EFFECTS OF TRADITIONAL WATER PIPE SMOKING ON PULMONARY FUNCTIONS AND CARDIOVASCULAR INDICES IN DITA WOREDÁ, GAMO GOFA ZONE, SNNPR, ETHIOPIA

A THESIS SUBMITTED TO THE SCHOOL OF GRADUATE STUDIES, ADDIS ABABA UNIVERSITY IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF SCIENCES IN MEDICAL PHYSIOLOGY

BY

HENOK TOGA

JULY, 2018
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ADVISOR: DIRESIBACHEW HAILE (PhD)

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This thesis is my original work, has not been presented as a thesis work for a degree in this or any other university and that all sources of material used for the thesis have been duly acknowledged.

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Abbreviations and Acronyms

ATS/ERS = American Thoracic Society/European Respiratory Society

CO = Carbon monoxide

COPD = Chronic Obstructive Pulmonary Disease

DBP = Diastolic Blood Pressure

$\text{FEF}_{25\%-75\%}$ = Forced Expiratory Flow between 25% and 75% of Forced Vital Capacity

$\text{FEV}_1$ = Forced Expiratory Volume at first second

$\text{FEV}_{1\%}$ = The Ratio of Forced Expiratory Volume at first second to Forced Vital Capacity $\times 100$

$\text{FVC}$ = Forced Vital Capacity

MAP = Mean Arterial Pressure

PAH = Polycyclic Aromatic Hydrocarbon

PEF = Peak Expiratory Flow

PFT = Pulmonary Function Test

PP = Pulse Pressure

PR = Pulse rate

SBP = Systolic Blood Pressure

SNNPR = Southern Nations Nationalities and Peoples Region

$\text{SpO}_2$ = Oxygen Saturation

WP = Water Pipe

WPS = Water Pipe Smoking
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ABSTRACT

Background: Tobacco use and exposure is a global public health risk causing more than 7 million preventable deaths per year globally; approximately one person dies every five seconds due to tobacco. Water pipe smoking (WPS), also known as gaya (in Ethiopia), is a form of tobacco use engaged by men and women. Water pipe tobacco smoke contains many toxicants that are known to cause diseases and deaths. The objective of this study was to investigate the effects of traditional WPS on pulmonary functions (FVC, FEV$_1$, FEV$_1\%$, PEF and FEF$_{25\%-75\%}$) and cardiovascular indices (SBP, DBP, PR and SpO$_2$) in Dita woreda, Gamo Gofa zone, Southern Ethiopia.

Methods: A comparative cross sectional study was employed. A total of 47 WP smokers and 47 age, weight, height and sex matched controls were investigated. Their lung function was measured with digital spirometer (Spiro-Pro JAEGER). A standardized questionnaire based on British Medical Research Council Questionnaire was used to assess respiratory symptoms. Furthermore, WHO standardized questionnaire was used to assess physical activity status. Aneroid sphygmomanometer was used to measure blood pressure. Portable pulse oximeter (Nellcor N-65) was used to measure SpO$_2$ and PR.

Results: Respiratory symptoms were significantly higher among WP smokers compared with non-smokers. Lung function indices (FVC, FEV$_1$, FEV$_1\%$, PEF and FEF$_{25\%-75\%}$) were significantly higher in non-smokers than WP smokers ($p<0.05$). The percentages of predicted values of all lung function indices were lower in WP smoker than non-smokers and the predicted percentage of FVC, FEV$_1$, PEF and FEF$_{25\%-75\%}$ between the groups were found statistically significant ($p<0.05$). SBP and PP were significantly higher in WP smokers than non-smokers.

Conclusions: The results showed that higher frequency of respiratory symptoms, reduction of oxygen saturation, accession of cardiovascular parameters (SBP, DBP, MAP, PP and PR) and reduction of all PFT in WP smokers when compared with those non-smokers.
1. INTRODUCTION

1.1 Background

Tobacco use and exposure is a global public health risk causing more than 7 million preventable deaths per year globally; approximately one person dies every five seconds due to tobacco [1]. Tobacco use is the leading cause of preventable death and disability in the world. If current trends continue, by 2030 tobacco will kill more than 8 million people worldwide each year, with 80% of these deaths among people living in low and middle income countries [2]. Water pipe smoking (WPS), also known as the hookah (Arab and Britain), shisha (German), narghile (Armenia and Bulgaria), hubble-bubble (India), goza (Egypt), chichi (France), qalyan (Persia), madaa (Yemen) and gaya (Ethiopia), is a form of tobacco use engaged in by men and women in the Middle East, Africa, and Asia [3]. It is a symbol of social sharing and cultural identity in the Middle East [4]. Since the early 1990s, there has been a significant increase in its use around the world and it is estimated that there are more than 100 million people worldwide who smoke water pipe daily [5].

Even though WPS is a common practice globally today, only few countries regularly conduct surveillance of WPS. As result, determining current WPS prevalence rates in many countries especially in Africa would be difficult. One study conducted in Egypt indicated that the prevalence of WPS was 6.2% in the country [6]. Despite the growing burden of this public health problem, high-quality trials looking at the long-term effects of water pipe smoking are still lacking [7].

Water pipe tobacco smoke contains many toxicants that are known to cause diseases and deaths. Exposure to these toxicants is associated with a variety of adverse health consequences including pulmonary and cardiovascular diseases [8]. Many of the toxicants found in water pipe tobacco smoke generated from a single session are found in significantly higher concentrations than in smoke generated from a single cigarette [9, 10]. A single water pipe tobacco smoking session produced an average of 2.5 times more phenanthrene, 4 times more fluoranthene, and 2.5 times more chrysene compared to the amount of toxins found in the smoke from a single cigarette [9]. The amount of tar, nicotine and CO are higher in WP smoke when compared with cigarette smoke [9]. Another study found that a single WPS session produced 27 times higher
formaldehyde in the mainstream smoke than what is produced from smoking a single tobacco cigarette [10]. In any case the amount of any toxicant contained in the smoke from a single water pipe tobacco smoking session is more than the amount of toxicant contained in the smoke from a single cigarette.

WPS is associated with acute and long-term health effects [7, 11]. In terms of acute effects, WPS can, in some cases, cause CO intoxication [12–19]. CO poisoning is a potential risk of WPS because of the high concentrations of CO found in mainstream and side stream water pipe tobacco smoke and greater volumes of smoke inhaled per puff and per session [9, 20–23].

In terms of the health effects associated with long term use, WPS is associated with many chronic health effects on respiratory and cardiovascular system. Most studies suggest that WPS significantly decreases pulmonary functions [24–31] and significantly increases cardiovascular indices [31–40], but some other studies show that there is no statistical significance between WP smokers and non-smokers on pulmonary function parameters [41, 42]. Indeed, a variety of adverse health consequences of water pipe smoking have been documented including chronic pulmonary health conditions [7]. WPS has been linked with lung cancer [43–45]. Being former water pipe tobacco smokers were associated with a 6 times greater risk of lung cancer compared to non-former water pipe tobacco smokers [43]. Chronic obstructive pulmonary disease (COPD) has also been linked with water pipe smoking [46–49]. Water pipe tobacco smokers were 2.5 times as likely as non-water pipe smokers to have COPD [49]. More dependent water pipe tobacco smokers may be at increased risk for certain chronic health conditions. Dependent water pipe tobacco smokers were 3.7 times more likely to have chronic bronchitis than non-dependent water pipe smokers [50]. Nicotine dependence maintains WPS for long-term [51-55]. Also like cigarette smokers, some frequent water pipe tobacco smokers report WPS within the first hour after waking, smoking a water pipe even when feeling ill, and finding abstaining from water pipe smoking even for less than a day between sessions is difficult [51].

