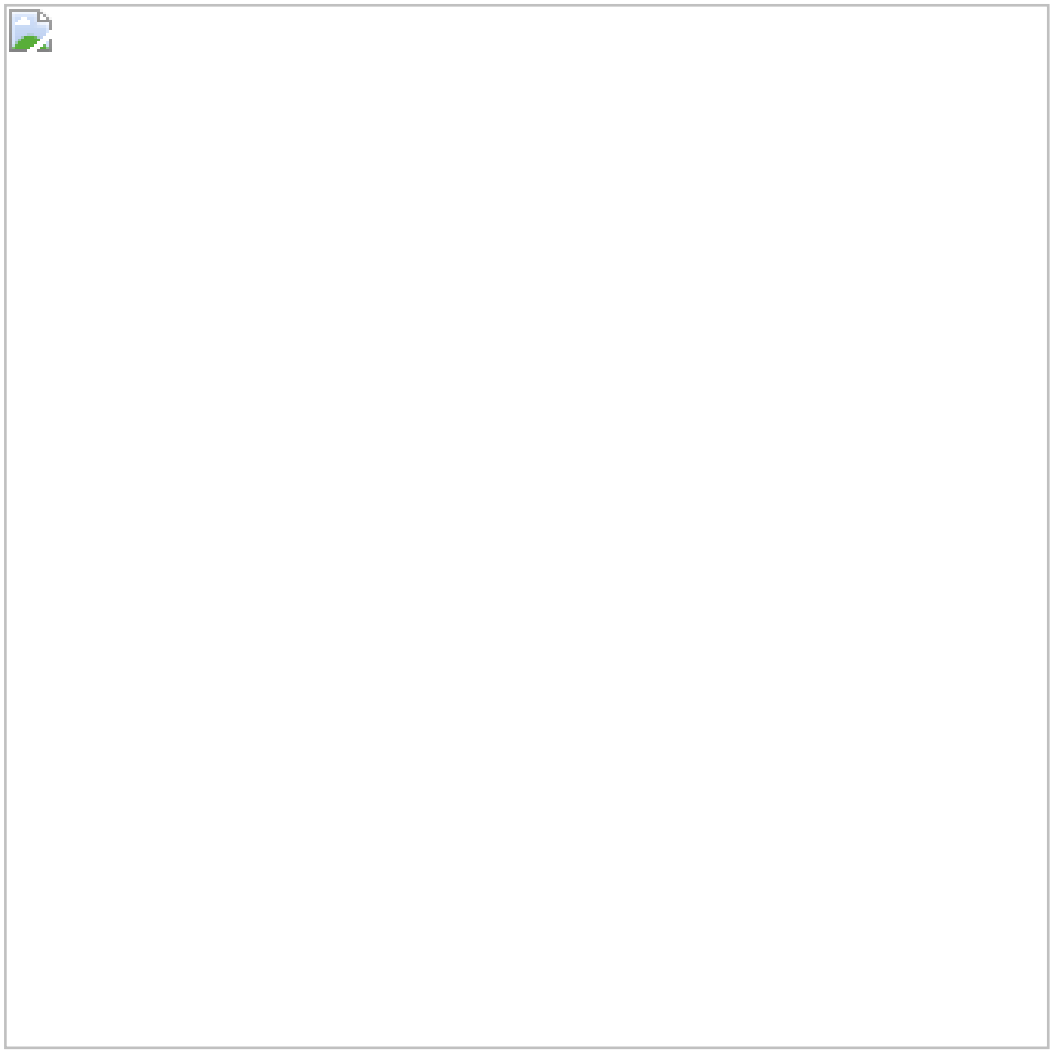

# The Cold Dusty Harmattan: A Season of Anguish for Cardiologists and Patients

Author: Okeahialam, Basil N.

Source: Environmental Health Insights, 10(1)

Published By: SAGE Publishing

URL: https://doi.org/10.1177/EHI.S38350

# The Cold Dusty Harmattan: A Season of Anguish for Cardiologists and Patients

Basil N. Okeahialam

Cardiology Sub-Unit 1, Department of Medicine, Jos University Teaching Hospital, Jos, Nigeria.

