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Cardio-Pulmonary Risk Markers in Automobile Sprayers and Saw Millers

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Authors' contributions

This work was carried out in collaboration between all authors. Author RNA designed the study. Author EIE performed the statistical analysis and wrote the protocol. Author CCI performed the literature searches. Author SI supervised the experimental protocol. All authors read and approved the final manuscript.

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ABSTRACT

Other than oxygen, the air that enters the respiratory system contains several substances. These substances are derived from a heterogeneous mixture of gases, liquids and particulate matter. Exposure to air pollutants cause deleterious effects on the cardio-respiratory system. This study sought to investigate the effect(s) that air pollutants have on markers of cardiovascular and respiratory health in occupationally exposed individuals. An ex post factor study was conducted on 60 ethically recruited individuals from Enugu metropolis of Enugu state, Nigeria. They were assigned into three groups (A-C) of 20 subjects each. With group A acting as control, groups B and C comprised of automobile spray painters and saw millers respectively. Participants' respiratory (Forced Expiratory Volume (FEV), Forced Vital Capacity (FVC), Peak Expiratory Flow Rate (PEFR)); cardiovascular (Electrocardiogram (ECG), systolic blood pressure (SBP), diastolic blood pressure (DBP)) and hematological parameters (Fibrinogen concentrations and C-Reactive Proteins)) were assayed. Blood samples were also collected with parameters checked and

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compared with control. Analysis of Variance (ANOVA) on collected data showed diastolic pressure (DBP) to have significantly increased with significant differences ($p < .05$) in FEV, whilst presenting insignificant values in PEFR and FVC of group C subjects. Also, a moderately strong and significant positive relationship (from Pearson Product Moment Correlation) was seen between ECG parameters (QRS and P-R) and duration of work for control (group A). Routine spirometry, haematological and ECG tests are recommended for individuals who are exposed to air pollutants as a result of occupational predisposition.

Keywords: Environmental pollution; cardio-respiratory system; lung volumes and capacities.

1. INTRODUCTION

Literarily known as the "apparatus of breathing", the respiratory system supplies a critical component of life; the oxygen, and disposes of a major waste product, carbon dioxide [1,2]. The air that enters the respiratory system contains many substances other than oxygen, including natural constituents (e.g., nitrogen), as well as human and environmental contributions (e.g., fossil fuel combustion by-products). Various defence mechanisms of the respiratory system eliminate the unnatural components of air from the body and help repair any damage they may likely have. But exposure to large amounts of toxic substances or chronic exposure to lower levels can overwhelm the ability of the respiratory system to protect and repair itself, sometimes resulting in impaired lung function. Such exposures to copious amounts of toxins can be traceable to a wide range of sources including the environment, human and industrial wastes/disposals, as well as exposures resulting from job-related or occupational activities [3].

An occupational disease is any chronic ailment that occurs as a result of work or occupational activity. It is typically identified when its prevalence is more in a given body of workers than in the general population, or in other worker populations [4]. Occupational cardio-respiratory disease is the name for a collection of conditions of the cardiovascular and/or respiratory systems, which can have occupational risk factors for the particular disease. This includes acute, sub-acute and chronic diseases, which may be malignant, non-malignant or in infectious origin. Common occupational diseases of such may include occupational asthma, black lung disease(coal worker's pneumoconiosis), chronic obstructive pulmonary disease (COPD), mesothelioma, and silicosis. Infectious lung diseases can also be acquired in an occupational context. Exposure to substances like dust, fumes, and gases such as isocyanate paint mist, silica, etc. can cause fibrotic lung diseases and impairments in cardio-respiratory functions, whereas exposure to carcinogens like asbestos and beryllium can cause lung cancer.

Significant decreases in Forced Expiratory Volume (FEV), Forced Vital Capacity (FVC) and Peak Expiratory Flow rate (PEF) show with varieties of symptoms including a cough, dyspnoea, and phlegm that have been associated with exposures to gases and fumes [5-7]. This study was developed to ascertain the effect that occupational exposures have on the respiratory system of automobile sprayers and saw millers.