WPS has also been associated with cardiovascular diseases. One study reported that participants with WPS experience had a three times greater risk of coronary artery disease compared to non-water pipe smokers [55]. The current study was intended to investigate the long-term effects of water pipe smoking on cardiovascular indices and pulmonary functions in Dita woreda, Gamo Gofa zone, Southern Ethiopia.
1.1.1 History

The history of water pipe goes back to around four centuries ago and its use has been a traditional habit in Asia and North Africa [56]. Water pipe has been used to smoke tobacco and other substances, such as flowers, spices, fruits, coffee, marijuana or hashish, by the indigenous people of Africa and Asia for at least four centuries [57]. Their origin is somewhat nebulous, but it is known that trade routes through India and China helped to disseminate the practice throughout parts of Asia, the Middle East and Africa [58].

It is not known exactly when tobacco was introduced to Africa. However, it is generally accepted that the Portuguese were the first to take tobacco to Africa, probably sometime in the early to mid-sixteenth century. Following its introduction along the east and west coasts, tobacco spread into the interior of Africa rapidly. Its cultivation moved quickly eastward from Senegal, along the trade routes of interior West Africa during the latter half of the sixteenth century, and in east-central Africa by the mid-seventeenth century [59].

Tobacco was introduced into other parts of Africa no later than the middle of the seventeenth century. It apparently spread into the interior of the Saharan region of North Africa, after being introduced by the Portuguese. The Portuguese also traded tobacco in the northeast region of Africa, where it was grown extensively in Ethiopia. Iain Gately, who is the author of a book entitled “Tobacco: A Cultural History of How an Exotic Plant Seduced Civilization” has explained the rapid dispersal of tobacco throughout the African continent during that time was due to pipe and smoking cultures had already been practiced by Africans prior to the introduction of tobacco during the sixteenth and seventeenth centuries. Gately argues that herbal fumes had long been drunk by African peoples [60].

In 1600, many European countries including Britain, France, Spain, Portugal, Germany, and Dutch were in political and economic competition for settlement in New World (South America), Brazil, Venezuela and Virginia. In 1612 colonialists started tobacco cultivation in the new world and the cultivation spread and intensified across Europe. The cultivation started in 1619 in England and subsequently, in 1620 in France and Germany [57]. Before 1865, tobacco plantations in the South America were usually worked by slave [59].
Tobacco has been consumed and cultivated in Africa since the end of the sixteenth century. It was not, however, until the nineteenth century that commercial cultivation began. North Africa, especially Algeria, was the main producer in the nineteenth and a good part of the twentieth century, and almost all of the output was exported to France. The Cape Colony in South Africa cultivated tobacco from as early as 1657, but production was meager. Elsewhere in Central and Southern Africa tobacco cultivation did not begin until the end of the nineteenth century, and started in the British colonies. In 1980 the African continent accounted for only 6 per cent of world output, the leading producers, in order, being Zimbabwe, Malawi and South Africa. What is interesting about African tobacco production in general is the extent to which tobacco played a similar role in settlement of colonialists to that which it had in the New World. The British South African Company worked hard to stimulate production and, by association, European farming and settlement. The control over tobacco production by Europeans was reflected in South African, Zimbabwe, Zambia and Malawi. Throughout the nineteenth and twentieth centuries Brazil has been the chief producer [57].

From the mid-seventeenth century to the early decades of the nineteenth century, tobacco played an important role in the transatlantic trade. The primary areas concerned were Brazil in South America, Ghana in West Africa, the Congo River basin and Angola. In the period between 1637 and 1642, the Dutch replaced the Portuguese as the principal European tobacco trading power along the West African coast. Portuguese ships from Brazil were obliged to stop at the Dutch fort at Elmina, in present day Ghana, to have their cargoes inspected and to pay a tax of ten percent of their tobacco. The Portuguese needed to procure African slaves for their plantations in Brazil, and the Dutch got the Bahian tobacco they required to trade with local Africans. That trade could be very profitable. For instance, in the first decades of the eighteenth century Africans were willing to trade a pound of ivory for every ten pounds of Bahian tobacco. By the eighteenth century tobacco was not only an important part of trade relations between Africans and Europeans; it had also become an important component of trade between African societies. For example, in the Congo River basin tobacco was grown by Africans who traded it, along with locally produced alcohol, for salt, cassava, and palm cooking oil [59].
1.1.2 Description of Traditional Water Pipe

A water pipe has four main components: the head, body, bowl, and hose. A head where tobacco is placed and heated by charcoal; a bowl, which is partially filled with water; a body connecting the head to the bowl by a tube that carries the smoke downward into the water; and a hose with a mouthpiece through which the smoke is drawn from the bowl [61].

Holes in the bottom of the head allow smoke to pass into the body’s central conduit. This conduit is submerged in the water that half-fills the water bowl. The hose is not submerged, exits from the water bowl’s top, and ends with a mouthpiece, from which the smoker inhales. A water pipe is prepared for tobacco smoking by filling the head with tobacco. The tobacco-filled head is covered with perforated clay foil onto which a little piece of charcoal is placed. Most of the time tobacco that is placed into the head is moist; it does not burn in a self-sustaining manner. Thus, charcoal is placed atop the tobacco-filled head. When the head is loaded and the charcoal lit, a smoker inhales through the hose, creating a vacuum above the water, and drawing air through the body from the tobacco and charcoal [8, 61].

When the user inhales from the mouthpiece, vacuum is created in the water bowl, sucking smoke from the head through the body into water bowl. The passage of smoke through the water causes bubbles. When the water pipe smoker inhales on the hose, charcoal heated air is drawn across the tobacco, thus producing the mainstream aerosol. This aerosol then passes through the body of the pipe, bubbles through the water in the bowl, travels the length of the hose, and finally, the user inhales the resulting smoke via the hose [61].
1.1.3 Parts of Traditional Water Pipe

Fig. 1. Schematic Diagram of Traditional Water Pipe Parts
Fig.2. A, B, C and D were photographs showing different parts of traditional water pipe. The pictures were taken from Dita woreda, Southern Ethiopia. January 2018.
Fig. 3. Men Smoking Traditional Water Pipe in Group. The picture was taken from Dita woreda, Southern Ethiopia. January 2018.
1.1.4 The Tobacco Plant

*Nicotina Tabacum Lancifolia* belongs to family *Solanaceae*. *Nicotina Tabacum Lancifolia* is cultivated in home gardens or in specialized tobacco-farms at an altitude of 300-2400m above sea level. Parts of Ethiopia it grows includes; Eastern Wollega, Gedio, West Gambella, Keffa, Gamo Gofa, Sidama and Harere. Originally it is indigenous in temperate South America. Now it is widely cultivated, escaped and naturalized throughout the warm parts of the world [62].

*Nicotina Tabacum Lancifolia* is an herb up to 2 m or more tall. Most parts are viscid and with numerous glandular hairs. Its leaves are alternate, not clustered, regularly decreasing in size towards the inflorescence; petiole 1-2 cm long, broadly winged. Flowers are many in much-branched terminal panicles, up to 25 cm long. Pedicels are 0.8-2 cm long. Calyces are 1.5-2.5 cm long; teeth unequal, acuminate, 0.6-1 cm long. Corolla greenish white, often pinkish in upper part, funnel-shaped, 3.5-5.5 cm long, with broadly triangular or rounded, unequal lobs, 0.3-0.6 cm long. Stamens are unequal, hardly exceeding the corolla tube. Capsules are ellipsoid or ovoid, 1.5-2 cm long. Seeds=0.5 mm in diameter [62].