**ABSTRACT:** Human health and disease often demonstrate seasonal patterns. Knowledge of these aspects aids anticipation and planning. Numerous studies have shown that hypertension and cardiovascular diseases demonstrate a seasonal pattern. The Harmattan, the cold dusty season in Sub-Saharan Africa, is the season of greatest concern in this regard. In this commentary, the author draws on his and other researchers' studies to explain the grounds for onset and worsening of existing cardiovascular diseases. As implied in the title, it is a season that puts great strain on the cardiologist and the health system, as well as greater disease burden on the patient. This should be taken into consideration in planning and pooling of resources for effective patient management and mitigation of impact of disease.

**KEYWORDS:** Harmattan, season, cold, dust, cardiovascular diseases

**CITATION:** Okeahialam. The Cold Dusty Harmattan: A Season of Anguish for Cardiologists and Patients. *Environmental Health Insights* 2016:10 143–146 doi: 10.4137/EHI.S38350.

**TYPE:** Commentary

**RECEIVED:** June 07, 2016. **RESUBMITTED:** July 05, 2016. **ACCEPTED FOR PUBLICATION:** July 07, 2016.

**ACADEMIC EDITOR:** Timothy Kelley, Editor in Chief

**PEER REVIEW:** Three peer reviewers contributed to the peer review report. Reviewers' reports totaled 605 words, excluding any confidential comments to the academic editor.

**FUNDING:** Author discloses no external funding sources.

**COMPETING INTERESTS:** Author discloses no potential conflicts of interest.

**CORRESPONDENCE:** basokeam@yahoo.com



Paper subject to independent expert blind peer review. All editorial decisions made by independent academic editor. Upon submission manuscript was subject to anti-plagiarism scanning. Prior to publication all authors have given signed confirmation of agreement to article publication and compliance with all applicable ethical and legal requirements, including the accuracy of author and contributor information, disclosure of competing interests and funding sources, compliance with ethical requirements relating to human and animal study participants, and compliance with any copyright requirements of third parties. This journal is a member of the Committee on Publication Ethics (COPE). Provenance: the author was invited to submit this paper.

Published by Libertas Academica. Learn more about this journal.

## Introduction

The environment impacts human health, just as human activities impact the environment. This interaction between human health and the environment has long been known and can be direct or indirect.[1] In this commentary, an attempt is made to highlight the specific ways that the cold temperature and atmospheric dust pollution of the Harmattan season experienced in Jos, Nigeria, and some parts of Sub-Saharan Africa bring about new onset of cardiovascular diseases and worsen existing ones, increasing disease morbidity and mortality. As shown in some European studies, dust from the Sahara is the main global source of atmospheric mineral dust, with particulate matter (PM) levels significantly exceeding limit values.[2] Saharan dust outbreaks adversely affect weather, increasing daily mortality by 8.4%.[3] The PM 2.5 to 10 of dust matter has the greatest effect on health,[4] with the cardiovascular system bearing the brunt.[5]

The Harmattan season usually occurs between the end of November and mid March. Over the period, dry dusty northeasterly trade winds blow from the Sahara desert over the West African subregion to the Atlantic Ocean. As the wind passes over the desert, it collects fine dust particles (0.5–10 μm) and causes low temperatures. As shown in the study by Enete et al.[6], silicon makes up the bulk of the elemental content of the Harmattan dust. It can be so dense as to reduce radiation from the sun, reducing its attendant warmth, hence the associated cold temperature. During this time of the year, the sun is near the Tropic of Capricorn, farthest away from West Africa and the Northern Hemisphere, which also explains the cold temperatures. Without doubt, the Harmattan constitutes an environmental challenge.[7] In West Africa, morbidity rates from desert dust (which is worse during the Harmattan) are higher than those in other areas, and yet it is the least studied area in this regard.[8]

