GAS FLARING AND CRUDE OIL CONTAMINANTS AS MODIFYERS OF BLOOD PRESSURE IN DELTA STATE

Abstract:

Several toxic effects of crude oil have been reported on human reproductive, respiratory, cardiovascular, and nervous systems. An instance is seen in the negative effect on fertility resulting from prolonged exposure to fumes from exhausts of vehicles. This study therefore investigated gender-specific changes in selected cardiovascular variables of residents of gas flaring and crude oil contaminated communities of Delta State, southern Nigeria. Two Hundred and Forty (240) subjects exposed to gas flaring and crude oil contamination (Experimental group) were ethically sourced from Agbarho [Ughelli North Local government Area (LGA)] and Bomadi (Bomadi LGA); both gas flaring communities in Delta State. One hundred and twenty (120) non-exposed individuals were also recruited (control group) from Abraka, a non-gas flaring community in Ethiope East LGA of the same state. Subjects were matched by gender and duration of stay (exposure) in the target communities. The cardiovascular variables [systolic blood pressure (SBP), diastolic blood pressure (DBP)] of all the subjects were measured, while pulse and mean arterial pressures (PP and MAP respectively) were calculated. Following statistical analysis (using the student t-test), results showed (at $p < 0.05$) a statistically significant increase in SBP and MAP of the experimental group. Study also found a significant increase in DBP and PP for experimental than control group; it also reflected a durational-dependent exposure of subjects to increased SBP and DBP. Exposed males showed an increase in average values of PP, MAP, SBP and DBP than their female counterparts. Also, oil contamination caused a greater negative percentage impact on MAP than gas flaring. This Study therefore ascertained the veracity of previous findings; confirming gas flaring and crude oil contamination as potent elicitors of hypertension. Hence, we recommend periodic epidemiological assessment of environmental pollutants as a factor of hypertensive individuals.

Keywords: Cardiovascular variables, Gas flaring, Gender, Environmental Pollutants

Introduction

Pollution is the contamination of Earth's environment with materials that interfere with human health, quality of life, or the natural functioning of the ecosystems (living organisms and their physical surroundings) in relation to the body's internal environment [1-3]. Although some environmental pollution is a result of natural causes such as volcanic eruptions, most are caused by human and industrial activities [3]. In the 1950s for instance, residents of Minamata, Japan, reportedly began experiencing unusual symptoms, which include numbness, vision problems, and convulsions; and death of hundreds of people caused by mercury ingestion from toxic chemicals dumped into the Minamata Bay by a local industry [4&5].

With the Niger-Delta region of Nigeria famous for oil and gas production and allied industrial activities like oil drilling and local refineries, gas flaring and industrial waste disposal have become rampant, with over 130 reported flaring sites [6]. This makes Nigeria one of the highest emitters of greenhouse gases in Africa [7]. The constant exposure to hazardous chemicals as these, with accompanying deleterious health implications is therefore expected to likely be more in humans that reside close to these refineries and gas flaring sites [8].

Globally, environmental air pollution has been associated with the development of a number of health problems including heart disease, high blood pressure, stroke, lung cancer, as well as chronic and acute respiratory ailments like asthma, bronchitis, etc [9-11]. A More recent research has revealed that many chemical pollutants, such as DDT and PCBs, mimic sex hormones and interfere with the human body's reproductive and developmental functions. These substances are known as endocrine disrupters [12]. The mortalities and morbidities associated with aforementioned disease pose enormous health and economic consequences that reflects on increased loss of productivity, reducing labor efficiency in low to middle income nations [13].

Gas flaring and oil refining activities may affect the sleep-wake cycle in healthy individuals [14]. Also, long term exposure to dioxins, a major product of gas flaring and crude oil refining has been shown to cause neurological symptoms; including neuroglia, sleep disturbances, and severe headache [15&16]. Available evidence suggests that sleep deprivation is positively correlated with increased cardiovascular risk, including hypertension [17].