1.1.5 Local Procedure of Preparing the Plant for Smoking

The leaf is collected from the plant, chopped with knife and boiled in pitcher for thirty minute. After thirty minute, it is taken out from the stove and placed for one week without opening the pitcher covering. After one week, the boiled leaf will be squeezed manually and remains within the pitcher for additional one week. Before it is ready for smoking, it is exposed to the sunlight for at least one day.
A. Nicotina Tabacum Lancifolia Plant

B. Nicotina Tabacum Lancifolia Prepared for Smoking

Fig.4. A and B were photographs showing Nicotina Tabacum Lancifolia plant and the leaf prepared for smoking. The picture was taken from Dita woreda, Southern Ethiopia. January 2018.
1.2 Statement of the Problem

WPS is spreading worldwide rapidly and various factors have contributed to the rapid spread of WPS. Most WP smokers perceive WPS as less harmful and less addictive than cigarette smoking. They consider that harmful substances are being filtered out through the water bowl, and think that WPS is more socially acceptable than cigarettes and it represents a good opportunity for gathering of friends and family [63, 64]. Factors for the increased WPS in Ethiopia are: Chat chewing experience as a precursor of water pipe tobacco smoking; less expensive; easy accessibility; attractive aroma of tobacco; lack of knowledge about its effect; absence of effective policy and source of income for lounge owners [65]. Anecdotal information shows that traditional WP smoking is prevalent in different parts of Ethiopia especially in Southern Ethiopia and Gambela. Even though it is widely distributed in many parts of the country, yet studies showing its impact on heath in Ethiopia remain limited. Furthermore, most studies globally have focused on the acute respiratory and cardiovascular effects of WPS. Few studies were done on its long-term (chronic) respiratory and cardiovascular effects.
1.3 Significance of the Study

According to WHO advisory note on water pipe tobacco smoking, research on water pipe use in Africa is limited [5]. Yet, no study has been done on the effects of water pipe smoking on pulmonary functions and cardiovascular indices in Ethiopia. Only three empirical studies in South Africa were conducted among students. Anecdotal evidence for Ethiopia, Kenya, Sudan, Uganda, Tanzania, Nigeria and Algeria indicates proliferation of fashionable water pipe bars in the larger urban centers in all these countries, visited mainly by the young and business people.

Cigarette smoking is known to have physiological effects on different systems. It is a cause for COPD, lung cancer, bronchitis, cardiovascular diseases, oral diseases and GIT disorders, but little is known about water pipe smoking especially in our country Ethiopia. Most people believe that WPS is less harmful than cigarette smoking.

To date no data were available on the chronic cardiovascular impact of WP smoke exposure. Such study is essential because as it would allow examining the exclusive effect of WP smoking and, hence, provide plausibility for reports describing the long-term cardiovascular adverse effects of WP smoking. Consequently, the present study was intended to verify the long-term effects of water pipe smoking on cardiovascular indices (SBP, DBP, SpO₂ and PR) as well as pulmonary functions (FVC, FEV₁, FEV₁%, PEF and FEF₂₅%-₇₅%) in Dita woreda, Gamo Gofa zone, Southern Ethiopia.

This research investigated the effects of water pipe smoking on pulmonary functions and cardiovascular indices. The data obtained from the study is expected to show the magnitude of the risk and will be used as a baseline for further studies as well as for developing prevention and cessation strategies of water pipe smoking.
2. LITERATURE REVIEW

2.1 Toxicants in Water Pipe Tobacco Smoke

As burning charcoal is usually used as the heat source in water pipes, the smoke contains toxicants emitted from both the charcoal and the tobacco product. Thus, the composition of both the charcoal and the tobacco can influence the toxicant contents of the smoke. Laboratory studies during the past decade with the use of modern analytical methods and reliable machine smoke generation and sampling protocols have begun to elucidate the toxicant content of water pipe smoke [66].

The volume of smoke inhaled during water pipe smoking session is much more than that of cigarette smoke. The large volume is due to longer session’s duration and large number of high volume puffs taken during each session. WPS session may last 45 minutes, much longer than approximately 5 minutes taken for cigarette smoking. The number of puff is as many as 76-269 puffs, compared to approximately 10 from cigarette smoking. Each puff from a WP involves inhalation of approximately 768 – 899 ml of smoke, compared to approximately 50 ml for cigarette smoke [20, 53, 67, 68].

In addition to its large volume, water pipe tobacco smoke contains many toxicants that can cause disability, disease, and death. These toxicants include carbon monoxide [9, 20, 21], polycyclic aromatic hydrocarbons (PAHs; [21, 69–71]), volatile aldehydes [72–74], nicotine [9, 20], nitrosamines and primary aromatic amines [9, 75, 76], furanic compounds [77], phenolic compounds [78], ultrafine particles [72], and even radioactive substances [79] and heavy metals [80]. The chemical composition of water pipe smoke produced from tobacco includes: arsenic, cobalt, chromium, lead, nickel and beryllium [80, 81].

The nicotine content of water pipe tobacco has been reported to be 2% to 4%, in comparison with 1% to 3% for cigarettes [24]. The concentration of carbon monoxide is 0.34% to 1.40% for water pipe smoke and 0.41% for cigarette smoke [82]. The mean carboxyhemoglobin concentrations were higher among water pipe smokers (10.1%) than among cigarette smokers (6.5%) or non-smokers (1.6%), and a linear relationship was found between smoking intensity and carboxyhemoglobin concentration [83]. Similarly another study shows nicotine and cotinine
in water pipe smokers found higher after smoking. After a single 45 minute smoking session, the mean plasma concentration of nicotine rose from 1.11 to 60.31 ng/ml, and cotinine rose from 0.79 to 51.95 ng/ml [84]. Another study of water pipe smokers found nicotine and cotinine increased up to 250% and 120%, respectively, after a typical 40 minute to 45 minute smoking session. WP use may increase exposure to carcinogens because smokers use a WP over a much longer period of time, often 40 to 45 minutes, rather than 5 to 10 minutes it takes to smoke a cigarette. Because of the longer, more sustained period of inhalation and exposure, water pipe smokers may inhale as much smoke as consuming 100 or more cigarettes [85].

There are numerous factors that can affect the smoke produced from a water pipe, including the type of coal, the quality of the tobacco, the volume of water used, the design of water pipe itself, the number/duration/volume of the puffs, and the length of the water pipe sessions and the number of users sharing a water pipe [86]. Tar content ranged from 242 to 2359 mg per smoking session, nicotine levels ranged from 1.04 to 7.75 mg per session [87], and the amount of CO ranged from 57.2 to 367 mg per session [22]. CO levels are directly proportional to the amount of charcoal used [22]. A meta-analysis study of water pipe and cigarette toxicant exposure suggested that these values roughly correspond to a session of water pipe tobacco being equivalent to 25 cigarettes worth of tar, 11 cigarettes worth of CO, and 2 cigarettes worth of nicotine [23].
2.2 Health Effects of Water Pipe Smoking

The American lung association called water pipe as “An emerging deadly trend”. Water pipe smoking results in variety of adverse health consequences, including pulmonary and cardiovascular diseases [88].