## The Harmattan and Cardiovascular Diseases

**Cold weather and low temperature.** Our experience in Jos, Nigeria, is that during the Harmattan period, blood pressure rises among hypertensives,[9] along with a rise in admissions for congestive cardiac failure and cerebrovascular accident (CVA).[10] This observation, albeit on hospital cohorts, can be extrapolated to the population. The burden of care on the cardiologist rises, constituting an additional burden, while readmission rates, higher morbidity, and reduced quality of life with attendant high economic burden constitute further burden for the patient. Occasionally, death results suddenly, leaving the patient in no position to tell his/her story.[11]

Particularly among temperature-sensitive subjects, during cold weather, mortality from hypertension is higher,[12] as blood pressure tends to rise. This has already been observed, as our own local experience[9,10] in Jos. This is thought to be a response to thermoregulatory vasoconstriction, which seeks to conserve core temperature.[13] Apart from this, exposure to cold increases the activity of the renin–angiotensin–aldosterone system activity, with resultant rise in blood pressure.[14] If the period of cold-induced hypertension is long, blood pressure

may not become normal again.[15] Also in cold ambient temperatures, sweating is reduced, leading to increased sodium loading, resulting in elevation of blood pressure. Other mechanistic explanations for rises in blood pressure as enunciated by Cuspidi et al.[16] include activation of the sympathetic autonomic nervous system and increased hemorheology with attendant rise in peripheral resistance. Additionally, the intensity of dust haze reduces the quantum of ultraviolet rays of the sun during Harmattan. This reduction results in low temperatures in the environment that reduces vitamin D3 and parathormone production with attendant hypertension.[17] This physiological change would naturally be more manifest if temperature variations are large, which as shown in the PAMELA study[18] increases blood pressure variability with a higher pre-awakening morning blood pressure surge. These perturbations significantly contribute to myocardial infarction (MI) and CVA, which arise during this season. They also create a dilemma for the cardiologist who does not know how to respond to these increases with drugs that could give rise to the problems when the weather warms.[12]

Heart failure (HF) admissions are also known to increase during the cold season.[10,19] This arises in the context of hypertension. The reduced sweating and insensible fluid loss that contribute to elevated blood pressure also result in fluid overload, resulting in HF. Those patients already in chronic HF are bound to decompensate due to elevation of blood pressure and its variability. The arrhythmogenicity trigger potential of cold weather[20,21] also results in acute HF or acute exacerbation of chronic stable HF. In cold weather, hemodynamic change in increased heart rate and total peripheral resistance with a fall in cardiac output result in acute pulmonary edema, especially in the background of hypertension or ventricular disease.[22] It is also likely that the causes of this seasonal variation go beyond temperature changes. Neuroendocrine and metabolic function changes have been reported to operate, especially with regard to thyroid and adrenal function.[23]

**Lifestyle.** There is a widely held albeit faulty view that it is beneficial to use alcohol when the weather is cold to keep warm. In our immediate environment, patronage of local brew parlours tends to rise during this period. Rather than warming the body, the ultimate effect of alcohol is hypothermia. Initially, users of alcohol may feel warm as cutaneous vessels dilate, but the long-term effect is egress of warmth from the core to the periphery. The alcohol used triggers arrhythmias and depresses the myocardium, the result of which is HF. Alcohol use and low temperature disrupt sleep and could thereby adversely impact cardiovascular health. Another related social habit that increased in cold weather is smoking, again erroneously for the purpose of keeping warm. Cigarette smoking through its effect on vessels, oxygenation of hemoglobin, and hemorheology as well as chronic bronchitis burden the heart, resulting in HF. Smoking has been shown to increase seasonal changes in blood pressure.[24] The low temperatures of the Harmattan season and the associated dust haze tend to keep people indoors. This is associated with physical inactivity that is longer over this period in West Africa because the nights are longer than the day, due to the position of the sun in the Southern Hemisphere. This promotes overeating with consequent obesity, which burdens the cardiovascular system.

