http://jmscr.igmpublication.org/home/
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v10i3.01

IGM Publication Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Age and cardiovascular risks in a market population in Southeast Nigeria

Authors
Innocent Chukwuemeka Okoye[1], Ernest Ndukaife Anyabolu[1], Sylvia Toochukwu Echendu[2], Chinyelu Uchenna Ufoaroh[3], Desmond Onyebuchukwu Ekeh[4], Anulika Nkechinyere Chukwumobi[5], Esther N Umeadi[3]

[1]Department of Medicine, Chukwuemeka Odumegwu Ojukwu University, Awka, Nigeria
[2]Department of Medicine, Nnamdi Azikiwe University Teaching Hospital, Nnewi, Nigeria
[3]Department of Medicine, Nnamdi Azikiwe University, Nnewi, Nigeria
[4]Department of Pharmacology, Nnamdi Azikiwe University, Nnewi, Nigeria
[5]Department of Chemical Pathology, Federal Medical Center, Owerri, Nigeria

Abstract

Background: *Globally, cardiovascular risks constitute a heavy healthcare burden, affecting people of different ages in both the developed and the developing ones. Cardiovascular risks and differences in age have not been completely defined.*

Objectives: *This study was set out to evaluate the influence of age on the risks of cardiovascular disease in Awka, Nigeria.*

Methodology: *This was a cross-sectional study conducted in an unstructured market workers' population. The subjects were classified according to their age and screened for cardiovascular risks. The cardiovascular risks were compared between the different age groups.*

Results: *The study subjects were 294. Females were 72.1% and males 27.9%. Their mean age was 43.13 ± 15.38 years, body mass index (BMI) 28.1 ± 6.0kg/m2), systolic blood pressure (SBP) 129.3 ± 23.7mmHg and diastolic blood pressure (DBP) 82.6 ± 14.7mmHg Majority (98.6%) of the study subjects were Igbos, whereas only 1.4% were Hausas. Tobacco prevalence was 5.4%. Those in the age group of 50-59 years had the highest prevalence of tobacco use (37.5%), followed by those of 30-39 years (25.0%) and 60-69 years (25.0%), p=0.026.Thickened arterial wall had a prevalence of 27.2%. It was high, 42.5%, in those aged 50-59 years, followed by 25.0% in those 60-69 years, p<0.001.Nighttime sleep duration (NTSD)<4hours had a prevalence of 5.4%. Those in the age groups of 30-39 years and 60-69 years had a prevalence of 37.5% of NTSD<4 hours, p=0.002.The prevalence of diabetes mellitus (DM) was 9.5%. The subjects in the age group of 50-59 years had the highest prevalence of DM of 50.0%, followed by 35.7% in those aged 60-69 years, p<0.001. The prevalence of GSM night use was 63.3%. In those aged 20-29 years it was high 25.8% but declined with increasing age, p=0.001. Underweight had a prevalence of 34.0% and occurred in 33.5% of the subjects aged 30-39 years, 50-59 years and 70-79 years. The prevalence of overweight was 28.8% and tended to decline with increasing age. However, obesity increased with age, and was high, 30.0%, in those in the age group of 30-39 years, but declined with increasing age, p=0.002.SBP had a prevalence of 41.5%. SBP of 32.8% was high among the subjects aged 50-59 years, increasing with age initially, p<0.001.The prevalence of DBP was 32.7%, and increased with age to peak at 50-59 years (31.3%), but declined at age >50-59 years, p<0.001.Cigarette smoking p=0.820, alcohol use, p=o.274, and seaster, p=0.210, had insignificant association with age.*

Conclusion: *The prevalence of tobacco use, thickened arterial wall, diabetes mellitus, systolic blood pressure and diastolic blood pressure was high among those in the age group 50-59 years, whereas nighttime sleep duration <4 hours, underweight and obesity was high among those aged 30-39years. GSM midnight use was common among those aged 20-29years in this study.*

Keywords: *Age, Tobacco, Thickened arterial wall, Diabetes mellitus, Hypertension, Nighttime sleep deprivation, Obesity.*