2.2.1 Effects of Water Pipe on Pulmonary System

Research identified the acute and long-term health effects associated with WPS. WPS for thirty minutes is associated with an increase in respiratory rate by 2 breaths per minutes [33]. Another study showed an acute decrease in PEF after 30 min of WPS [32]. In one study, involving 47 volunteers in Israel with an average age of 25 years, carboxyhemoglobin level increased after a water pipe tobacco smoking session, with 6 volunteers having a rise of 25% and 2 with a 40% increase [34]. Furthermore, study looking at pulmonary function, respiratory rate and oxygen consumption before and after water pipe tobacco smoking sessions showed that oxygen consumption decreased from 1.9 to 1.7 L/min [89]. Therefore, acute water pipe tobacco smoking does appear to cause an impairment of lung function and reduce oxygen consumption at both molecular and physiological levels.

Studies examining the effect of WPS on pulmonary function parameters reported mixed results. In most studies pulmonary function was impaired when measured by FVC [26, 29], FEV$_1$ [26, 28, 29, 31], FEV$_1\%$ [25,28–30], FEF$_{25\%-75\%}$ [28, 32] and PEF [24, 31] while other studies did not show impairment of these pulmonary function parameters [41, 42].

Kiter et al. [24] compared lung function of subjects categorized in four groups: exclusive water pipe smokers, present water pipe smokers who smoked cigarettes in the past, cigarettes smokers, and a control group of non-smokers. The study demonstrated a reduction in lung function tests among all the smokers. When compared with non-smokers, there was a statistically significant decrease in PEF of water pipe smokers and FEV$_1\%$ value was found to be significantly decreased in water pipe smokers when compared with passive smokers [25].

Several studies evaluated the long-term effects of WPS on pulmonary function tests. Most of them reported impairment of pulmonary function parameters after long-term WPS. WPS had significantly lowered FVC, FEV$_1$ and PEF [24].
Matched to age, height, weight and ethnicity there was a significant decline in the lung function parameters of water pipe smokers including FEV\textsubscript{1} and FEV\textsubscript{1\%} when compared to non-water pipe smoker subjects [26]. In comparison with non-smokers, there is statistically significant decrease in PEF of water pipe smokers [90].

Carbon monoxide and pulmonary function changes have also been reported in long-term water pipe tobacco smokers. In one cross-sectional study in Pakistan, blood CO concentration was significantly higher in water pipe tobacco smokers (10.5\%) compared with cigarette smokers (6.2\%) and non-smokers (0.9\%). Oxyhaemoglobin levels were significantly lower in water pipe tobacco smokers compared with cigarette smokers and non-smokers [91].

WPS is also associated with respiratory symptoms. A significantly greater proportion of WPS reported to have any respiratory symptoms (e.g. bringing up phlegm, having shortness of breath upon exertion, cough, chest illness in the past three years that kept a participant off work, and coughing with phlegm that lasted at least three weeks) [89]. The association between WPS and chronic obstructive pulmonary disease (COPD) symptoms or chronic bronchitis was studied. In one study that included 62,086 smokers (2174 WP smokers) from 11 countries in the Middle East and North Africa [48], WPS contributed to the development of productive cough, dyspnea and chronic bronchitis. The relationship of WPS and development of chronic bronchitis was also studied in 833 Lebanese subjects, 274 cases of chronic bronchitis (using the standard definition of chronic cough with sputum production for 3 consecutive months for 2 years) and 559 controls without the condition aged ≥40 years. The results indicated that the odds of having bronchitis were 5.7 times higher in water pipe tobacco smokers compared with non-smokers. Previous WPS were significantly associated with chronic bronchitis [50]. Contrarily, two studies found no association between WPS and COPD [46, 92].
2.2.2 Effects of Water Pipe on Cardiovascular System

Both the acute and chronic cardiovascular health effects of water pipe tobacco smokers have been documented. Most studies showed the acute effects of WPS on HR, SBP and DBP were statistically significant between water pipe smokers and non-smokers [31–40].

For short-term cardiovascular effects, studies have generally measured heart rate and blood pressure before and after smoking sessions with a period of abstinence before the study. In one study, after 1 session of water pipe tobacco smoking, the systolic and diastolic blood pressure levels of 45 men and women (average age: 32 years) was significantly increased after smoking (systolic: 120 vs. 132 mm Hg, diastolic: 75 vs. 83 mm Hg). Heart rates were increased from 80 to 96 beats per minute [32]. In other study, WPS for thirty minutes is associated with an increase in systolic blood pressure of 12-16 mmHg, an increase in diastolic blood pressure of 2-8 mmHg and a rise in heart rate by 6-15 beats per minute [33]. Study by Murtaza et al. [38] also showed that mean arterial blood pressure increased from 96 mmHg to 108 mmHg and heart rate increased from 77 to 91 beats per minute [38]. Acute short-term water pipe smoking elicits a modest increase in heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial blood pressure [36]. SBP, DBP and HR were significantly increased in water pipe smokers after 15 minutes of smoking compared with non-smoker [93].

Long-term cardiovascular effects were also showed that SBP, DBP and HR were higher among WP smokers than non-smokers [94, 95]. SBP and HR were significantly higher in WP smokers than those non-smokers [94, 96].
3. OBJECTIVES

3.1 General Objective:

➢ To investigate the effects of water pipe smoking on pulmonary functions and cardiovascular indices

3.2 Specific Objectives:

➢ To compare respiratory symptoms of water pipe smokers and non-smokers
➢ To compare pulmonary functions of water pipe smokers and non-smokers
➢ To compare cardiovascular indices of water pipe smokers and non-smokers
4. MATERIALS AND METHODS

4.1 Materials

4.1.1 Study Area

The study was conducted in Dita woreda, located 57 kilometers north of Arbaminch. Arba Minch town is located in Gamo Gofa Zone, Southern Nations Nationalities and Peoples Region (SNNPR) at a distance of 435.4 km from Addis Ababa. Dita woreda is one of the fourteen woredas in Gamo Gofa Zone. It has a total population of 108,954.

4.1.2 Study Subjects

4.1.2.1 Study Group: The study was conducted in 47 exclusive water pipe smokers selected from the study area. Each subject who participated in this study was selected based on the inclusion criteria and after informed consent was obtained.

4.1.2.2 Control Group: A total of 47 volunteers from the same area who have been free from exposure to water pipe smoke were selected as a control group. These were healthy individuals who had similar characteristics (sex, age, weight and height) with the study groups.

4.1.3 Study Equipments

The following equipments were utilized in the study:

- Spiro meter - a pocket size digital Spiro meter called Spiro Pro made by JAEGER (Germany) was used to measure lung function parameters.
- Digital balance and measuring tape were used to measure weight and height respectively.
- A standard questionnaire based on British Medical Research Council (BMRC) questionnaire format was used to assess respiratory symptoms and past illness.
- WHO standardized questionnaire was used to assess the physical activity status.
- Aneroid sphygmomanometer was used to measure blood pressure.
- Pulse oximeter (Nellcor N-65 Ireland) was used to measure oxygen saturation and pulse rate.
4.1.4 Inclusion and Exclusion Criteria

4.1.4.1 Study Subjects

4.1.4.1.1 Inclusion Criteria

To avoid misinterpretation of the data, only men, exclusive water pipe smoker (non-cigarette smokers), clinically stable and free of comorbidities were included.

4.1.4.1.2 Exclusion Criteria

Women were not included in this study despite the fact that there were women smokers, because women were exposed to smokes and pollutants from biomass fuels. Smokers aged less than 20 and over 60 were excluded due to the fact that aging affect pulmonary function and cardiovascular system. Mixed smokers who smoke both water pipe and cigarette were excluded. Smokers with imperfect performance of the respiratory maneuvers were also excluded. Individuals who had history of alcohol drinking and who are currently drinking were excluded.