**Infection.** In low temperatures, the respiratory tract is dried of mucus and the bare epithelium loses its first line of defense. There is a proneness to infections primarily viral, which increase platelet stickiness, thrombus formation, and hypercoagulability of the blood due to cytokines and other inflammatory factors elaborated. These increase morbidity and mortality.[25] In people with chronic bronchitis, the acute exacerbations caused by infections acutely upset the pulmonary vascular hemodynamics, placing a heavy burden on the heart. There is also a worsening of the airway disease with significant background low-grade inflammation. This accelerates atherosclerosis, increasing risk of MI, sudden cardiac death, and CVA. Again in cold weather, the lipid profile becomes atherogenic also increasing atherosclerosis.[26] Heating needs during the cold season usually lead to people trying to manipulate indoor climate conditions. This results in seasonal blood pressure and cardiovascular disease (CVD) morbidity and mortality being attenuated if not completely abolished.[16] In our environment, attempts to increase the indoor ambient temperatures lead to pollution through smoke emission by biomass fuel combustion, lamps, and stoves. The benefit of greater warmth is counteracted by the smoke that brings about cough, infection, and obstructive airway disease. The inflammation, secondary polycythemia, and pulmonary hypertension conjointly produce cardiovascular diseases.

**Atmospheric dust pollution.** The Harmattan is not only associated with low temperature, but it also involves pollution of the atmosphere by the dust-laden northeast trade winds blowing across the Sahara desert. The bulk of elemental content of the dust is silicon.[2] The size of the fine dust particles 0.5–10 μm diameter puts them in the PM10 particulate matter air pollutant group, which has already been shown by WHO to lead to multiple adverse health effects.[27] Inhalation of such pollutants results in airway inflammation, leukocyte migration, and cytokine release, which impose oxidative stress burden.[28] In humans, exposure to air pollution particles has been shown to cause significant acute increases in blood pressure. This creates de novo hypertension or worsens existing hypertension, which may even result in catastrophic events such as HF, CVA, and MI. Hypertensives with diabetes face a worse version of this.[29] These effects may not arise concurrently with the development of the atmospheric dust pollution where it is seasonal. There is usually a lag period, which has been shown in some studies to be a minimum of 15 days.[27] In some cases, it could be less, as short as one to three days.[30] Normally, the status of the patient in the pathogenetic process of the disease would determine how soon after exposure to dust pollution the disease would manifest. The inhalation of these particles in

the context of dry mucosa and ciliae disabled by cold results in infections, which adversely affect both the cardiovascular and respiratory systems. If the inhalation becomes sustained (many weeks in the Harmattan season), there is an increased risk of cardiorespiratory disease.[31] Recently, ambient air pollution has been linked to diabetes and obesity,[32] disease states that have a significant impact on the cardiovascular system. Atmospheric dust pollution in the Harmattan varies in quantum from day to day. However, this does not constitute a respite for the cardiorespiratory system because low-level silica exposure (under the acceptable limit) is still associated with increased risk of mortality from pulmonary and ischemic heart diseases.[33] In areas with high ambient atmospheric pollution (which may originate from industry and construction sources), surges in dust content brought about by dust worsen the dust's effect on health.[4] PM is also known to result in cardiac arrhythmias, which can precipitate HF in the context of cardiovascular disease.[34] Subclinical atherosclerosis has also been shown to result from PM ambient pollution and is reflected in carotid intima media thickness studies.[35]

The low temperature of the Harmattan and atmospheric dust pollution combine to produce an amplified deleterious effect on the cardiovascular system. Increased mortality as reported in the work by Zanobetti and Peters[36] has been linked to exposure to colder weather conditions. The result is de novo manifestation in this temporal pattern or seasonal rhythm of CVD and decompensation of stable cases. In our center, cardiovascular admissions on the medical service (Okeahialam and Dare, unpublished) increased from 23.2% in October 2015 (prior to Harmattan) to 33.4%, 30.3%, and 26.7%, respectively, for November 2015, December 2015, and January 2016 (during the Harmattan). Various complications of cardiovascular diseases that increase morbidity, and at times mortality, are commonplace. All these put a significant burden of care on the cardiologist and for the patient add an economic burden and impaired quality of life. To ameliorate this situation, cardiologists should take proactive steps to care for their patients during this season. There may be a need for public health education to discourage seasonal lifestyles that are adverse to good health and to encourage adoption of favorable lifestyles, including modification of indoor climate. These may not completely remove the risk (since the outdoor environment is beyond individual ability to modify), but should significantly mitigate the anguish of cardiologists and patients.