A meta-Analysis of epidemiological studies has established a positive correlation between cardiovascular risk and exposure to such environmental pollutants as polycyclic aromatic hydrocarbons, sulfur oxides, nitrogen oxides, and polycyclic biphynyles [16]. Findings have also found that inflammatory dose of particulate matter (PM) is linked with increased plasma fibrinogen and blood viscosity, as well as systemic and local inflammatory events [17]. Attenuations in blood coagulability and endothelial dysfunction have also recently been associated with health implications of human exposures to gas flaring [18]. Specifically, in chronic doses, acute exposure to these PM in high concentrations reportedly increases the risk of cardiovascular disorder [19&20]. Currently, reviewers have found that the prevalence of hypertension and other cardiovascular risk factors are significantly higher in urban than rural communities [14]. Though this may be traceable to nutritional and industrialization factors, the situation may differ in the Niger Delta rural communities where

environmental oil and gas pollution has been reported to increase in recent times with a likely, but unexplored impact on health outcomes, particularly cardiovascular health risk. Hence, this study was undertaken.

Aim of Study

This study was designed to examine changes in selected cardiovascular parameters of subjects residing in gas flaring and crude oil contaminated communities in Delta State, Nigeria. Specifically, study determined the comparative effects of gas flaring and crude oil contamination on systolic and diastolic blood pressures, as well as pulse and mean arterial pressures of resident male and females. Study also investigated the duration-dependent effects of exposures to gas flaring and crude oil contamination on systolic and diastolic blood pressures of samples male and female subjects.

Materials and Methods

Scope of Study

Study was non-invasive, and was designed to examine in humans, the comparative changes in cardiovascular parameters by gender, and their durational impact, following exposure to gas flaring and oil contamination in selected communities of Delta State, Nigeria. The work was exclusively designed to involve communities where gas flaring and/or refining activities occur. Abraka, a non-gas flaring community was targeted for non-exposed subjects (control) while Bomadi and Agbarho communities were the gas-flaring sites for experimental subjects.

Study Design

Study adopted the cross-sectional design, geared towards comparing selected cardiovascular parameters by gender and also between residents of gas flaring and oil contaminated communities; and those of non-flaring and non-oil-contaminated areas of Delta State.

Study Location

Three different communities each with similar social-economic and cultural characteristic features, from three different local government areas (LGA) of Delta State, Nigeria were chosen for the study. Bomadi, a rural community in Bomadi LGA of the state was chosen. Bomadi covers an area of 129 km^2, with a population density of about

918.6/km^2. The community is about 118, 500 populated and represented crude oil contaminated communities in this study. Agbarho, another gas flaring community was also selected from Ugheli North LGA of the state. The community is estimated to have 170,000 people in an 818 km^2 area of land. Abraka, a non-gas-flaring, non-oil-contaminated community was selected as control. Abraka is a rural community in Ethiope East LGA of Delta State, and is 276,000 populated

Ethical Clearance

Ethical approval was obtained from the Research and Ethics committee of the Faculty of Basic Medical Sciences, Delta State, University, Abraka, Delta State. Informed Consent forms were carefully structured and given to those who volunteered to participate in the study.

Selection Criteria

For participants to be qualified for selection, several factors were considered in the course of this study; most importantly were; age, non-disability and exceptions to use of heavy drugs.

Eligibility/Inclusion Criteria

Subjects who reside in the study area for more than two consecutive years, who were within the age brackets of 18-45 years were selected for this study.

Exclusion Criteria

Structured questionnaires and interview were used to exclude residents less than 18 years, and those who were above 45 years; also excluded were residents who have lived less than 2 years in various target communities. Subjects who smoke, consume alcohol, and suffer from disorders like diabetes mellitus, hyperlipidaemia, peripheral vascular disease, renal disease, and chronic ailments like sickle cell and asthma were also exempted.

