed: 28096146.
38. Hu S, Xu X, Li C, et al. Long-term exposure to ambient ozone at workplace is positively and non-linearly associated with incident hypertension and blood pressure: longitudinal evidence from the Beijing-Tianjin-Hebei medical examination cohort. BMC Public Health. 2023; 23(1): 2011, doi: 10.1186/s12889-023-16932-w, indexed in Pubmed: 37845647.
39. Abba MS, Nduka CU, Anjorin S, et al. Household air pollution and high blood pressure: a secondary analysis of the 2016 albania demographic health and survey dataset. Int J Environ Res Public Health. 2022; 19(5): 2611, doi: 10.3390/ijerph19052611, indexed in Pubmed: 35270304.
40. Kanagasabai T, Xie W, Yan Li, et al. Household air pollution and blood pressure, vascular damage, and subclinical indicators of cardiovascular disease in older Chinese adults. Am J Hypertens. 2022; 35(2): 121–131, doi: 10.1093/ajh/hpab141, indexed in Pubmed: 34505873.
41. Pedersen M, Nobile F, Stayner LT, et al. Ambient air pollution and hypertensive disorders of pregnancy in Rome. Environ Res. 2024; 251(Pt 1): 118630, doi: 10.1016/j.envres.2024.118630, indexed in Pubmed: 38452913.
42. Gogna P, Villeneuve PJ, Borghese MM, et al. An exposure-response meta-analysis of ambient PM during pregnancy and preeclampsia. Environ Res. 2022; 210: 112934, doi: 10.1016/j.envres.2022.112934, indexed in Pubmed: 35150719.
43. Lin Z, Wang X, Liu F, et al. Impacts of short-term fine particulate matter exposure on blood pressure were modified by control status and treatment in hypertensive patients. Hypertension. 2021; 78(1): 174–183, doi: 10.1161/HYPERTENSIONAHA.120.16611, indexed in Pubmed: 34058854.

44. Tang J, Chen A, He F, et al. Association of air pollution with dementia: a systematic review with meta-analysis including new cohort data from China. Environ Res. 2023; 223: 115048, doi: 10.1016/j.envres.2022.115048, indexed in Pubmed: 36529331.
45. El-Saifi N, Moyle W, Jones C, et al. Medication adherence in older patients with dementia: A systematic literature review. J Pharm Pract. 2018; 31(3): 322–334, doi: 10.1177/0897190017710524, indexed in Pubmed: 28539102.
46. Falchi F, Cinzano P, Duriscoe D, et al. The new world atlas of artificial night sky brightness. Sci Adv. 2016; 2(6): e1600377, doi: 10.1126/sciadv.1600377, indexed in Pubmed: 27386582.
47. The Lancet Regional Health-Europe. Shedding light on light pollution. Lancet Reg Health Eur. 2023; 31: 100710, doi: 10.1016/j.lanepe.2023.100710, indexed in Pubmed: 37622167.
48. Bonanni A, Basile M, Montone RA, et al. Impact of the exposome on cardiovascular disease. Eur Heart J Suppl. 2023; 25(Suppl B): B60–B64, doi: 10.1093/eurheartjsupp/suad069, indexed in Pubmed: 37091631.
49. Mason IC, Grimaldi D, Reid KJ, et al. Light exposure during sleep impairs cardiometabolic function. Proc Natl Acad Sci U S A. 2022; 119(12): e2113290119, doi: 10.1073/pnas.2113290119, indexed in Pubmed: 35286195.
50. Obayashi K, Saeki K, Kurumatani N. Association between light exposure at night and insomnia in the general elderly population: The HEIJO-KYO cohort. Chronobiol Int. 2014; 31(9): 976–982, doi: 10.3109/07420528.2014.937491, indexed in Pubmed: 25025617.
51. Wang R, Hu X, Ou Y, et al. Association between outdoor light at night and hypertension and high-normal blood pressure: A nationwide cross-sectional study among Chinese adults. J Clin Hypertens (Greenwich). 2024; 26(2): 134–144, doi: 10.1111/jch.14760, indexed in Pubmed: 38192039.
52. Xu YX, Zhou Yi, Huang Y, et al. Physical activity alleviates negative effects of bedroom light pollution on blood pressure and hypertension in Chinese young adults. Environ Pollut. 2022; 313: 120117, doi: 10.1016/j.envpol.2022.120117, indexed in Pubmed: 36087897.
53. Ebi KL, Vanos J, Baldwin JW, et al. Extreme weather and climate change: Population health and health system implications. Annu Rev Public Health. 2021; 42: 293–315, doi: 10.1146/annurev-publhealth-012420-105026, indexed in Pubmed: 33406378.