## Author Contributions

Conceived the concepts: BNO. Analyzed the data: BNO. Wrote the first draft of the manuscript: BNO. Developed the structure and arguments for the paper: BNO. Made critical revisions and approved final version: BNO. The author reviewed and approved of the final manuscript.

## REFERENCES


1. Remoundou K, Koundoun P. Environmental effects on public health: an economic perspective. *Int J Environ Res Public Health*. 2009;6(8):2160–78.
2. Karanasiou A, Moreno N, Moreno T, Viana M, de Leeuw F, Querol X. Health effects from Sahara dust episodes in Europe: literature review and research gaps. *Environ Int*. 2012;47:107–14.
3. Perez L, Tobias A, Querol X, et al. Coarse particles from Saharan dust and daily mortality. *Epidemiology*. 2008;19:800–7.
4. Tobias A, Perez L, Diaz J, et al. Short term effects of particulate matter on total mortality during Saharan dust outbreaks: a case-crossover analysis in Madrid (Spain). *Sci Total Environ*. 2011;412–3:386–9.
5. Mallone S, Stafoggia M, Faustini A, Gobbi GP, Marconi A, Forastiere F. Saharan dust and associations between particulate matter and daily mortality in Rome, Italy. *Environ Health Perspect*. 2011;119:1409–14.
6. Enete JC, Obienusi EA, Igu IN, Ayadiulo R. Harmattan dust: composition, characteristics and effects on soil fertility in Enugu, Nigeria. *Br J Appl Sci Technol*. 2012;2(1):72–81.
7. Ogunseitan OA. Harmattan haze and environmental health. (Editorial). *Afr J Environ Sci Technol*. 2007;1(4). Available at: http://www.academicjournals.org/AJEST.
8. DeLongueville F, Hountodji YC, Henry S, Ozer P. What do we know about effects of desert dust on air quality and human health in West Africa compared to other regions? *Sci Total Environ*. 2010;409:1–8.
9. Okeahialam BN. Seasonal variation of blood pressure in hypertensive patients studied in a cardiology clinic setting. *J Med Med Sci*. 2000;2(2):116–7.
10. Okeahialam BN, Zoakah AI. Climatological elements and hospital admission from congestive cardiac failure and cerebrovascular accident in Jos, Nigeria. *J Med Trop*. 2002;4(2):41–6.
11. Gerer Y, Jacobsen SJ, Killian JM, Weston SA, Roger VL. Seasonality and daily weather conditions in relation to myocardial infarction and sudden cardiac death in Olmsted County, Minnesota, 1979 to 2002. *J Am Coll Cardiol*. 2006;48:287–92.
12. Aubiniere-Robb L, Jeemon P, Hastie CE, et al. Blood pressure response to patterns of weather fluctuations and effects on mortality. *Hypertension*. 2013;62:190–6.
13. Greif R, Laciny S, Rajek A, Doufas AG, Sessler DI. Blood pressure response to thermoregulatory vasoconstriction during isoflurane and desflurane anesthesia. *Acta Anaesthesiol Scand*. 2003;47:847–52.
14. Sun Z, Cade R, Morales C. Role of central angiotensin II receptors in cold-induced hypertension. *Am J Hypertens*. 2002;15(1 pt 1):85–92.
15. Shechtman O, Papanek PE, Fegly MJ. Reversibility of cold-induced hypertension after removal of rats from cold. *Can J Physiol Pharmacol*. 1990;68:830–5.
16. Cuspidi C, Ochoa JE, Parato G. Seasonal variations in blood pressure: a complex phenomenon. (Editorial Comment). *J Hypertens*. 2012;30:1315–20. doi: 10.1097/HJH.0b013e.328355d7f9.
17. Rostard SG. Ultraviolet light may contribute to geographic and racial blood pressure differences. *Hypertension*. 1997;30(2 pt 1):150–6.
18. Sega R, Cesana G, Bombelli M, et al. Seasonal variations in home and ambulatory blood pressure in the PAMELA population. Pressione Arteriose Monitorate E Loro Associazioni. *J Hypertens*. 1998;16:1585–92.
19. Boulay F, Berthier F, Sisteron O, Gendreila Y, Gibelin P. Seasonal variation in chronic heart failure hospitalizations and mortality in France. *Circulation*. 1999;100:280–6.
20. Kupari M, Koskiren P. Seasonal variation in occurrence of acute atrial fibrillation and relation to air temperature and sale of alcohol. *Am J Cardiol*. 1990;15:1519–20.
21. Fries RP, Heisel AG, Jung JK, Schieffer HJ. Circannual variation of malignant ventricular tachyarrhythmias in patients with implantable cardioverter-defibrillators and either coronary artery disease or idiopathic dilated cardiomyopathy. *Am J Cardiol*. 1997;79:1194–7.
22. Wilmhurst PT, Nuri M, Crowther A, Webb-Pepploe MM. Cold induced pulmonary oedema in scuba divers and swimmers and subsequent development of hypertension. *Lancet*. 1989;1:62–5.
23. Nicolau GY, Hans E. Chronobiology of the endocrine system. *Endocrinology*. 1989;27:153–83.
24. Kristal-Boneh E, Harari G, Green MS. Seasonal changes in 24-h blood pressure and heart rate is greater among smokers than non-smokers. *Hypertension*. 1997;30(3 pt 1):436–41.
25. Keatings WR, Coleshaw SR, Cotter F, Mattock M, Murphy M, Chelliah R. Increase in platelet and red cell counts, blood viscosity and arterial pressure during mild surface cooling. Factors in mortality from coronary and cerebral thrombosis in winter. *Br Med J*. 1984;289:1405–8.
26. Ockene IS, Chiriboga DE, Stanek EJ 3rd, et al. Seasonal variation in serum cholesterol levels: treatment implications and possible mechanisms. *Arch Intern Med*. 2004;164:863–70.
27. Zuniga J, Tarajia M, Herrera V, Urriola W, Gomez B, Motta J. Assessment of the possible association of air pollutants $PM_{10}$, $O_3$, $NO_2$ with an increase in cardiovascular respiratory and diabetes mortality in Panama City. A 2003 to 2013 Data analysis. *Medicine*. 2016;95(2):e2464.