Introduction

Cardiovascular diseases (CVDs) are a group of disorder affecting the heart and its blood vessels.[1] They are the leading cause of preventable deaths globally.[2] Globally, there are estimated 485.6 million cases of CVD with majority of the deaths occurring in adults 75 years and above.[3,4] World Health Organization (WHO) reported an estimated 17.9 million deaths from CVDs in 2019, representing about 32% of all global deaths.[2] About three quarters of this CVD deaths occur in low and middle income countries.[2] The cause is largely unknown but there are several modifiable and non-modifiable risks factors. The modifiable factors are behavioral risk factors such as tobacco use, sedentary life styles, excessive intake of alcohol, unhealthy diets.[2] This behavioral risk factors culminate into overweight, obesity, hypertension, hypercholesterolaemia and diabetes mellitus etc.[2] Age is a major modifiable and independent risk factor for CVDs. Aging is associated with a progressive deterioration of the physiological function of the body as well as structural changes which reduces the functional capacity of the heart leading to increased CVDs.[5,6] The prevalence of CVDs has also been shown to increase in age in both men and women.[7] The American Heart Association reported that the incidence of CVDs increases with age with approximately about 40% for ages 40-59years, 75% for 60-75years and ≥ 86% for individuals that are 80yrs and above. This translates to increased overall healthcare costs from CVDs as one ages.[7] Age is also associated with increased likelihood of development of other risk factors like obesity and hypertension. The structural and functional changes in the heart result in diastolic, systolic and electrical dysfunctions including arrhythmias.[8] functional and electrical defects results from oxidative stress, inflammation, apoptosis, myocardial deterioration and degeneration.[5] An increase in reactive oxygen radicals is known to occur with the onset of advanced age.[5,6] and its linked to persistent inflammation and progression to chronic diseases such as CVDs.[5] Increased production of proinflammatory markers such as IL-6, alpha-TNF and C- reactive proteins are hallmark of aged hearts. These inflammatory factors and other mediators result in cardiac remodeling leading to hypertrophy and fibrosis[5], both of which are significant structural changes that eventually lead to cardiac dysfunction in aging patients.[9] Cardiometabolic functions and lifestyle behaviors may translate into different susceptibility to CVDs between older people and their younger counterparts.[4,10]. Identifying risk factors peculiar to different age groups including those older than 75 years will help promote individualized management strategies to reduce the risk of CVDs. The aim of this study therefore, was to evaluate the influence of age on cardiovascular risk factors.

Materials And Methods

The study population consisted of 294 subjects recruited from Eke Awka main market, Anambra State, Nigeria. The approval for this study was given by the management of the market and from ethics committee of Chukwuemeka Odumegwu Ojukwu University Teaching Hospital, Awka.

The participants were informed of this study through a gong crier, instructed by the management leadership of the market. Each of the participants gave informed oral consent for this study. To them the objectives and aims of the study were explained. A questionnaire was used to collect data which addressed biodata, age, cigarette smoking, tobacco (snuff) use, alcohol use, night-time sleep duration, diabetes mellitus, seaster, GSM use at midnight.

Demographic and anthropometric data were collected. Weight (kg) and height (m) were taken and BMI was determined as weight/height^2 (kg/m^2). Radial and brachial arteries were examined for thickening, and pulse rate was obtained. Blood pressure (mmHg) was recorded from the non-dominant hand in sitting position, after the subjects had rested for 10 minutes, using appropriate cuff size to cover about 80% of the

arm, and Accoson mercury sphygmomanometer. Three blood pressure readings were taken 5 minutes apart, and the average obtained.[11]

The variables were graded into groups as follows:

Smoking: 1) those who were smokers and 2) those who were non-smokers

Tobacco snuff use: 1) those who used tobacco snuff and 2) those who did not

Alcohol: 1) those who have positive alcohol history and 2) those who have not.

Arterial wall: 1) those who have thickened arterial wall and 2) those with normal arterial wall

Night-time sleep duration: A) >6 hours, B) 4 – 6 hours, C) <4 hours

Night vigil: 1) keeping night vigil and 2) no night vigil

The influence of age on the variables were compared between the groups for each variable,

The association of age with the variables was determined. The potential risks evaluated in this study were: tobacco snuff, smoking, arterial wall thickening, alcohol, short night time sleep duration<4hrs, seaster, BMI and hypertension.

Data Analyses

The data were analyzed using the Statistical Package for Social Sciences (SSPS Inc, Chicago, IL) version 17.0 statistical software. For continuous variables, mean values and standard deviations were calculated and the means compared using ANOVA or two sample t-test. Categorical variables were compared using the nonparametric tests Chi-square. The distribution and characterization of variables with age were analyzed using cross tabulation. All tests were two-tailed with $P < .05$ taken as statistically significant.