Sample Size

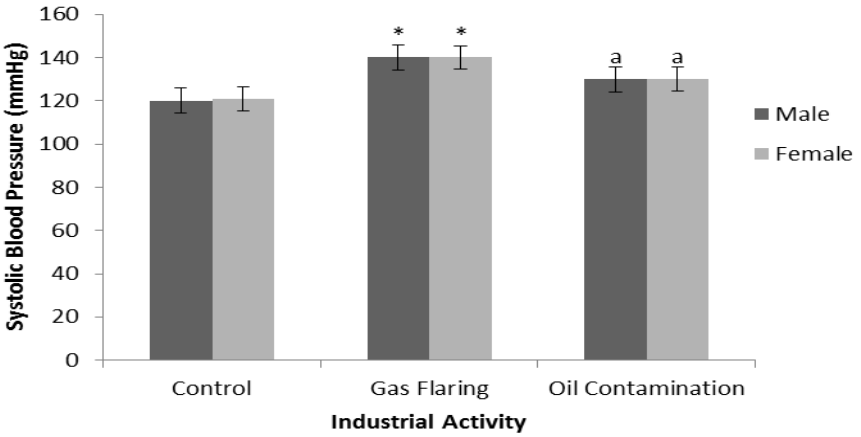
Three hundred and sixty (360) subjects were drawn from three LGAs of Delta State. The sample size of eligible adults was calculated based on the assumed prevalence of hypertension of 18% as earlier reported [21].

Statistical Analysis

Results obtained from the study were expressed as Mean ± SEM (Standard Error of Mean). With P-value of less than 00.05 ($p < 00.05$) considered to be statistically significant, a one-way analysis of variance (ANOVA) was used to determine the mean differences for variables between groups.

Results

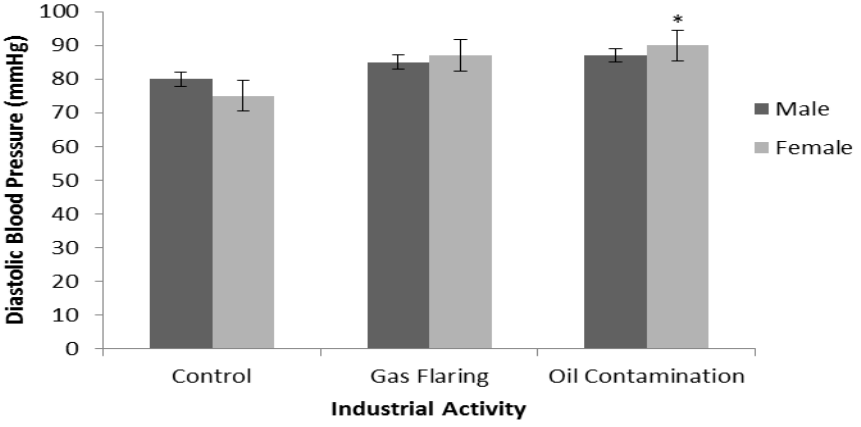
Figure I: Comparative Effect of Gas Flaring and oil Contamination on Systolic Blood Pressure (SBP)



**: significant at $p < 0.05$ as compared to control*

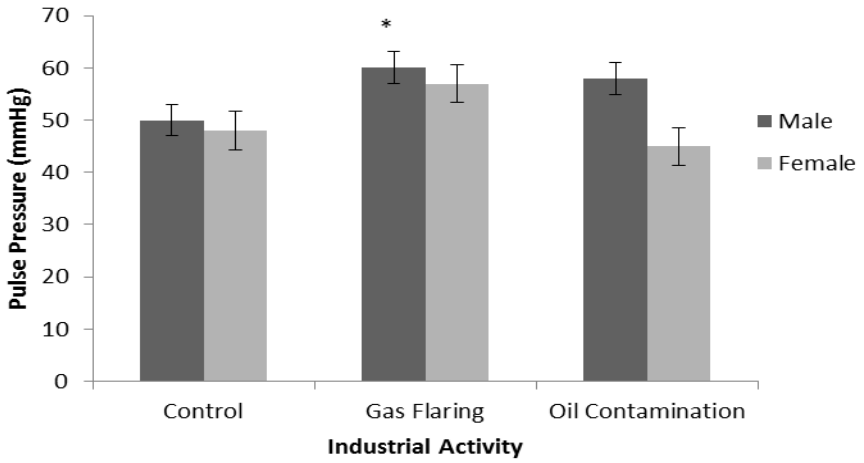
a: significant at $p < 0.05$ upon comparison between gas flaring and oil contamination exposed subjects