4.1.4.2 Control Subjects

4.1.4.2.1 Inclusion Criteria

Men, who were non-smokers and non-exposed for passive smoking (second hand smoking) with similar age range with study subjects were included.

4.1.4.2.2 Exclusion Criteria

Individuals who had probable exposure to water pipe smoke (e.g., family members of water pipe smoker). Individuals who had history of alcohol drinking and who are current drinkers were excluded.
4.2 Methods

4.2.1 Ethical Consideration

Before data collection, ethical clearance was obtained from ethics and research committee of the department of Medical Physiology. Additionally, permission to conduct the study in the areas was also obtained from Dita Woreda Health Office.

The study participants were informed about the study as it was non-invasive and the procedure might have little inconvenience while doing the spirometric maneuvers but is with no unwanted side effects. All participants were provided written informed consent with the option to withdraw from participation without any precondition at any time. Eventually, only those gave their oral and written informed consent were included in the study. Each participant had a unique code that was used in the study.

They were also being told to have the right to ask for the deletion of all the collected data related to him from the database.

4.2.2 Study Design

Comparative cross sectional study design was employed.

4.2.3 Sampling Method and Sample Size Determination

There are 24 kebeles in Dita woreda. Out of 24 kebeles, 4 kebeles (Giyassa, Lisha, Egirssa Woke and Donne) were selected by simple random sampling method. From 3 kebeles (Giyassa, Lisha and Egirssa Woke), 12 WP smokers were selected from each (a total of 36) and 11 WP smokers were selected from Donne kebele. Similar number of non-smokers who meet the inclusion criteria for the control subjects were selected from the aformentioned kebeles.

To determine the sample size, the prevalence value for exposed group was 32.3% and for control group was 6.3%, taken from previously published article that was done in Lebanon [50]. Sample size was calculated using the following formula:
n (in each group) = \((P_1q_1 + P_2q_2) ((z_{1-a/2} + z_1) - \alpha/2)^2 \)
\((P_1-P_2)^2\)

Where:

\(P_1\) = Prevalence of exposed group = 32.3% = 0.323

\(q_1 = 1-P_1\) = 0.677

\(P_2\) = Prevalence of control group = 6% = 0.063

\(q_2 = 1-P_2\) = 0.937

\(\alpha\) = the significance level = 0.05, \(\beta = 0.1\)

\(1 - \beta\) = the power of test

\(z_{(1-a/2)} = 1.96\) = value of the standard normal distribution corresponding to 0.05 significance level.

\(z_{(1-\beta)} = 1.28\) = value of the standard normal distribution corresponding to the 90% level of power

\(n\) (in each group) = \(((0.323 \times 0.677) + (0.063 \times 0.937)) (1.96 + 1.28)^2 \)
\((0.323 - 0.063)^2\)

\(= 0.277 \times 10.5\)

0.0676

\(= 43\)

Based on the calculation, the determined sample size in each group was 43 and assuming non-response rate, 10% of the calculated sample size was added.

Therefore, \(43 \times 10\% = 4.3 \approx 4\)

\(n\) (in each group) = 43 + 4 = 47

The total sample size was 94; 47 for study subject and 47 for control group.
4.2.4 Data Collection Methods

4.2.4.1 Questionnaire

British Medical Research Council Questionnaire (BMRC), were used to assess respiratory symptoms and past illness. WHO standardized questionnaire was used to assess the physical activity status. Before the interview, detail explanation was given to the subjects about the purpose of the study and procedures to be undertaken. All the participants can listen and speak Amharic. The questionnaire was then translated to Amharic language.

4.2.4.2 Anthropometric Measurements

Weight and height of both the study and control subjects were measured with light clothing to the nearest one kilogram and without shoes to the nearest one centimeter, respectively.

4.2.4.3 Lung Function Measurements

Spirometric measurement was done in both control and exposed subjects. FVC, FEV$_1$, FEV$_{1\%}$, PEF and FEF$_{25\%}$-$75\%$ were measured by using digital spirometer. Before performing the actual procedure of the test, data relevant to the study subject including sex, age, height, and weight were entered in to the spirometer. Subsequently, the subject was familiarized to the procedure. Before making the actual measurement, the subject has taken about 10-15 minutes of rest. After putting a nose clip, the subject was instructed to breath normally for a while, take full inspiration followed by expiration as hard and as fast as possible into the spirometer. Based on ATS/ERS task force on standardization of lung function tasting [97], all measurements were made with the subject in a sitting position. The subjects had not smoked during or 12 hours prior to the interview and lung function testing. All tests were performed in the morning between 8 am and 10 am. Each subject performed three acceptable PFT. At least three readings were taken by giving three to five minutes rest in between the maneuvers and the highest readings were taken. Percentage predicted values for spirometry measurement were based on age, gender and height as per the international reference values provided by the manufacturer.
4.2.4.4 Measurements of Cardiovascular Indices

Cardiovascular indices including SBP, DBP, PR and SpO₂ were measured in both control and study groups. Subjects were ordered not to take alcohol and caffeine 12 hours before measurement. During the measurement, the subject, sat in a chair with his back supported, legs uncrossed and feet on the floor. He was also instructed to loosen any tight clothing or remove long sleeved garments to access upper arm easily. Arms were supported so that the upper arm was at the level of heart and took rest for 5 to 10 minutes before starting the procedure. SpO₂ and PR were measured using pulse oximeter which involves inserting a subject’s index finger in to the device. Factors that can affect accurate pulse oximeter reading such as nail varnish, bright light (sun light), patient’s movement and improper probe positionig were checked and care was taken.

4.3 Study Variables

<table>
<thead>
<tr>
<th>Dependent Variables:</th>
<th>Independent Variables:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory symptoms</td>
<td>- Age</td>
</tr>
<tr>
<td>Lung function test indices</td>
<td>- Duration of water pipe smoking</td>
</tr>
<tr>
<td>Cardiovascular indices</td>
<td>- Anthropometric values</td>
</tr>
<tr>
<td></td>
<td>- Physical activity level</td>
</tr>
</tbody>
</table>
4.4 Statistical Analysis and Interpretation of the Data

Statistical Package for Social Science (SPSS) version 21 was used to analyze the data. Descriptive statistics was used to summarize years of smoking and anthropometric measurements of subjects. Odds ratio with 95% confidence intervals was calculated to compare the prevalence of respiratory symptoms and respiratory diseases in case and controls. Independent sample t-test was applied to compare the anthropometric variables, mean respiratory and cardiovascular values of exposed and non-exposed groups. P< 0.05 was taken for significance difference. Pearson correlation was used to assess the relationship between age and lung function indices, duration of smoking (in years) and lung function measurements and duration of smoking (in years) and cardiovascular indices.

4.5 Result Dissemination

The final result of the thesis will be disseminated to AAU Department of Medical Physiology and AAU School of Postgraduate Studies.
5. RESULTS

5.1 General Characteristics of the Study Participants

The total number of participants was 94. All were males. General characteristics of participants are presented in Table 1 below.