Definition of terms

Hypertension: SBP ≥140 mmHg and or DBP ≥90 mmHg[12]

Mild hypertension: SBP 140–159 mmHg and/or DBP 90–99 mmHg

Moderate hypertension: SBP 160–179 mmHg and/or DBP 100–109 mmHg;

Severe hypertension: SBP ≥ 180 mmHg and/or DBP ≥ 110 mmHg.

Body mass index:[13]

Underweight: BMI <18,5kg/m^2

Normal body weight: BMI 18.5 – 24.9kg/m^2

Overweight: BMI 25.0 – 29.9kg/m^2

Mild obesity: BMI 30.0 – 34.9kg/m^2

Moderate obesity: BMI 35.0 – 39.0kg/m^2

Severe obesity: BMI ≥40.0kg/m^2

Obesity: BMI ≥30.0kg/m^2.

In this study Obesity was defined as BMI ≥30.0kg/m^2

The subjects who were found to have the risks of cardiovascular disease were counseled and advised to see clinicians in the hospitals.

Results

Sixteen of the 294 subjects smoked cigarette, showing a prevalence of smoking of 5.4%. Out of this number, those aged 30-39 years, 40-49 years and 50-59 years constituted 4(25.0%) each. However, this association between age and smoking was not significant, p=0.820 (Table 3).

Sixteen subjects smoked cigarette. Out of this number, those aged 30-39years, 40-49years and 50-59years constituted 25.0% each. However, this association between age and smoking was not significant, p=0.820 (Table 3).

Sixteen of the subjects used tobacco (Snuff), indicating a tobacco prevalence of 5.4%. Out of this number that used tobacco, those in the age group 50-59 years had the highest prevalence of tobacco use (37.5%), followed by those of 30-39 years (25.0%) and 60-69 years (25.0%). This association between age and tobacco use was significant, p=0.026 (Table 3).

One hundred and twenty-four subjects used alcohol, showing a prevalence of alcohol use of 42.2%. Out of this number that used alcohol, the prevalence of alcohol use was high, 29.0%, among those in the age group 30-39 years, followed by 22.6% among those in 20-29 years. However, this association between age and alcohol use was not significant, p=o.274 (Table 3).

The number of subjects that had thickened arterial wall was 80, with a prevalence of 27.2% for

thickened arterial wall in this study. In these subjects with thickened arterial wall, the prevalence of thickened arterial wall was high, 42.5%, in those aged 50-59 years, followed by 25.0% in those 60-69 years. Thus, this association between age and thickened arterial wall was significant, p<0.001 (Table 3).

The subjects who had NTSD <4hours was 16, showing a prevalence of 5.4% of poor night sleep in the study population. Those in the age groups of 30-39 years and 60-69 years had a prevalence of 37.5% of NTSD<4 hours. Expectedly, this association between age and NTSD was significant, p=0.002 (Table 3).

Twenty-eight subjects had DM, indicating a prevalence of DM of 9.5%. The subjects in the age group of 50-59 years had the highest prevalence of DM of 50.0%, followed by 35.7% in those aged 60-69 years. This association between age and DM was significant, p<0.001 (Table 3).

Seaster during business hours was observed by 112 subjects showing a prevalence of 38.1% of seaster in this study. Those aged 30-39 years had the highest prevalence of 30.4% of lack of seaster during business hours, followed by 21.4% in those aged 20-29 years and 0.0% in those in 90-99 years. However, this association between age and seaster was not significant, p=0.210 (Table 3).

The use of GSM at midnight was observed in 186 subjects showing a prevalence of GSM night use of 63.3%. The prevalence found in those aged 20-29 years was high 25.8% but declined with increasing age. This association between age and GSM midnight use was significant, p=0.001 (Table 3).

The number of subjects who had underweight was 100, indicating a prevalence of underweight of 34.0%. The prevalence of underweight was 33.5% in subjects aged 30-39 years, 50-59 years and 70-79 years. One hundred and four subjects had overweight with a prevalence of 28.8% in this study; and this tended to decline with increasing age. However, obesity increased with age, and was high, 30.0%, in those in the age group of 30-39 years, but declined with increasing age. Expectedly, this association between BMI and age was significant, p=0.002 (Table 3).

SBP ≥140 was found in 122 subjects, showing a prevalence of 41.5% in this study. The prevalence of SBP of 32.8% was high among the subjects aged 50-59 years, increasing with age initially. Thus, this association between age and SBP was significant, p<0.001 (Table 3).