54. Stergiou GS, Palatini P, Modesti PA, et al. Seasonal variation in blood pressure: Evidence, consensus and recommendations for clinical practice. Consensus statement by the European Society of Hypertension Working Group on Blood Pressure Monitoring and Cardiovascular Variability. J Hypertens. 2020; 38(7): 1235–1243, doi: 10.1097/HJH.0000000000002341, indexed in Pubmed: 31990898.
55. Park S, Kario K, Chia YC, et al. The influence of the ambient temperature on blood pressure and how it will affect the epidemiology of hypertension in Asia. J Clin Hypertens (Greenwich). 2020; 22(3): 438–444, doi: 10.1111/jch.13762, indexed in Pubmed: 31851429.
56. Bauer F, Lindtke J, Seibert F, et al. Impact of weather changes on hospital admissions for hypertension. Sci Rep. 2022; 12(1): 5716, doi: 10.1038/s41598-022-09644-5, indexed in Pubmed: 35383236.
57. Su D, Du H, Zhang X, et al. Season and outdoor temperature in relation to detection and control of hypertension in a large rural Chinese population. Int J Epidemiol. 2014; 43(6): 1835–1845, doi: 10.1093/ije/dyu158, indexed in Pubmed: 25135908.
58. Yang L, Li L, Lewington S, et al. Outdoor temperature, blood pressure, and cardiovascular disease mortality among 23 000 individuals with diagnosed cardiovascular diseases from China. Eur Heart J. 2015; 36(19): 1178–1185, doi: 10.1093/eurheartj/ehv023, indexed in Pubmed: 25690792.
59. Hanazawa T, Asayama K, Watabe D, et al. Seasonal variation in self-measured home blood pressure among patients on antihypertensive medications: HOMED-BP study. Hypertens Res. 2017; 40(3): 284–290, doi: 10.1038/hr.2016.133, indexed in Pubmed: 27682651.
60. Barbosa ECD, Feitosa ADM, Sentalin MVR, et al. Impact of environmental temperature on blood pressure phenotypes: A nationwide home blood pressure monitoring study. Eur J Prev Cardiol. 2024; 31(6): e35–e37, doi: 10.1093/eurjpc/zwad387, indexed in Pubmed: 38091969.
61. Wang Q, Li C, Guo Y, et al. Environmental ambient temperature and blood pressure in adults: A systematic review and meta-analysis. Sci Total Environ. 2017; 575: 276–286, doi: 10.1016/j.scitotenv.2016.10.019, indexed in Pubmed: 27750133.
62. Xu Y, Han Y, Chen Wu, et al. Susceptibility of hypertensive individuals to acute blood pressure increases in response to personal-level environmental temperature decrease.

Environ Int. 2024; 185: 108567, doi: 10.1016/j.envint.2024.108567, indexed in Pubmed: 38460242.