Figure II: Comparative Effect of Gas Flaring and oil Contamination on Diastolic Blood Pressure (DBP)



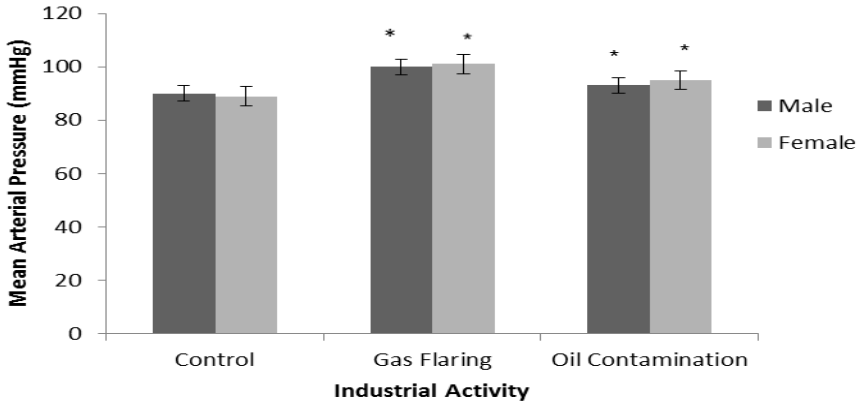
**: significant at $p < 0.05$ as compared to control*

Figure III: Comparative Effect of Gas Flaring and oil Contamination on Pulse Pressure (PP)



*: significant at $p < 0.05$ as compared to control

Figure IV: Comparative Effect of Gas Flaring and oil Contamination on Mean Arterial Pressure (MAP)



*: significant at $p < 0.05$ as compared to control

Figure V: Comparative Percentage Effect of Gas Flaring and oil Contamination on Systolic Blood Pressure (SBP)

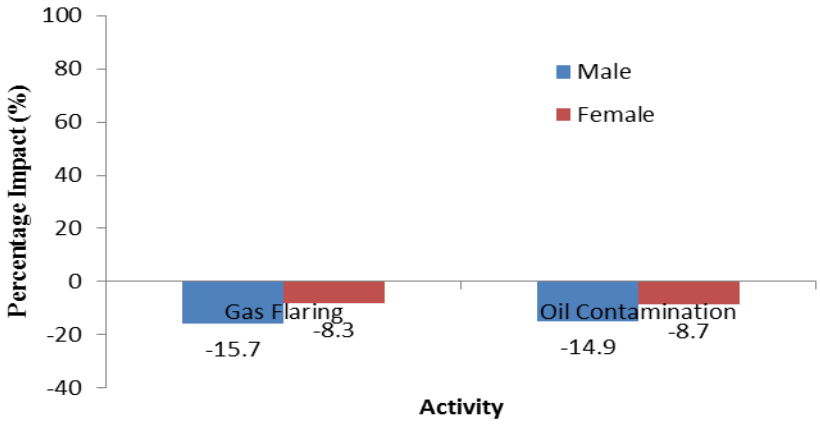


Figure VI: Comparative Percentage Effect of Gas Flaring and oil Contamination on Diastolic Blood Pressure (DBP)