DBP ≥90 was observed in 96 subjects, indicating a prevalence of 32.7% in this study. DBP increased with age to peak at 50-59 years (31.3%), but declined at age >50-59 years. Expectedly, this association between age and DBP was significant, p<0.001 (Table 3).

Table 1: Variables in study population (n=294)

Variables	Mean (standard deviation) years
Age	43.1 ± 15.9
Body mass index	28.1 ± 6.0
SBP	129.3 ± 23.6
DBP	82.6 ± 14.0
SBP=systolic blood pressure. DBP=diastolic blood pressure	

Table 2: Age distribution of study subjects

Age group (years)	Frequency
20-29	23.1
30-39	24.5
40-49	15.0
50-59	21.1
60-69	11.6
70-79	4.1
80-89	0.0
90-99	7.0

Table 3: Distribution and characterization of variables among subjects with varying occupations

Variables	20-29	30-39	40-49	50-59	60-69	70-79	90-99	Chi square	df	LHR	p value
Smoking Yes No	2(12.5 66(23.7))	4(25.0) 63(24.5)	4(25.0) 40(14.4)	4(25.0) 58(20.9)	2(12.5) 32(11.5)	0(0.0) 12(4.3)	0(0.0) 2(0.7)	2.908	6	0.725	0.820
Tobacco use Yes No	0(0.0) 68(24.5)	4(25.0%) 68(24.5)	0(0.0) 44(15.8)	6(37.5) 56(20.1)	4(25.0) 30(10.8)	2(12.5) 10(3.6)	0(0.0) 2(0.7)	14.303	6	0.005	0.026
Alcohol use Yes No	28(22.6) 40(23.5)	36(29.0) 36(21.2)	14(11.3) 30(17.6)	24(19.4) 38(22.4)	16(12.9) 13(10.6)	4(3.2) 8(4.7)	2(1.6) 0(0.0)	7.535	6	0.217	0.274
ThickArtWall Yes No	0(0.0) 68(31.8)	6(7.5) 66(30.8)	8(10.0) 36(16.8)	34(42.5) 28(13.1)	20(25.5) 14(6.5)	12(15.0) 0(0.0)	0(0.0) 2(0.9)	114.082	6	<0.001	<0.001
Nighttime sleep duration (hrs) >6 4-6 <4	44(28.9) 24(19.0) 0.0.0)	36(23.7) 30(23.8) 6(37.5)	20(13.2) 22(17.5) 2(12.5)	24(15.8) 36(28.6) 2(12.5)	16(10.5) 12(9.5) 6(37.5)	10(6.6) 2(1.6) 0(0.0	2(1.3) 0(0.0) 0(0.0	31.519	12	0.001	0.002
DM yes No	0(0.0 68(25.6)	0(0.0) 72(27.1)	4(14.3) 40(15.0)	14(50.0) 48(18.0)	10(36.7) 24(9.0)	0(0.0) 12(4.5)	0(0.0) 2(0.8)	44.903	6	<0.001	<0.001
Seaster Yes No	44(24.2) 24(21.1)	38(20.9) 34(30.4)	30(16.5) 14(12.5)	40(22.0) 22(19.6)	18(9.9) 16(14.3)	10(5.5) 2(1.8)	2(1.1) 0(0.0)	8.450	6	0.156	0.210
GSM at midnight yes No	48(25.8) 20(18.5)	44(23.7) 28(25.9)	36(19.4) 8(7.4)	36(19.4) 20(24.1)	26(10.8) 14(13.0)	2(1.1) 10(9.3)	0(0.0) 2(1.9)	23.826	6	<0.001	0.001
BMI: <18.5kg/m^2 18.5-24.9kg/m^2 25.0-29.9kg/m^2 ≥30.0kg/m^2	0(0.0) 24(27.9) 30(28.8) 14(14.3)	2(33.3) 18(20.9) 22(21.2) 30(30.6)	0(0.0) 8(9.3) 18(17.3) 18(18.4)	2(33.3) 16(18.6) 22(21.2) 22(22.4)	0(0.0) 12(14.0) 8(7.7) 14(14.3)	2(33.3) 6(7.0) 4(3.8) 0(0.0)	0(0.0) 2(2.3) 0(0.0) 0(0.0)	40.563	18	0.002	0.002
SBP≥140+DBP≥90 SBF<140+DBP<90	14(11.5) 54(31.4)	22(18.0) 50(29.1)	14(11.5) 30(17.4)	40(32.8) 22(12.8)	24(19.7) 10(5.8)	8(6.6) 4(2.3)	0(0.0) 22(1.2)	47.429	6	<0.001	<0.001
DBP≥90+DBP≥90 DBF<90+DBP<90	10(10.4) 58(29.3)	16(16.7) 56(28.3)	12(12.5) 32(16.2)	30(31.3) 12(6.1)	22(22.9) 6(3.0)	6(6.3) 6(3.0)	0(0.0) 2(1.0)	39.573	6	<0.001	<0.001