Descriptive characteristics of WP smokers and non-smokers were compared as shown in Table 1. The exposed group consisted of male WP smokers (n=47), with age (mean±SD) of 48.28±7.46, ranging from 33 to 59 years. The control group consisted of similar number (n=47) of male non-smokers with age (mean±SD) of 46.81±6.45, ranging from 37 to 58 years. Among WP smokers, 10 (21.3%) of the subjects were between 31 to 40 years old, 20 (42.5%) of the subjects were between 41 to 50 years old and 17 (36.2%) of the subjects were between 51 to 60 years old. In the case of control group, 8 (17 %) of the subjects were between 31 to 40 years old, 23 (49%) of the subjects were between 41 to 50 years old and 16 (34%) of the subjects were between 51 to 60 years old (see Figure 3 for detail). The mean±SD height was 165.49±8.26 that ranges from 148-183 cm for WP smokers and 165.80±4.40 that ranges from 157-176 cm for non-smokers. The mean±SD weight was 55.06±8.16 that ranges from 40-69.30 kg and 57.01±6.41 that ranges from 46.50-71 kg for WP smokers and control group, respectively. The mean age was not significantly different between water-pipe smokers and non-smokers. The mean weight and height were also not significantly different between water-pipe smokers and non-smokers.

Majority of WP smokers were uneducated (80.8%); all were married (100%), and about 95.7% were farmers. In the control groups, 63.8% had a primary level of education, 89.4% were married, and 93.6% were farmers. Regarding characteristics of water pipe smoking the mean duration of smoking in years, number of smoking days per week, frequency of smoking per day were 25.43±11.86 years, 6.83±0.84 days and 4.17±1.72 respectively. Most water pipe smokers smoke 3 to 4 times every day. The last time they had smoked was 12 hours before the data collection. All measurements were taken from WP smokers after a minimum of 12 hours smoking abstinence.
Table 1. General Characteristics of the Study Subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>WP smokers (n=47)</th>
<th>Non-smokers (n=47)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48.28±7.46</td>
<td>46.81±6.45</td>
</tr>
<tr>
<td>(Range)</td>
<td>33–59</td>
<td>37–58</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>165.49±8.26</td>
<td>165.80±4.40</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>55.06±8.16</td>
<td>57.01±6.41</td>
</tr>
<tr>
<td>BMI (kg. m⁻²)</td>
<td>19.95±2.08</td>
<td>20.76±2.50</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uneducated</td>
<td>38(80.8%)</td>
<td>15(31.9%)</td>
</tr>
<tr>
<td>Primary education</td>
<td>9(19.2%)</td>
<td>30(63.8%)</td>
</tr>
<tr>
<td>Secondary education</td>
<td>0(0%)</td>
<td>2(4.3%)</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>47(100%)</td>
<td>42(89.4%)</td>
</tr>
<tr>
<td>Unmarried</td>
<td>0(0%)</td>
<td>5(10.6%)</td>
</tr>
<tr>
<td>Occupation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Farmer</td>
<td>45(95.7%)</td>
<td>44(93.6%)</td>
</tr>
<tr>
<td>Labor</td>
<td>2(4.3%)</td>
<td>3(6.4%)</td>
</tr>
<tr>
<td>Monthly income (Birr)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than 600</td>
<td>14(29.8%)</td>
<td>3(6.4%)</td>
</tr>
<tr>
<td>601-1500</td>
<td>15(31.9%)</td>
<td>6(12.8%)</td>
</tr>
<tr>
<td>1501-3000</td>
<td>18(38.3%)</td>
<td>33(70.2%)</td>
</tr>
<tr>
<td>3001-5000</td>
<td>0(0%)</td>
<td>5(10.6%)</td>
</tr>
<tr>
<td>Duration of smoking (Years)</td>
<td>25.43±11.86</td>
<td>-</td>
</tr>
<tr>
<td>Number of smoking days per week (Days)</td>
<td>6.83±0.84</td>
<td>-</td>
</tr>
<tr>
<td>Frequency of smoking per day (Number)</td>
<td>4.17±1.72</td>
<td>-</td>
</tr>
<tr>
<td>Age of initiation (Years)</td>
<td>22.72±8.36</td>
<td>-</td>
</tr>
</tbody>
</table>

- Results are expressed as (mean±SD) and number (percentage).
Table 2, demonstrates the number of WP smokers and non-smokers under different weight category. BMI was calculated for each subject using the formula: BMI = weight/height^2 (kg.m^{-2}). Based on their BMI result, subjects were classified as underweight (BMI<18.5), normal (18.5 to 24.99) and overweight (25 to 29.99). Most participants 38 (80.8%) and 36 (76.6%) were WP smokers and non-smokers, respectively, were categorized under normal weight based on their BMI. Physical activity status was classified based on WHO standardized questioner for physical activity. Individuals who were categorized as physical active they do vigorous-intensity activity that causes large increases in breathing or heart rate like [carrying or lifting heavy loads, digging or construction work] for at least 10 minutes continuously [98]. All study participants were farmers, did vigorous intensity activities as part of their work for at least 5 days per week and spent a minimum of 9 hours per day regularly.
Table 2. BMI Classification and Physical Activity Information

<table>
<thead>
<tr>
<th>Weight status</th>
<th>WP smokers</th>
<th>Non-smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>9(19.2%)</td>
<td>9(19.1%)</td>
</tr>
<tr>
<td>Normal weight</td>
<td>38(80.8%)</td>
<td>36(76.6%)</td>
</tr>
<tr>
<td>Overweight</td>
<td>0(0%)</td>
<td>2(4.3%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physical activity status</th>
<th>Active</th>
<th>Inactive</th>
<th>Non-smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active</td>
<td>47(100%)</td>
<td></td>
<td>47(100%)</td>
</tr>
<tr>
<td>Inactive</td>
<td>0(0%)</td>
<td></td>
<td>0(0%)</td>
</tr>
</tbody>
</table>

- Results are expressed as number (percentage).

Fig 4, shows that among WP smokers, 20(42.6%) had started smoking between 21 to 30 years of age and 10 (21.1%) had begun smoking at the age of between 31 to 40 years.

Fig 6. Age of WP Smoking Initiation among WP Smokers
Table 3, shows that most water pipe smokers, 63.8% had smoked 3 to 4 times per day and 25.5% of them smoked more than five times per day.

Table 3. Frequency of Smoking (per day) among WP Smokers

<table>
<thead>
<tr>
<th>Frequency of smoking (per day)</th>
<th>WP smokers (number)</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>6.4%</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>4.3%</td>
</tr>
<tr>
<td>3 to 4</td>
<td>30</td>
<td>63.8%</td>
</tr>
<tr>
<td>≥ 5</td>
<td>12</td>
<td>25.5%</td>
</tr>
</tbody>
</table>

Table 4 shows the duration of WP smoking in years among WP smokers. Out of 47 WP smokers, 3(6.4%) had smoked WP for less than 5 years, 7(14.9%) were smoked for 6-15 years, 26(55.3%) were smoked for 16-30 years and 11(23.4%) were smoked for 31-45 years. The minimum and maximum duration of smoking were 3 and 45 years, respectively.

Table 4. Duration of WP Smoking (in Years) among WP Smokers

<table>
<thead>
<tr>
<th>Duration of WP Smoking (in Years)</th>
<th>Number of WP Smokers</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;5</td>
<td>3</td>
<td>6.4%</td>
</tr>
<tr>
<td>6-15</td>
<td>7</td>
<td>14.9%</td>
</tr>
<tr>
<td>16-30</td>
<td>26</td>
<td>55.3%</td>
</tr>
<tr>
<td>31-45</td>
<td>11</td>
<td>23.4%</td>
</tr>
</tbody>
</table>
5.2 Respiratory Symptoms

The prevalence of cough, phlegm, shortness of breath and wheezing were 61.7%, 51.1%, 31.9% and 25.5% for WP smokers and 8.5%, 8.5%, 6.4% and 6.4% for non-smokers, respectively as shown in table 5. The result showed higher percentage prevalence of respiratory symptoms among WP smokers than non-smokers and respiratory symptoms were significantly higher among WP smokers when compared with non-smokers.