1.1 Aim of Study

This study was aimed at determining amongst workers in Enugu metropolis, the influence of airborne pollutants on markers of cardiovascular risk and lung function; using automobile sprayers and saw millers. Specifically, the study sought to examine:

1. The influence of occupational exposure to particulate air pollutants (saw dust and paint mist) on vital signs (systolic and diastolic blood pressures as well as heart rates).
2. The influence of occupational exposure to particulate air pollutants (saw dust and paint mist) on lung functions (Forced Expiratory Volume, Vital Capacity, and Peak Expiratory Flow rate)
3. The influence of occupational exposure to particulate air pollutants (saw dust and paint mist) on electrical activity (QRS duration, P-R interval and QTc value) of cardiac muscles.

2. MATERIALS AND METHODS

2.1 Scope of Study

This study was conducted in Enugu metropolis of Enugu state, Nigeria. Enugu State is a 215 sq mi

(556 km^2) area of land in Nigeria that lies approximately between Longitude 7° 31’ East and Latitude 6° 26’ North (Duckworth, 1961).

2.2 Study Design

The study adopted the ex-pose facto research design. This was considered appropriate because selected subjects were studied after changes in the dependent variables (Lung function, electrocardiogram, fibrinogen, and C-reactive protein) have occurred due to occupational exposure to air pollutants (independent variables), and thus, the investigator does not manipulate the independent variable (Polite et al. 2005). Using the random sampling technique, a total of Sixty (60) participants were selected for the study. They were randomly assigned into three groups (A-C) of 20 subjects each. While group A served as control, group B comprised of automobile spray painters, leaving group C exclusively for saw millers. An abridged version of the National Institute for Occupational Safety and Health Asthma Identification questionnaire was administered before eligible for inclusion; if proven to be non-asthmatic.

2.3 Ethical Issues

Ethical approval was obtained from the Research and Ethics committee of the University of Nigeria, Enugu campus. Participants’ confidentiality were maintained by using code numbers instead of names, and ensuring that records were destroyed at the end of the study. Subjects’ informed consents were also obtained prior to study; after the procedure and purpose of research had been clearly explained to them [8,9].

2.4 Selection Criteria

2.4.1 Inclusion criteria

- Only workers with a minimum of 5 years uninterrupted work experience [10,11]
- Only workers who have not taken any form of sedative for 72 hours (at least) before investigation [12]
- Workers without any form of anti-hypertensive medication for the past 72 hours (at least) before the investigation
- Only paint workers who were directly physically, and routinely involved in paint mixing and vehicle spraying

2.4.2 Exclusion criteria

- Individuals with any history of smoking were excluded. This was because nicotine inhalation overtime has been found to alter cardio-pulmonary functionalities [13]
- Habitual tobacco snuffers were also excluded as it has been found to cause acute and chronic pulmonary diseases [14].
- Subjects with known history of asthma or obstructive airway diseases
- Individuals whose ages were above 65years

2.4.3 Procedure

Questionnaires were administered to each participant to be sure if they are asthmatic or not. Next, they were required to rest for 30 minutes prior to investigation. For each subject, Lung function [Forced Expiratory Volume (FEV), Forced Vital Capacity (FVC), and Peak Expiratory Flow rate (PEF)], systolic and diastolic blood pressure, heart rates, as well as electrocardiogram readings (QRS, P-R interval and QTC values) were measured. To obtain spirometry readings, subjects were instructed to breathe in and out in upright, relaxed position. Forced expiration and inspiration readings were then read at maximum breathe outs and breathe in respectively.

2.4.4 Analytical approach

A data analysis was conducted with SPSS (version 21) using the one way analysis of variance (ANOVA). Post-hoc analysis was carried out with the Bonferroni test. Pearson Product Moment Correlation Coefficient was used to determine the linear relationship between duration of exposure to air pollutant (work experience) and various parameters of lung function and electrocardiogram. All tests were one-tailed, with alpha (α) set at .05. Results are presented as means and standard deviations.

3. RESULTS

See tables and graphs below for detailed presentation of results.

4. DISCUSSION

This study indicates that cardio-pulmonary risk markers vary in individuals according to their occupations and duration of exposure of to environmental pollutants. Our results show the following:

4.1 Systolic Blood Pressure and Exposure to Paint Mist and Saw Dust

The result from this study indicates that changes in systolic blood pressure (SBP) occurred in 23.3% of the population. The effect of this on paint mist exposed workers was small, while high in sawdust exposed workers. Therefore, the change of SBP due to occupational exposure in sawdust exposed workers is of immense clinical relevance. This finding aligns with that of AHA, 2002, which compared several consistencies in the effect of paint mist on blood pressure [3].