ThickArtWall=thickened arterial wall. BMI=body mass index. SBP=systolic blood pressure. DBP=diastolic blood pressure. Hr=hours

Discussion

Tobacco use is a major risk factor for CVDs. Its use is responsible for over 8million death per annum and it is projected to be about 10 million in 2030.[14] It has been ranked as the leading cause of preventable death. The prevalence of cigarette smoking was about 5.4% in this study this is similar to the global adult tobacco survey report of 5.6%.[15] Aniwada et al also reported a similar finding of 6.6% in males and 6.3% in females in a study done in Nigeria.[16] However, this is lower than the global rate of 20.7% in 2015.[17] It is a risk factor for a wide range of diseases such as lung cancers, cardiovascular diseases, stroke andchronic respiratory diseases.[18,19] About 12.5% of the study participants between the ages of 20-29yrs were smoking while 25% each of ages 30-39, 40-49, and 50-59 were found to be smoking in this study. A study by Aniwada et al reported similar finding that those 25-34 years and ≥35 years were two times more likely to use any form of tobacco than the younger age groups 15-24 years.[16] This study was also supported by a study done in Nepal which showed that those in the age group of 36-49 years were 2.4 x more likely to use any form of tobacco than the younger age group 15-24years.[20] A study in Zaria, Nigeria also reported age to be associated with tobacco use.[21] Age of initiation of smoking is an important determinant of smoking behavior in later life. Global research has reported that tobacco smoking is usually initiated before 18years of age.[22] It is established that people who started smoking at a young age may end up being smokers in adulthood.[23] This trend increased at 30years and remained the same till 59years. The reasons for the high rate of smoking between the ages of 20-29yrs could be explained by effect of peer pressures, youthful exuberances and the increased urge to explore the environment. Peer pressure as a driving force for smoking has also been documented in other findings.[24,25]Another important reason among the youth may be that those whose parents smoke may likely go into

smoking. Smoking among family members have been documented to be a predictor of current smoking in some other studies.[25,26] Those between the ages of 30 -59years are probably apparently healthy to explore. However, this trend declined beyond 59years. The reason for this is not far- fetched because from about 60years individuals have diseases such as hypertension, diabetes, atherosclerosis and other ailment that come with age to contain with and therefore, tend to modify their lifestyles.

Smokeless tobacco such as snuff is a known risk factor for cardiovascular disease.[27-29] The prevalence of tobacco smoking in this study was 5.4%. This is slightly higher than the prevalence obtained from Demographic and Health survey reported prevalence of 2.6% in males and 1.9% female as smokeless tobacco users.[30] No participant was recorded to be smoking tobacco (snuff) between ages 20-29years. The traditional perception of our adolescents and young adults to snuff. It is believed that it is mostly for the elderly. The use of tobacco peaked at between 50-59years and started declining again beyond 59 years probably as a means of lifestyle modification following diseases that come with aging. Smoking leads to plaque formation in the blood vessels, thickens the blood, and results in clot formation. This results to narrowing of vessels with resultant cardiovascular heart disease.[31]

Alcohol is the most commonly abused psychoactive substances in the country.[24,32] In this study, alcohol intake peaked between 20-39 years. This is expected as social life and peer influences are very high within these ages. However, Onodugo et al reported a higher peak of alcohol consumption among those aged 40-49 years followed by those 50-59years old. This difference may probably be because of the differences in sample size. He used a sample size of 1411 which was by far higher than the 294 used in this study and this might have given a better representation of the alcohol consumption in the community. He also reported age as an important correlates of alcohol consumption.[33] Heavy alcohol intake is an important cause of death and disability.[34] There is also a complex association between alcohol and cardiovascular diseases.[35-37] Observational studies have reported that alcohol consumption is positively associated with heart failure[35] and hemorrhagic stroke.[36] and atrial fibrillation.[37] However, moderate consumption of alcohol lowers risks of cardiovascular disease and stroke.[35-37]