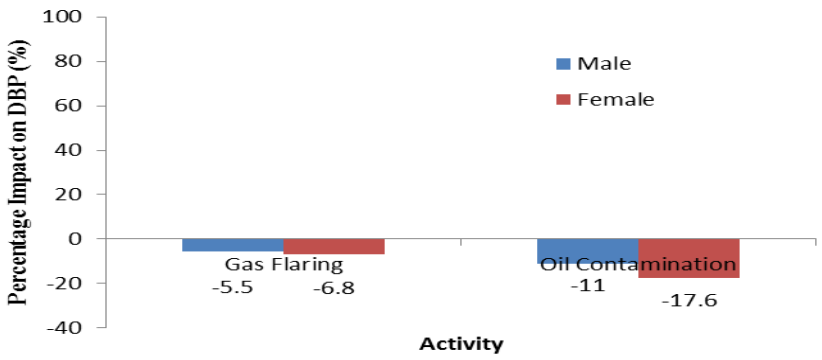
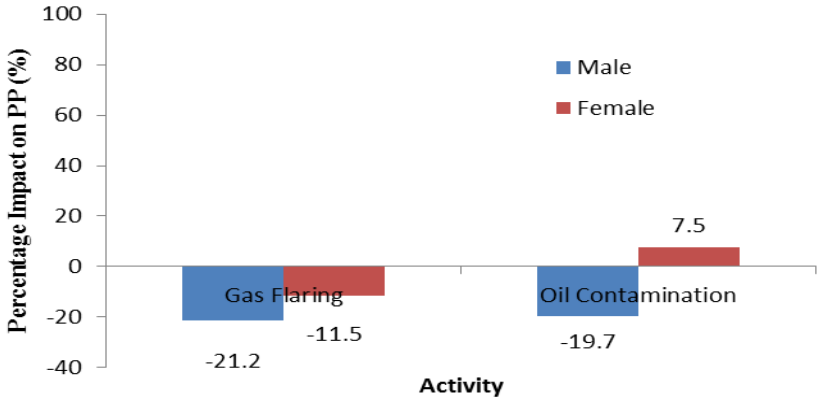
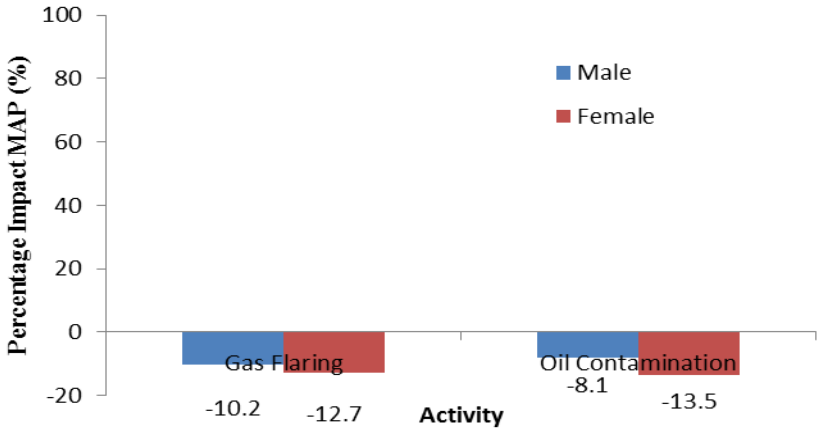


Figure VII: Comparative Percentage Effect of Gas Flaring and oil Contamination on Pulse Pressure (PP)



-%: Negative Impact, +%: Positive Impact

Figure VIII: Comparative Percentage Effect of Gas Flaring and oil Contamination on Mean Arterial Pressure (MAP)



Discussion

Blood pressure (BP) is an important predictor of cardio-vascular events. In recent times, clinicians have traditionally recognized its importance to systolic BP, especially in older adults (JNCP. 1997). Blood pressure may be steady (Mean Arterial Pressure -MAP) or pulsatile (Pulse Arterial Pressure –PAP). This study examined the changes in selected cardiovascular parameters of subjects residing in gas flaring and crude oil contaminated communities of Delta State, Nigeria.

Upon data collection and careful observation, results from this study showed a statistically significant increase in the levels of systolic blood pressure (SBP), Diastolic blood pressure (DBP), and mean arterial pressure (MAP) for subjects exposed to gas flaring and oil contamination. This finding was consistent with Bogers *et al* report of 2007, who observed that prolonged exposure to gas flaring increases the risk of hypertension. Also from this study, MAP was observed to increase significantly with duration, following prolonged exposure of participants to gas flaring and oil contamination. With map saying lot about perfusion pressure, which is the continuously regulated pressure, necessary to maintain end organ-tissue perfusion as required for adequate cellular oxygenation. Thus, though tissue oxygenation was improved with increased MAP, the detrimental changes induced by gas flaring and crude oil contamination could be restricted to the effect on vascular tissues. In accordance with current study, findings from Opie et al., (2007) and Bogers et al., (2007) showed increased risk of hypertension for polluted environments; Similar to an environmental impact assessment study conducted by UNEP (UNEP, 2011). Therefore, inhabitants of oil polluted communities like Bomadi and Agbarho are not only exposed to various air and soil pollutants, but also to water and food pollutants, especially due to bioaccumulation of heavy metals and other agents. Investigations from this study also showed that gas flaring caused more negative impacts on systolic and pulse pressures than crude oil contamination effects when compared (Figure V). Furthermore, the less negative impact of gas flaring on MAP compared to the more negative impact of crude oil contamination could imply that gas flaring has a potent cardio-toxic effect on cardiovascular parameters.