Table 1. Physical characteristics and occupational history of participants

Variables	Category	X±SD	Range	F- Value	P-Value
	Control (A)	42.30+9.06	23-53		
Age (Years)	Paint (B)	40.75+9.85	17-65	5.386	0.002*
	Saw dust (C)	52.20+9.77	34-71		
	Control (A)	79.60+11.38	49-105		
Weight (kg)	Paint (B)	71.80+11.50	59-100	5.064	0.003*
	Saw dust (C)	78.65+13.48	51-102		
	Control (A)	1.72+0.06	1.43-1.56		
Height (m)	Paint (B)	1.73+0.79	1.60-1.91	2.250	0.089
	Saw dust (C)	1.69+0.08	1.58-1.90		

**Significant at p < .05, X=mean, SD= standard deviation*

Table 2. Cardiovascular parameters of participants

Variables	Control (Grp A) X±SD	Paint (Grp B) X±SD	Saw dust (Grp C) X±SD	f - Value	p - Value
HR	76.85±11.55	73.45±9.50	75.45±16.81	0.557	0.645
SBP	123.50±21.90	121.10±22.85	146.50±23.90	2.903	0.040*
DBP	94.85±12.58	63.30±14.86	96.20±12.91	19.073	0.001*

*Keys: HR=Heart Rate, SBP=Systolic Blood Pressure, DBP=Diastolic Blood Pressure *Significant at p < .05*

Table 3. Lung function parameters of participants

Variables	Control (Grp A) X±SD	Paint (Grp B) X±SD	Saw dust (Grp C) X±SD	f - Value	p – Value
FVC	3.34±3.45	3.06±0.72	3.01±2.47	0.995	0.400
FEV	2.36±1.13	2.21±0.87	1.86±0.98	6.128	0.001*
PEF	3.74±1.71	4.40±1.71	3.12±1.31	5.523	0.002*

*Keys: FVC=Forced Vital Capacity, FEV=Forced Expiratory Volume, PEF=Peak Expiratory Flow rate, X=mean, SD=Standard Deviation, *Significant at p<.05*

Table 4. Electrocardiogram profile of participants

Variables	Control (Grp A) X±SD	Paint (Grp B) X±SD	Saw dust (Grp C) X±SD	f - Value	p – Value
P-R Interval	154.20±22.65	160.90±27.86	168.40±21.43	1.097	0.356
QRS duration	87.40±13.37	86.70±12.44	84.60±15.32	0.859	0.466
QTc	418.05±24.44	405.35±4.13	431.50±5.75	4.521	0.054

Keys: X=mean, SD=Standard Deviation

Table 5. Haematological parameters of sampled participants

Variables	Control (Grp A) X±SD	Paint (Grp B) X±SD	Saw dust (Grp C) X±SD	f - Value	p – Value
FC	2.54±0.70	2.54±0.99	2.37±0.92	2.669	0.054
CRP level	3.06±0.82	2.90±1.19	2.79±1.19	0.864	0.464

Keys: X=mean, SD=Standard Deviation, FC=Fibrinogen Concentration, CRP=C-reactive protein

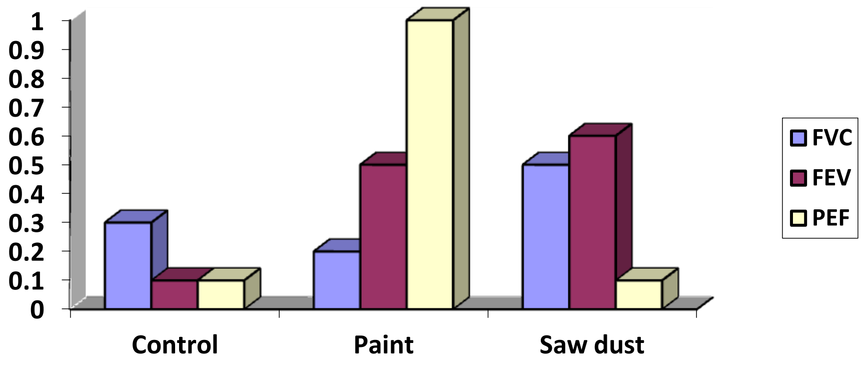

Chart 1. Relationship between participants' duration of work and lung function parameters based on pearson product moment correlation coefficient