Aging is associated with increases in wall thickness of medium and large sized arteries due to smooth vessel hypertrophy resulting to thickening of the intima medial layer.[38] This is similar to what was observed in this study with peak in 50-59years. However, the trend declined from age 60-79years. Arterial wall thickening is a key factor in the progression of arterial disease and this creates an enabling environment for the formation of atherosclerosis.[39]

Diabetes mellitus also increased with age in this study with peak at 50-59 years (50%) followed by 60-69 years. Other studies also reported increasing prevalence of diabetes with advancing age.[40,41] Beyond the age of 70years the prevalence dropped probably because diabetes mellitus being a chronic condition may have led to deaths beyond the age of 70years. Over time, high blood sugar damages the blood vessels and nerves controlling the heart. This is worsened by other morbidities present in people with diabetes.

Sleep is an important physiologic process that helps to calm the nerves and keep us mentally alert. The sleep pattern tends to change with advancing age due to reduced levels of growth hormones and melatonin. This study showed a progressive decline in night time sleep (>6hrs) with age. Night time sleep deprivation acts as a stressor, activating the sympathetic pathway via the rennin-angiotensin-aldosterone pathway with resultant increase in central catecholamine which causes blood vessel constriction with consequent increase in the blood pressure resulting in hypertension.[42,43] There was significant association between use of GSM night sleep and

sleep deprivation. GSM use was highest among participants between the ages of 20-29 years. This is the age when web exploration and social media use is highest. Most university and college students also fall into this group and are more likely to study at night with Global System for mobile communication. Intracellular concentration of magnesium decreases with poor sleep leading to arterial constriction.[44] NTSD< 4hrs was highest among those 30-39 years. This is probably because people at this age group are very energetic and are more likely involved in night activities.

In this study, obesity increased with age and was highest among the age group of 30-39years but started to decline beyond 39 years of age. Stevens et al reported similar findings that relative risk of obesity was higher among those aged 30-44years than among 65-74 years.[45] the reasons for this might be due to increased consciousness of the potential risks of obesity and its role in the development of diabetes, hypertension, hypercholesterolemia as one ages. Secondly, the management of these conditions now compel them to aim at achieving better management through weight control programs such as dieting and exercise with resultant decline in weight as one moves beyond 40years of age.

Both systolic and diastolic blood pressure increased with age, and peaked at 50-59 years in this study. Most of the risk factors reported in this study peaked between 50-59 years of age. The combined effects of age and other risk factors may have resulted in increase in blood pressure. Other similar studies supported similar findings.[46,47]

Overall, age is an independent, non- modifiable risk factors in the development of CVDs. It also contributes to the development or worsening of most of other risk factors as seen in this study. Therefore, having an understanding of the mechanism that accelerates aging could slow the development of CVDs and enhance advancement into the management of CVDs.

Conclusion

The prevalence of tobacco use, thickened arterial wall, diabetes mellitus, systolic blood pressure and diastolic blood pressure was high among those in the age group 50-59 years, whereas nighttime sleep duration <4hours, underweight and obesity was high among those aged 30-39years. GSM midnight use was common among those aged 20-29 years in this study.

Funding

The study was funded solely by the researchers. There was no direct or indirect cost to the study participants.

Acknowledgement

Great thanks to all that contributed to this work from the initial design, data collection, analysis manuscript writing, revisions to its final form.

Competing Interests

There was no conflict of interest declared by the authors.

References

1. Farley A, McLafferty E, Hendry C. The cardiovascular System. Nurs Stand 2012; 27:34-9.
2. World Health Organization. Health Fact Sheet for cardiovascular diseases. June, 2021. Accessed January, 2022.
3. GBD 2017 causes of death, collaborators, global, regional and national age-sex-specific mortality for 282 causes of death in 195 countries and territories, 1980-2017: A systematic analysis for global burden of disease study 2017. Lancet 2018; 392:1736-88.
4. Rich MW, Chyun DA, Skolnick AH Et al. Knowledge gaps in the cardiovascular of the older adult population: A scientific statement from the American Heart Association, American College of Cardiology, and American Geriatrics