63. Zhu W, Liu Y, Zhang L, et al. Ambient temperature variability and blood pressure in a prospective cohort of 50,000 Chinese adults. J Hum Hypertens. 2023; 37(9): 818–827, doi: 10.1038/s41371-022-00768-9, indexed in Pubmed: 36257970.
64. Fan P, Xue X, Hu J, et al. Ambient temperature and ambulatory blood pressure: An hourly-level, longitudinal panel study. Sci Total Environ. 2023; 864: 160854, doi: 10.1016/j.scitotenv.2022.160854, indexed in Pubmed: 36521627.
65. Aubinière-Robb L, Jeemon P, Hastie CE, et al. Blood pressure response to patterns of weather fluctuations and effect on mortality. Hypertension. 2013; 62(1): 190–196, doi: 10.1161/HYPERTENSIONAHA.111.00686, indexed in Pubmed: 23648702.
66. Sheng CS, Cheng YB, Wei FF, et al. Diurnal blood pressure rhythmicity in relation to environmental and genetic cues in untreated referred patients. Hypertension. 2017; 69(1): 128–135, doi: 10.1161/HYPERTENSIONAHA.116.07958, indexed in Pubmed: 27821618.
67. Tabara Y, Matsumoto T, Murase K, et al. Seasonal variation in nocturnal home blood pressure fall: The Nagahama study. Hypertens Res. 2018; 41(3): 198–208, doi: 10.1038/s41440-017-0003-3, indexed in Pubmed: 29382900.
68. Xiong T, Chen P, Mu Yi, et al. Association between ambient temperature and hypertensive disorders in pregnancy in China. Nat Commun. 2020; 11(1): 2925, doi: 10.1038/s41467-020-16775-8, indexed in Pubmed: 32522990.
69. Part C, le Roux J, Chersich M, et al. Ambient temperature during pregnancy and risk of maternal hypertensive disorders: A time-to-event study in Johannesburg, South Africa. Environ Res. 2022; 212(Pt D): 113596, doi: 10.1016/j.envres.2022.113596, indexed in Pubmed: 35661733.
70. Stocker TF, Qin D, Plattner GK, Tignor M, Allen SK, Boschung J, Nauels A, Xia Y, Bex V, Midgley PM. IPCC, 2013: Climate Change 2013: The Physical Science Basis. Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge University Press, Cambridge, United Kingdom and New York, NY, US 2013.

71. Chen H, Zhang X. Influences of temperature and humidity on cardiovascular disease among adults 65 years and older in China. Front Public Health. 2022; 10: 1079722, doi: 10.3389/fpubh.2022.1079722, indexed in Pubmed: 36699927.
72. Howe CM, Coull BA, Papatheodorou S, et al. Relative humidity, temperature, and hypertensive disorders of pregnancy: Findings from the Project Viva cohort. Environ Res. 2024; 257: 119211, doi: 10.1016/j.envres.2024.119211, indexed in Pubmed: 38782342.
73. Bo Y, Guo C, Lin C, et al. Dynamic changes in long-term exposure to ambient particulate matter and incidence of hypertension in adults. Hypertension. 2019; 74(3): 669–677, doi: 10.1161/HYPERTENSIONAHA.119.13212, indexed in Pubmed: 31303109.
74. Hudda N, Eliasziw M, Hersey SO, et al. Effect of reducing ambient traffic-related air pollution on blood pressure: A randomized crossover trial. Hypertension. 2021; 77(3): 823–832, doi: 10.1161/HYPERTENSIONAHA.120.15580, indexed in Pubmed: 33486990.
75. Bayo Jimenez MT, Gericke A, Frenis K, et al. Effects of aircraft noise cessation on blood pressure, cardio- and cerebrovascular endothelial function, oxidative stress, and inflammation in an experimental animal model. Sci Total Environ. 2023; 903: 166106, doi: 10.1016/j.scitotenv.2023.166106, indexed in Pubmed: 37567316.
76. Sofia D, Gioiella F, Lotrecchiano N, et al. Mitigation strategies for reducing air pollution. Environ Sci Pollut Res Int. 2020; 27(16): 19226–19235, doi: 10.1007/s11356-020-08647-x, indexed in Pubmed: 32279263.
77. Kim M, Vu TH, Maas MB, et al. Light at night in older age is associated with obesity, diabetes, and hypertension. Sleep. 2023; 46(3): zsac130, doi: 10.1093/sleep/zsac130, indexed in Pubmed: 35729737.