Also noticeable from our result was the prevalence of all blood pressure variables (SBP, DBP, PP and MAP) that were higher in males than in females. This compared male than female increase was statistically significant ($p < 0.05$) in control than test subjects. Physiologically, the increased BP in males may be attributable to the influence of different

developmental renal injury that is reportedly worse in men [22]. In fact, a drop in androgen levels in men with cardiovascular and other chronic diseases has also been implicated. Many investigators now believe that it is the reduction in androgen levels that frequently accompanies chronic disease and may exacerbate cardiovascular diseases in men [23 & 24]. Here, men tend to have higher blood pressure than women upon comparison; irrespective of race, culture and ethnicity [25 & 26]. It has also been observed in other species such as rats, mice, dogs, and chickens to be same.

Again, noise pollution has been proven to aggravate chronic illnesses like hypertension and other cardiopulmonary diseases [27]. Noise pollution does not only contribute to cardiovascular diseases, but it also affects sleep, disrupting its cycle, causes social handicap, hearing loss, increased drug use, impaired teaching, as well as diminished productivity and learning. Recent studies have established a relationship between noise and cardiovascular diseases (CVDs), with the causal route ascribed to neuroendocrine alterations characterized by increased release of cortisol and catecholamine. Furthermore, chronic noise exposure has been associated with hyperlipidaemia, which is a corollary to hypertension [28].

Conclusion

Current study confirmed past findings that gas flaring and crude oil contamination causes hypertension. This implies that environmental pollutants may be useful for screening purposes in the identification of high-risk pollution, long before a diagnosis of hypertension is established. This will help in targeting appropriate intervention. This study has also shown that gas flare and crude oil contamination create a great risk to the cardiovascular system.

Recommendations

While routine surveillance and management of hypertensives remain an important public health priority, periodic epidemiological assessment of environmental pollutants in human tissues is important

References

1. Mancia G, De Backer G, Dominiczak A (2007). Guidelines for the management of arterial hypertension: the Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *Eur Heart J*. 28:1462–1536

2. Manini T M and Clark B C (2009). "Blood flow restricted exercise and skeletal muscle health," *Exercise and Sport Sciences Reviews*, vol. 37, no. 2, pp. 78–85.

3. *Manson JE, Willett WC, Stampfer MJ (*2005*). Body weight and mortality among women. N Engl J Med* 333**:**677*–85.*

4. Marconnet P, Slaoui F, Gastaud M, Ardisson JL (1984). Preexercise, exercise and early post exercise arterial blood pressure in young competitive swimmers versus non-swimmers. J Sports Med Phys Fitness. 24:252–8.

5. Marinoni M, Ginanneschi A, Inzitari D, Mugnai S, Amaducci L (2008). Sex-related differences in human cerebral hemodynamics. *Acta Neurol Scand* 97: 324–327.

6. Martin, HL, Loomis, JL, Kenney, WL (2015). Maximal skin vascular conductance in subjects aged 5–85 yr. *J Appl Physiol* 79: 297–301.

7. Mateos-Caceres P J, Zamorano-Leon J J, Rodriguez-Sierra P, Macaya C and Lopez-Farre A J (2012). "New and old mechanisms associated with hypertension in the elderly," International Journal of Hypertension, vol. 2012, Article ID 150107, 10 pages

8. Mathew E D and Curt D (2006). Sigmund; Genetical basis of hypertension: Revisiting Angiotensinogen; *Hypertension* 48;14-20.

9. *Matteis M, Troisi E, Monaldo BC, Caltagirone C & Silvestrini M (*2008*).* Age and sex differences in cerebral hemodynamics: a transcranial Doppler study. *Stroke 29*, 963–967.