Keys: FVC=Forced Vital Capacity, FEV=Forced Expiratory Volume, PEF=Peak Expiratory Flow rate, X=mean

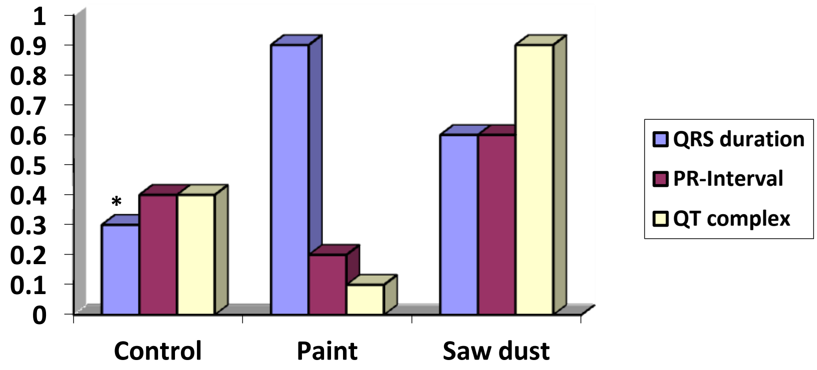

Chart 2. Relationship between participants' work duration and electrocardiogram parameters based on pearson product moment correlation coefficient

*Keys: *correlation is significant at the .05 level*

4.2 Diastolic Blood Pressure and Exposure to Paint Mist and Saw Dust

Results from this study show that changes in diastolic blood pressure (DBP) occurred in 50.5% of the population, revealing that the effect of the size of saw dust (d=0.105) on DBP was small; even though it became high on systolic BP of paint mist and sawdust exposed individuals. Etiologically, the small effect indicates a weak influence of occupational exposure to sawdust on DBP. Therefore, the changes of DBP due to occupational exposure to sawdust in this population-based apparently healthy sample may be clinically irrelevant. However, the high effect size of paint mist indicates a strong influence of DBP. Thus, DBP changes due to occupational exposure to paint mist and sawdust may be of great clinical relevance.

4.3 FVC, FEV, PEV and Exposure to Paint Mist and Saw Dust

FVC is a measure of lung volume and is usually reduced in diseases that cause the lungs to be smaller. Etiologically, the small effect is indicative of a weak influence of occupational exposure to paint mist and sawdust on measures of lung volume (FVC).

This study revealed a significant reduction in FEV and PEV of individuals occupationally exposed to sawdust. However, the reduction in FVC, FEV and PEF were insignificant as compared with control. Contrarily, there was virtually no change in FVC and FEV among those exposed to paint mist, while their PEF was higher but insignificant, compared to control. This explains the fact that various pollutants initiate different responses on lungs functions as also seen in the works of Chan-Yeung and Lee (1986). Chan-Yeung and Lee found no significant changes in FVC of their subjects who were exposed to sawdust, culminating with the fact that such inflammatory alterations did not occur in their subjects as the inert material which was used. Sawdust is probably removed by lung clearance before producing any significant modification of the bronchial mucosa.

For paint mist workers, the study did not find any significant relationship between the duration of occupational exposure to paint mist and lung function parameters. However, the result indicated that the magnitude of the association between FEV and duration of work remained statistically significant; however, with a significant inverse relationship between duration of work and FEV. This finding was consistent with that of Baghdad, 2013 [14].

4.4 QT Interval and QRS Complex in Exposure to Paint Mist and Saw Dust

Table 4 shows the Electrocardiogram profile of participants in the exercise. The study revealed that QTc interval for workers exposed to sawdust had the most prolonged value, while values obtained for control and other workers were nearly the same, with the least values observed for workers exposed to paint mist. However, all values obtained were less than 440ms and were thus within the normal range. Also, the QRS complex had greatest values in workers exposed to cement dust, followed by those exposed to paint mist. Whereas, subjects exposed to sawdust had the least value. Invariably, the results suggest that with reference to control, QRS complex narrowed in workers exposed to paint mist, with the least recorded among those exposed to sawdust.