Table 1. The recommended limits for the most common air pollutants and specific noise sources

Air pollutants	**Limit**
PM2.5	5 μg/m^3 (12-month exposure)
	15 μg/m^3 (24-hour exposure)
PM10	15 μg/m^3 (12-month exposure)
	45 μg/m^3 (24-hour exposure)
O_3	60 μg/m^3 (peak season[a])

	100 μg/m^3 (8-hour exposure)
NO_2	10 μg/m^3 (12-month exposure)
	25 μg/m^3 (24-hour exposure)
	200 μg/m^3 (1-hour exposure)
SO_2	40 μg/m^3 (24-hour exposure)
	500 μg/m^3 (10-minute exposure)
CO	4 mg/m^3 (24-hour exposure)
	10 mg/m^3 (8-hour exposure)
	35 mg/m^3 (1-hour exposure)
	100 mg/m^3 (15-minute exposure)
Noise	
Aircraft noise	<45 dB L_{den}[b]
	<40 dB L_{night}[c]
Road traffic noise	<53 dB L_{den}
	<45 dB L_{night}
Railway noise	<54 dB L_{den}
	<44 dB L_{night}
Wind turbine noise	<45 dB L_{den}

[a]The six consecutive months per year with the greatest 6-month running-average O_3 concentration. Away from the equator, this period corresponds to the warm season in a calendar year (northern hemisphere) or two calendar years (southern hemisphere). Close to the equator, peak season is usually identified by existing monitoring or modelling data. [b]Average day-evening-night sound level. [c]Average night sound level

Table 2. A summary of mechanisms behind hypertensive effects of environmental stressors

Noise pollution
• Increased sympathetic nervous activity and catecholamines levels • Release of cortisol further enhances the effect of catecholamines • Increased arterial stiffness and carotid-femoral pulse wave velocity
Air pollution
• Autonomic nervous system imbalance and sympathetic overactivity

• Oxidative stress and endothelial dysfunction • Release of pro-inflammatory mediators and inflammation • Activation of prothrombotic pathways
Light pollution
• Decreased deficiency levels and disrupted circadian rhythm • Increased sympathetic nervous activity
High temperature
• Sympathetic overactivity • Endothelial dysfunction

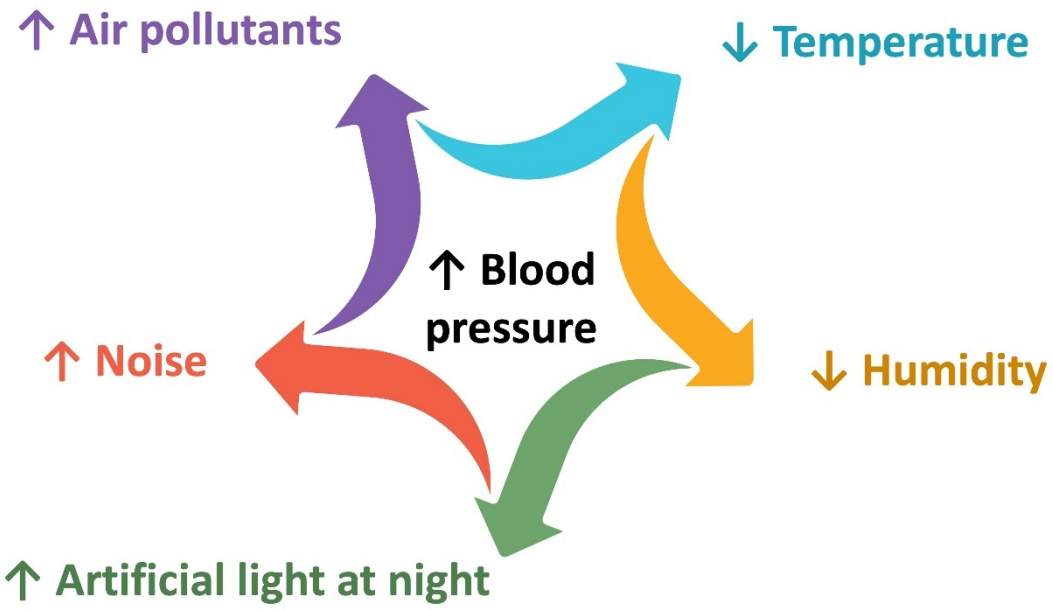

Figure 1. The main environmental stressors that might impair the burden of hypertension