28. Moller P, Danielsen PH, Karottki DG, et al. Oxidative stress and inflammation generated DNA damage by exposure to air pollution particles. *Mutat Res Rev Mutat Res*. 2014;762:133–66.
29. Yitshak-Sade M, Kloog J, Liberty IF, et al. Air pollution and serum glucose levels. A population based study. *Medicine*. 2015;94:e1093.
30. Vodonos A, Friger M, Katra I, et al. Individual effect modifiers of dust exposure: effect on cardiovascular morbidity. *PLoS One*. 2015;10(9):e0137714. doi: 10.1371/journal.pone.0137714.
31. Brook RD, Rajagopalan S, Pope CA 3rd, et al. American Heart Association Council on Epidemiology and Prevention Council on the Kidney in Cardiovascular Disease and Council on Nutrition, Physical Activity and Metabolism. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation*. 2010;121:2331–78.
32. Eze IC, Schaffner E, Foraster M, et al. Long-term exposure to ambient air pollution and metabolic syndrome in adults. *PLoS One*. 2015;10:e0130337.
33. Liu Y, Rong Y, Steenland K, et al. Long-term exposure to crystalline silica and risk of heart disease mortality. *Epidemiology*. 2014;25:689–96.
34. Wang F, Jia X, Wang X, Zhao Y, Hao W. Particulate matter and atherosclerosis: a bibliometric analysis of original research articles published in 1972–2014. *BMC Public Health*. 2016;16:348.
35. Adar SD, Sheppard L, Vedal S, et al. Fine particulate air pollution and the progression of carotid intima media thickness study of atherosclerosis and air pollution. *PLoS Med*. 2013;10(4):e1001430.
36. Zanobetti A, Peters A. Disentangling interactions between atmospheric pollution and weather. *J Epidemiol Community Health*. 2015;69(7):613–5.