Previous studies on the daily variation of the particulate nature of air pollution and heart rate variability in elderly subjects showed an increase in heartrate [15] associated with particulate air pollution. Animal data support the concept that the autonomic nervous system may be a target for the adverse effects of air pollution. All of these findings suggest that inhaled particles may affect the balance between sympathetic and parasympathetic control of the heart, and promote a stress response that potentially leads to arrhythmias, which is one of the cardiac death triangles. This view appears corroborated by the findings of this study, suggesting that occupational exposure to airborne pollutants affect the other two components of the “Cardiac Death Triangle”.

Again, a weak positive correlation was established between the QRS complex, P-R interval, QTC duration, and duration of exposure to paint mist among the relevant workers respectively. This implies that as the duration of exposure to these pollutants increased, there is a likelihood of a concomitant increase in the QRS complex, P-R interval, and QT duration respectively. However, the magnitude of the relationship was insignificant.

From Table 1, the physical characteristics and occupational history of participants is shown. Here, age and weight of sampled subjects were seen to be significant ($p < .05$) for paint and saw dust exposed workers as against the control. This was however insignificant for height upon the comparison. The reason for this may not be far-fetched from the impart that toxic air has on metabolic activities. Carbon IV oxide (CO_2) for instance reduces haemoglobin’s affinity for oxygen, lowers transportation of oxygen as such to tissues for oxidative phosphorylation. This thus impedes enzymes of the electron transport chain and affects the processes of glycogenesis, lipogenesis and glycolysis. The resultant implication of this may show up in weight and other anthropometric indices of the victim with time [5].

Tables 2 – 4 respectively shows the cardiovascular, respiratory and ECG profiling of selected participants. As seen, a statistically significant difference was observed for Systolic and Diastolic blood pressures of participants who were exposed to saw dust and paint mist. This however proved insignificant for heart rate upon comparison with control the reason for this may be traceable to the dependency of heart rate on age as against blood pressures [7]. Again, Table 3 observed that other than FVC, FEV and PEV were of statistical significance in paint mist and saw dust exposed workers as compared with control. This finding is in line with that of Kelly, 2011 [14]. Haematological parameters (From Table 4) proved insignificant across the board for saw dust and paint mist exposed workers upon comparison with control. Though the reason for this is quite puzzling, however, Fibrinogen concentration and C - reactive protein levels were each higher in paint mist than saw dust exposed workers. Apparently, Findings from Popkin in 2003 [9] appears to disagree with this.

Chart 1 differentiates between participants' duration of work and Lung function parameters. As seen, paint mist exposed participants had a higher PEF value than those exposed to saw dust. However, upon comparison with control, FVC and FEV were seen to be lower in the paint mist that saw dust exposed workers. The reason for this may be explained by the various concentrations and toxicity levels of various elements that compose inhaled air in any case of the various studied workers.

5. BENEFITS OF STUDY

The study will be of immense clinical value in various ways, including the provision of data that will clearly delineate the cardiovascular and pulmonary variables most affected by exposure to specific industrial pollutants, so that intervention is more precise and reflective of the defined changes in each instance. The study will also provide insight into the exact components of the lung function affected by specific pollutants across the groups. In essence, it will further provide a basis for appropriate policy and regulatory intervention, necessary to protect workers who are at risk of cardiovascular and pulmonary dysfunction due to exposure to airborne particulate pollutants.

6. CONCLUSION

Diastolic blood pressure has been shown to reduce in workers exposed to paint mist, while those exposed to sawdust recorded a significant increase in both systolic and diastolic pressures. Compared to control, Functional capacity (FCV) has also been shown to significantly reduce in workers exposed to paint mist and sawdust. Therefore, the changes in FVC due to occupational exposure to paint mist and saw dust in this population may be of huge clinical relevance.

7. RECOMMENDATIONS

Based on the outcome of this study, we make the following recommendations:

1 Protective devices like aprons, hand gloves, long shoes dust preventive nose and mouth filters/masks are provided to exposed workers
2 Workers should regularly undertake periodic and routine checks for spirometry and ECG parameters in order to identify their susceptibility to hazardous particulate pollutants.