10. Proctor DN**,** Parker BA (2006). *Vasodilation and vascular control in contracting muscle of the aging human. Microcirculation* 13*:* 315*–327.*

11. Proctor DN**,** Shen PH, Dietz NM, Eickhoff TJ, Lawler LA, Ebersold EJ, et al (2008). *Reduced leg blood flow during dynamic exercise in older endurance-trained men. J Appl Physiol* 85*:* 68*–75.*

12. Profenno LA, Porsteinsson AP, Faraone SV (2010). Meta-analysis of Alzheimer's disease risk with obesity, diabetes, and related disorders. *Biol Psychiatry*. 67:505–12.

13. Querido JS**,** Sheel AW (2007*). Regulation of cerebral blood flow during exercise. Sports Med* 37*:* 765*–782.*

14. Rachel Hallmark, James T. Patrie, Zhenqi Liu, Glenn A. Gaesser, Eugene J. Barrett, and Arthur Weltman(2014). The effect of exercise intensity on endothelial function in physically inactive lean and obese adults. *PLoS One*, 9(1): e85450.

15. Black MA, Green DJ *a*nd Cable NT *(*2008*).* Exercise prevents age-related decline in nitric-oxide-mediated vasodilator function in cutaneous microvessels. J Physiol 586, 3511–3524.

16. Blair DA, Glover WE & Roddie IC (2011). Vasomotor responses in the human arm during leg exercise. *Circ Res* 9, 264–274.

17. Bland JM, Altman DG (2009). Measuring agreement in method comparison studies. Stat Meth Med Res. 8:135–160.

18. Boegli Y, Gremion G, Golay S, Kubli S, Liaudet L, Leyvraz P-F, Waeber B & Feihl F (2003). Endurance training enhances vasodilation induced by nitric oxide in human skin. J Invest Dermatol 121, 1197–1204.

19. Bogers RP, Bemelmans WJ, Hoogenveen RT (2007). Association of overweight with increased risk of coronary heart disease partly independent of blood pressure and cholesterol levels: a meta-analysis of 21 cohort studies including more than 300,000 persons. *Arch Intern Med*. 167:1720–8.

20. Bogert LW, van Lieshout JJ (2005). *Non-invasive pulsatile arterial pressure and stroke volume changes from the human finger. Exp Physiol 90: 437–446.*

21. Bolli GB, Knuuti J, Nuutila P (2003). Insulin- and exercise-stimulated skeletal muscle blood flow and glucose uptake in obese men. *Obes Res 11: 257–265.*

22. Borer KT, Wuorinen EC, Lukos JR, Denver JW, Porges SW, Burant CF; Wuorinen; Lukos; Denver; Porges; Burant (2009). "Two bouts of exercise before meals but not after meals, lower fasting blood glucose". Medicine in Science and Sports and Exercise 41 (8): 1606–14.

23. Ono T, Guthold R, Strong K (2005): WHO Global Comparable Estimates: Global Infobase data for saving lives; 2012. https://apps.who.int/infobase/Index.aspx

24. Opie LH, Mayosi BM (2005). Cardiovascular disease in sub-Saharan Africa. Circulation 2005; 112:3536–40.

25. Panel, E. (1998). Executive summary of the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults.*Archives of Internal Medicine*, 158, 1855-1867.

26. Paul, P, Thomas, D. Giles, George, A. Bray, Yuling, Hong, Judith S. Stern, F. Xavier Pi-Sunyer, Robert H & Eckel 2006, 'Obesity and Cardiovascular Disease: Pathophysiology,Evaluation, and Effect of Weight Loss'' An Update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease From the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism *Circulation* 113:898-918.

27. Prentice A, Jebb S. Energy intake/physical activity interactions in the homeostasis of body eight regulation. Nutr Rev. 2004; 62: S98–S104.

28. Poirier, P, Martin, J, Marceau, P, Biron, S & Marceau, S (2004), 'Impact of bariatric surgery on cardiac structure, function and clinical manifestations in morbid obesity', Expert Review on Cardiovascular Ther.2:193–201.