Society. J Am Coll Cardiol 2016; 687:2419-40.
5. Curtis AB, Karki R, Hattoum A, Sharma UC. Arrhythmias in patients ≥80 years of Age: pathophysiology, Management, and Outcomes. J Am Coll Cardiol 2018; 71:2041-57.
6. North BJ, Sinclair DA. The intersection between aging and cardiovascular disease. Circ Res 2012; 110:1097-108.
7. Yazdanyar A, Newman AB. The burden of cardiovascular disease in the elderly: Morbidity, mortality and costs. Clin Geriatr Med 2009; 25:563-77.
8. Steeman M, Lance G. Cardiac aging and heart disease in humans. Biophys Rev 2017; 9:131-137.
9. Meschiari CA, Ero OK,Pan H, Finkel T, Lindsey ML. The impact of aging on cardiac extracellular matrix. Geroscience 2017; 39:7-18.
10. Madhavan MV, Gersh BJ, Alexander KP, Granger CB, Stone GW. Coronary artery disease in patients ≥80 years of age. J Am Coll Cardiol 2018; 7:12015-40.
11. Ogedegbe G, Pickering T. Principles and techniques of blood pressure measurement Cardiol Clin 2010;28:571-86.
12. Whitworth JA. World Health Organization (WHO)/ International Society of Hypertension (ISH) statement on management of hypertension. Journal of Hypertension. 2003; 21(11): 1983–1992.
13. WHO (1995) Physical Status: The Use and Interpretation of Anthropometry. Technical Report Series 854, 1-1-9950. World Health Organization, Geneva.
14. Townsend L, Flisher AJ, Gilreath T, King GA. A systemic literature review of tobacco use among adults 15 years and older in Subsaharan Africa. Drug Alcohol Depend 2006; 81:14-27.
15. 15Global Adult Tobacco Survey: Country report 2012. World Health Organization. http//www.who.int/tobacco/sc. Accessed Feb 8, 2022.
16. Aniwada EC, Uleanya ND, Ossai ED, Nwobi EA, Anibueze M. Tobacco use: Prevalence, pattern and predictors among those aged 15-49years in Nigeria- a secondary analysis. Tob Induc Dis 2018; 16:7. Doi: https://doi.org/10.18332/tid/82926.
17. Scollo M, Windstanley M. Tobacco in Ausralia: facts and issues, Melbourne :Cancer Council Victoria.2018.
18. Moore S, Texeira A, Stewart S. Effect of network social capital on the chances of smoking relapse: A 2year follow up study of urban dwelling adults. Am J Public Health 2014;104:e72-6.
19. Drope J, Schluger N, Cahn S, Drope J, Hamill S, Islami F et al. Tobacco Atlas 6TH edition. Atlanta: American Cancer Society and Vital Strategies2018.
20. 20Sreeramareddy CT, Ramakri Shnareddy N, Harsha Kumar H, Sathian B, Arokiasamy JT. Prevalence, distribution and correlates of tobacco smoking and chewing in Nepal: a secondary data analysis of Nepal Demographic and Health Survey 2006. Substance Abuse Treatment, Prevention and Policy 2011;6. doi:10.1186/1747-597X-6-33.
21. Idris SH, Sambo MN. Psychoactive substance use among in-school adolescents in Zaria, North Western Nigeria: what are the triggers? Niger J Med; 18:291-4.
22. 22Pierce JP, Whit MV, Emery SL. What public healthcare needed to reduce smoking initiation? Tob Control 2012; 21:258-64.
23. Funatogawa I, Funatogawa T, Yano E. Impacts of early smoking initiation: Long term trend of lung cancer mortality and smoking initiation from repeated cross-sectional surveys in Great Britain. BMJ open 2012;22e167 British Medical Journal Publishing Group.