To government agencies, we make the following recommendations:

1 That more attention should be given to the environmental needs of exposed workers
2 Appropriate steps to avoid environmental disasters and adverse should be legislated

CONSENT

As per international standard or university standard, patient's written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard, written approval of Ethics committee has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Balogun JA, Obajuluwa BA, Olaogun MO, Abereoje OK, Oyeyem AY, Adeodu OO, Balogun MO. Anthropometric determinants of resting blood pressures and heart rates of Nigerian school children. Annals of Tropical Pediatric. 1990;10:425-431.
2. Berry PA, Jones SW, Cicuttini FM, Wluka AE, Maciewicz RA. The temporal relationship between serum adipokines, biomarkers of bone and cartilage turnover, and cartilage volume loss in a population with clinical knee osteoarthritis. Arthritis Rheum. 2011;63(3):700–7.
3. Alan R. Schwartz, Susheel P. Patil, Alison M. Laffan, Vsevolod Polotsky, Hartmut Schneider, Philip L. Smith. Obesity and obstructive sleep apnea: Pathogenic mechanisms and therapeutic. Approaches Proceedings of the American Thoracic Society. 2008;5(2):185–192.
4. Escribano BM, Tunez I, Requena F, Rubio MD, Miguel RD, Montilla P, Tovar P, Aguera EI. Effects of an aerobic training program on oxidative stress biomarkers in bulls. Veterinarini Medicina. 2010;55(9): 422-428.
5. Grotle M, Hagen KB, Natvig B, Dahl FA, Kvien TK. Obesity and osteoarthritis in the knee, hip and/or hand: An epidemiological study in the general population with 10

years follow-up. BMC Musculoskeletal Disorder. 2008;9:132–7.

6. Grundy SM. Multifactorial causation of obesity: Implications for prevention. Am J Clin Nutr. 1998;67(Suppl 3):563S–572S.
7. Grundy SM, Hansen B, Smith SC Jr, Cleeman JI, Kahn RA. American Heart Association; National Heart, Lung, and Blood Institute; American Diabetes Association. Clinical management of metabolic syndrome: Report of the American Heart Association/National Heart, Lung, and Blood Institute/American Diabetes Association Conference on Scientific Issues Related to Management. Circulation. 2004;109:551–556.
8. Poirier P, Giles TD, Bray GA, Hong Y, Stern JS, Pi-Sunyer FX, Eckel RH. American Heart Association; Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. Obesity and cardiovascular disease: Patho-physiology, 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. 2006;113:898–918.
9. Popkin B. Dynamics of the nutrition transition and its implications for the developing world. Forum Nutr. 2003;56: 262-264.
10. Canadian Physical Activity, Fitness & Life Style Approach. (CPAFLA). CSEP-Health & Fitness Program's Health-Related Appraisal and Counselling Strategy, Canadian Society for Exercise Physiology and Health Canada, Ottawa, Canada, 3rd Edition; 2003.
11. Caterson ID, Hubbard V, Bray GA, Grunstein R, Hansen BC, Hong Y, Labarthe D, Seidell JC, Smith SC Jr. American Heart Association. Prevention Conference VII: Obesity, a worldwide epidemic related to heart disease and stroke: Group III: Worldwide comorbidities of obesity. Circulation. 2004;110:e476–e483.
12. Morimoto LM, White E, Chen Z, Chlebowski RT, Hays J, Kuller L, Lopez AM, Manson J, Margolis KL, Muti PC, Stefanick ML, McTiernan A. Obesity, body size, and risk of postmenopausal breast cancer: The Women's Health Initiative (United States). Cancer Causes Control. 2002;13:741–751.
13. Church T. Exercise in obesity, metabolic syndrome, and diabetes. Progress in Cardiovascular Diseases. 2011;53(6):412–418.
14. Manning Kelly. Effects of exercise training on fat oxidation in untrained overweight and obese females. Dissertation Georgia State University; 2011.
15. Gold AR, Schwartz AR, Wise RA, Smith, PL. Pulmonary function and respiratory chemo-sensitivity in moderately obese patients with sleep apnea. Chest. 1993;103:1325–1329.

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