24. Okpataku CT. Pattern and reasons for substance use among long distance drivers in a Nierian city. Indian J Public Health 2015; 59:259-63.
25. Mak kk, Ho Sy, Day JR. Smoking parents and best friend- independent and combined effects on adolescent smoking and intention to initiate and quit smoking. Nicotine Tob Res 2012;14: 1057-64.
26. Lewinsohn PM, Brown RA, Seeley JR, Ramsey SE. Psychosocial correlates of cigarette smoking abstinence, experimentation, persistence and frequency during adolescence. Nicotine Tob Res. 2000; 2:121–131.
27. Teo KK, Ounpuu S, Hawken S, et al. ; INTERHEART Study Investigators Tobacco use and risk of myocardial infarction in 52 countries in the INTERHEART study: a case-control study. Lancet. 2006;368(9536):647–658.
28. Boffetta P, Straif K. Use of smokeless tobacco and risk of myocardial infarction and stroke: systematic review with meta-analysis. BMJ. 2009;339: b3060.
29. Critchley JA, Unal B. Is smokeless tobacco a risk factor for coronary heart disease? A systematic review of epidemiological studies. Eur J Cardiovasc Prev Rehabil. 2004;11(2):101–112.
30. Mdege ND, Shah S, Ayo-Yusuf OA, Hakim J, Siddiqi K. Tobacco use among people living with HIV: Analysis of data from Demographic and Health Surveys from 28 low-income and middle-income countries. Lancet Glob Health. 2017;5(6):e578–e592.
31. Collaborators GBDA. Alcohol use and burden of 195 countries and territories 1990-2016.: A systematic analysis for the global burden of disease study 2016. Lancet 2018; 292:1015-35.
32. Adelekan ML, Abiodun OA, Obayan AO, Oni G, Ogunremi, OO. Prevalence and Pattern of Substance Use among Undergraduates in a Nigerian University. Drug and Alcohol Dependence 1992; 29:255-261.
33. Wood AM, Kaptoye S, Butterworth AS, Willeit P, Waranakula S, Botton T et al. Emerging risk factors Collaboration/ Epic-CVD/ UK Biolank Alcohol study Group. Risk thresholds for alcohol consumption: combined analysis of individual participant data for 599912 current drinkers in 83 prospective studies. Lancet 2018; 391: 1513-23.
34. Larson SC, Wallin A, Wolk A, Markus HJ. Differing association of alcohol consumption with different stroke type: Asystemic review and metaanalysis. BMC Med 2016; 14:178. doi :10.1186/s/12916-016-0721-4.
35. Larson SC, Drca N, Wolk A. Alcohol consumption and risk of atrial fibrillation: a prospective study and dose response metaanalysis. J Am Coll Cardiol 2014; 64:281-9.
36. Ronksley PE, Brien SE, Turner PJ, Mukamal KJ, Ghals WA. Association of alcohol consumption with selected cardiovascular disease outcomes: A systemic review and metaanalysis. BMJ 2011;342: d671. doi:10.1136[BMJ. D671].
37. Tellides G, Pober JS. Inflammatory and immune responses in the arterial Media. Circ Res 2015; 116:312-22.
38. Lakatta EG, Levy D. Arterial and cardiac aging: Major shareholders in cardiovascular disease enterprises. Circulation 2003; 107:346-54.
39. Somers VK, Dyken ME, Mark AL, Abbound FM. Sympathetic-nerve activity during sleep in normal subjects. N Engl J Med 1993; 328:303-7.
40. Nyenwe EA, Odia OJ, Ihekwaba AE, Ojule A, Babatunde S. Type 2 diabetes in adult Nigerians: a study of its prevalence and risk factors in Port Harcourt,

Nigeria. *Diabetes* Res Clin Pract 2003;62(3):177–185.

41. Saquib N, Saquib J, Ahmed T, Khanam MA, Cullen MP. Cardiovascular diseases and type 2 diabetes in Bangladesh: a systematic review and meta-analysis of studies between 1995 and 2010. BMC Public Health. 2012; 12:434.
42. Aston-Jones G, Chen S, Zhu Y, Oshinsky ML. A neural circuit for circadian regulation of arousal. Nat Neurosci 2001; 4:732-8.
43. Joo EY, Yoon CW, KOO DL, Kim D, Hong SB. Adverse effect of 24hours sleep deprivation on cognition and stress hormones.J Clin Neurol 2012;8:146-50.
44. Takase B, Akima T, Uehata A, Ohsuzu F, Kurita A. Effect of chronic stress and sleep deprivation on both flow-mediated dilation in the brachial artery and the intracellular magnesium level in humans. Clin Cardiol 2004; 27:223-7.
45. June S, Jianwem C, Elsie RP, David FW, Micheal J, Thun MD and Joy L. The effect of age on the association between body mass index and mortality. N Engl J Med 1998; 338:1-7
46. Franklin SS, Gustin W, Wong ND et al. Haemodynamic patterns of age related changes in blood pressure. The Framingham Heart Study. Circulation 199796308-315.
47. Stott DJ, Bowman A. blood pressure and aging. J Hum Hypertens 200014771-772.