Respiratory symptom data are summarized in Table 5. WP smokers had higher prevalence of cough (61.7% in WP smokers vs. 8.5% in non-smokers), phlegm (51.1% in WP smokers vs. 8.5% in non-smokers), shortness of breath (31.9% in WP smokers vs. 6.4% in non-smokers), and wheezing (25.5% in WP smokers vs. 6.4% in non-smokers). The odds of cough were 17.32 times higher in WP smokers than in non-smokers (OR=17.32, 95% CI: 5.32, 56.44) and the statistical association was found to be significant. WP smokers had phlegm 11.22 times (OR=11.22, 95% CI: 3.47, 36.27) higher than it was in non-smokers and it was statistically significant. The odds of shortness of breath was 6.88 times (OR=6.88, 95% CI=1.84, 25.75) higher for WP smokers than non-smokers and the association was statistically significant. The odds of wheezing was 5.03 times (OR=5.03, 95% CI=1.32, 19.23) higher for WP smokers than non-smokers and the association was found statistically significant.

Table 5. Prevalence of Respiratory Symptoms among WP Smokers and Non-Smokers

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>WP smokers</th>
<th>Non-smokers</th>
<th>OR</th>
<th>CI (95%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cough</td>
<td>29(61.7%)</td>
<td>4(8.5%)</td>
<td>17.32</td>
<td>5.32-56.44</td>
<td>0.000*</td>
</tr>
<tr>
<td>Phlegm</td>
<td>24(51.1%)</td>
<td>4(8.5%)</td>
<td>11.22</td>
<td>3.47-36.27</td>
<td>0.000*</td>
</tr>
<tr>
<td>Shortness of breath</td>
<td>15(31.9%)</td>
<td>3(6.4%)</td>
<td>6.88</td>
<td>1.84-25.75</td>
<td>0.001*</td>
</tr>
<tr>
<td>Wheezing</td>
<td>12(25.5%)</td>
<td>3(6.4%)</td>
<td>5.03</td>
<td>1.32-19.23</td>
<td>0.011*</td>
</tr>
</tbody>
</table>

- Values in the first and second column are the numbers and percentages of subjects in each group experiencing the corresponding symptoms.
- *p<0.05
5.3 History of Respiratory Diseases in WP Smokers and Non-Smokers

There is an increased prevalence of respiratory diseases among WP smokers in the past; bronchitis (48.94% in WP smokers vs. 6.4% in non-smokers; OR= 14.06), pneumonia (19.15% in WP smokers vs. 6.4% in non-smokers; OR=1.62), bronchial asthma (12.76% in WP smokers vs. 4.3% in non-smokers; OR =3.29) and heart trouble (10.63% in WP smokers vs. 2.1% in non-smokers; OR=5.48). Only bronchitis was statistically significant (p = 0.000*)

Table 6. Prevalence of Respiratory Diseases among WP Smokers and Non-Smokers

<table>
<thead>
<tr>
<th>Disease</th>
<th>WP smokers</th>
<th>Non-smokers</th>
<th>OR</th>
<th>CI (95%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bronchitis</td>
<td>23(48.94%)</td>
<td>3(6.4%)</td>
<td>14.06</td>
<td>3.82-51.67</td>
<td>0.000*</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>9(19.15%)</td>
<td>3(6.4%)</td>
<td>1.62</td>
<td>0.53-4.98</td>
<td>0.398</td>
</tr>
<tr>
<td>Bronchial Asthma</td>
<td>6(12.76%)</td>
<td>2(4.3%)</td>
<td>3.29</td>
<td>0.63-17.24</td>
<td>0.139</td>
</tr>
<tr>
<td>Heart trouble</td>
<td>5(10.63%)</td>
<td>1(2.1%)</td>
<td>5.48</td>
<td>0.61-48.80</td>
<td>0.091</td>
</tr>
</tbody>
</table>

- Values in the first and second column are the numbers and percentages of subjects in each group experiencing the corresponding diseases.

Bronchitis was recorded based on the standardized questionnaire that was used to diagnosis bronchitis. Others history of respiratory diseases were recorded from subjects who had been told that they have specific disease by health professionals.
5.4 Changes in Lung Function Tests

An independent sample t-test was used to compare lung function indices (FVC, FEV₁, FEV₁%, PEF and FEF₂₅% -75%) of WP smokers and non-smokers. FVC, FEV₁, FEV₁%, PEF and FEF₂₅% -75% were higher in non-smokers than WP smokers and the differences were statistically significant (p< 0.05) as shown in table 7 below:

Table 7. Comparison of Pulmonary Function Test Data between Water Pipe Smokers and Non-Smokers

<table>
<thead>
<tr>
<th>PFT parameters</th>
<th>WP smokers</th>
<th>Non-smokers</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC(L)</td>
<td>4.76±0.99</td>
<td>6.11±0.75</td>
<td>-7.51</td>
<td>0.000*</td>
</tr>
<tr>
<td>FEV₁(L)</td>
<td>3.53±0.83</td>
<td>4.93±0.65</td>
<td>-9.15</td>
<td>0.000*</td>
</tr>
<tr>
<td>FEV₁%</td>
<td>75.30±10.54</td>
<td>80.66±5.19</td>
<td>-3.13</td>
<td>0.002*</td>
</tr>
<tr>
<td>PEF(L/S)</td>
<td>6.06±1.90</td>
<td>8.32±1.84</td>
<td>-5.84</td>
<td>0.000*</td>
</tr>
<tr>
<td>FEF₂₅% -75%(L/S)</td>
<td>3.05±1.13</td>
<td>5.00±1.15</td>
<td>-8.34</td>
<td>0.000*</td>
</tr>
</tbody>
</table>

PFT Parameters (Percent Predicted) of Study Participants for Each Group

The percentages of predicted values of lung function indices (FVC, FEV₁, PEF and FEF₂₅% -75%) were significantly higher in non-smokers than WP smokers (p<0.05) as shown in table 8 below:

Table 8. PFT Parameters (Percent Predicted) of Study Participants for Each Group

<table>
<thead>
<tr>
<th>PFT parameter</th>
<th>WP smokers</th>
<th>Non-smokers</th>
<th>t-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (% predicted value)</td>
<td>108.49±20.31</td>
<td>130.87±12.93</td>
<td>-6.37</td>
<td>0.000*</td>
</tr>
<tr>
<td>FEV₁ (% predicted value)</td>
<td>102.34±22.04</td>
<td>127.85±13.69</td>
<td>-6.74</td>
<td>0.000*</td>
</tr>
<tr>
<td>FEV₁% (% predicted value)</td>
<td>95.94±13.35</td>
<td>98.66±6.27</td>
<td>-1.26</td>
<td>0.209</td>
</tr>
<tr>
<td>PEF (% predicted value)</td>
<td>68.04±20.23</td>
<td>90.75±20.05</td>
<td>-5.46</td>
<td>0.000*</td>
</tr>
<tr>
<td>FEF₂₅% -75% (% predicted value)</td>
<td>96.38±33.63</td>
<td>124.87±25.83</td>
<td>-4.61</td>
<td>0.000*</td>
</tr>
</tbody>
</table>
Pearson correlation was used to assess the relationship between age and lung function measurements among WP smokers. There was a negative correlation between age and all lung function indices (FVC ($r$=-0.387; $p=0.007$), FEV$_1$ ($r$=-0.460; $p=0.001$), FEV$_1$% ($r$=-0.156; $p=0.294$), PEF ($r$=-0.436; $p=0.002$) and FEF$_{25\%\text{-}75\%}$ ($r$=-0.347; $p=0.017$). Except FEV$_1$% ($p=0.294$), all other values were statistically significant ($p<0.05$). Pearson correlation was also used to assess the relationship between duration of smoking (in years) and lung function measurements. There was a negative correlation between duration of smoking and all lung functions. FVC, FEV$_1$ and FEF$_{25\%\text{-}75\%}$ were statistically significant ($p<0.05$) as shown in table 9 below:

Table 9. Correlation between Duration of Smoking (in Years) and Lung Function Test Indices

<table>
<thead>
<tr>
<th>PFT</th>
<th>FVC</th>
<th>FEV$_1$</th>
<th>FEV$_1$%</th>
<th>PEF</th>
<th>FEF$_{25%\text{-}75%}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson correlation coefficient</td>
<td>-0.461</td>
<td>-0.486</td>
<td>-0.062</td>
<td>-0.231</td>
<td>-0.322</td>
</tr>
<tr>
<td>Sig.</td>
<td>0.001*</td>
<td>0.001*</td>
<td>0.680</td>
<td>0.119</td>
<td>0.027*</td>
</tr>
</tbody>
</table>

- * Correlation is significant at 0.05 level
5.5 Changes in Cardiovascular Indices

SBP, DBP, SpO₂ and PR were measured to evaluate cardiovascular functions. Additionally, MAP and PP were calculated from SBP and DBP results. The mean SBP were 111.70±10.49 in WP smokers and 104.04±9.48 for non-smokers, DBP were 70.43±9.77 for WP smokers and 68.62±7.98 for non-smokers, MAP were 84.18±9.07 in WP smokers and 80.42±7.17 in non-smokers, PP were 41.27±8.99 in WP smokers and 35.21±9.83 in non-smokers, SpO₂ were 95.47±2.18 in WP smokers and 95.71±1.61 in non-smokers and PR were 74.53±10.87 in WP smokers and 71.17±13.20 in non-smokers. SBP and PP were significantly different between the two groups but DBP, MAP, SpO₂ and PR were not significantly different between the two groups.

Table 10. Cardiovascular Function Indices Result for Each Group

<table>
<thead>
<tr>
<th>Group</th>
<th>SBP (mm Hg)</th>
<th>DBP (mm Hg)</th>
<th>MAP (mm Hg)</th>
<th>PP (mm Hg)</th>
<th>SpO₂ (%)</th>
<th>PR (beats/min.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>WP smokers</td>
<td>111.70±10.49</td>
<td>70.43±9.77</td>
<td>84.18±9.07</td>
<td>41.27±8.99</td>
<td>95.47±2.18</td>
<td>74.53±10.87</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>104.04±9.48</td>
<td>68.62±7.98</td>
<td>80.42±7.17</td>
<td>35.21±9.83</td>
<td>95.71±1.61</td>
<td>71.17±13.20</td>
</tr>
</tbody>
</table>

| t-value | 3.71 | 0.98 | 2.22 | 3.12 | 0.592 | 1.348 |
| p-value | 0.000* | 0.328 | 0.28 | 0.002* | 0.555 | 0.181 |

Pearson correlation was used to assess the relationship between duration of smoking (in years) and cardiovascular indices. There was a positive correlation between duration of smoking and SBP, DBP, MAP and PP, but there was negative correlation between duration of smoking with SpO₂ and PR. Only SBP was statistically significant (p<0.05) as shown in table 11 below:
Table 11. Correlation between Duration of Smoking (in Years) and Cardiovascular Function Indices

<table>
<thead>
<tr>
<th>Cardiovascular Indices</th>
<th>SBP</th>
<th>DBP</th>
<th>MAP</th>
<th>PP</th>
<th>SpO₂</th>
<th>PR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson correlation coefficient</td>
<td>0.328</td>
<td>0.156</td>
<td>0.238</td>
<td>0.213</td>
<td>-0.110</td>
<td>-0.61</td>
</tr>
<tr>
<td>Sig.</td>
<td>0.025*</td>
<td>0.295</td>
<td>0.107</td>
<td>0.151</td>
<td>0.463</td>
<td>0.683</td>
</tr>
</tbody>
</table>

- * Correlation is significant at 0.05 level
6. DISCUSSION

The results of current study showed all respiratory symptoms were higher among WP smokers. Cough and phlegm were the most prevalent respiratory symptoms among WP smokers. This result is similar to a study conducted in New York City that compared self-reported never smokers (n=19) with water pipe-only smokers (n=21). Both groups were comparable in terms of sex, ethnicity and body mass index. Cough and sputum were significantly higher in water pipe smokers than in non-smokers (p=0.008). Thirty-three percent of water pipe smokers had cough compared with 5% of non-smokers (p=0.03), and 19% of water pipe smokers had sputum production compared with 0% of non-smokers (p=0.04) [99]. A study conducted in China on Chinese water pipe smoking showed that the risk of COPD and pulmonary symptoms (i.e., cough and sputum) were higher in the Chinese water pipe smokers than in the non-smoker group and it was significantly higher in water pipe smokers than in non-smokers (p<0.01) [30]. Another study conducted in Jordan showed that with regards to respiratory symptoms, a significantly greater proportion of WPS (72.5%) than non-smokers (21.7%) reported any respiratory symptoms (e.g. bringing up phlegm, having shortness of breath upon exertion, cough, chest illness in the past three years that kept a participant off work, and coughing with phlegm that lasted at least three weeks) [100]. Similarly, a study conducted in Mashhad (Iran) showed that the prevalence of cough and wheezing was significantly higher among WP smokers when compared with non-smokers (p=0.017, p=0.026, respectively) [101].

Evaluation of multiple lung components demonstrated a significant number of lung clinical and biological abnormalities in exclusive water pipe smokers. The water pipe smokers had increased cough and sputum, as well as biological abnormalities in several anatomic components in the lung, including in the lower respiratory tract, with increased numbers of secretory and intermediate cells and decreased numbers of ciliated and basal cells [102]. Additionally, the possible mechanisms for increased respiratory symptoms were decreased mucociliary clearance function of respiratory tract due to chronic smoke exposure; inhalation of irritants and pollutant particles were also responsible for increased respiratory symptoms [42].
Prevalence of history of respiratory diseases was also higher among WP smokers than non-smokers; bronchitis (48.94% in WP smokers vs. 6.4% in controls; OR=14.06, 95% CI=3.82, 51.67), pneumonia (19.15% in WP smokers vs. 6.4% in controls; OR=1.62, 95% CI=0.53, 4.98), bronchial asthma (12.76% in WP smokers vs. 4.3% in controls; OR=3.29, 95% CI=0.63, 17.24) and heart trouble (10.63% in WP smokers vs. 2.1% in controls; OR=5.48, 95% CI=0.61, 48.80) (Table 6). Related study showed that chronic bronchitis were more pronounced in WP smoker group as 11.75% of subjects in the WP smoker group had positive response to questions on the respiratory symptoms of chronic bronchitis compared with none in the control group [41]. Similarly, compared to never smokers, WP smokers were associated with a higher risk of chronic bronchitis. Water pipe smokers had a 6 times increased risk of chronic bronchitis (OR=6.40, 95% CI: 2.55–16.11, p<0.001) [27].

Lung function indices (FVC, FEV1, FEV1%, PEF and FEF25% -75%) were significantly lower in WP smokers than non-smokers (p<0.05) (Table 7). The percentages of predicted values of lung function indices (FVC, FEV1, FEF25% -75% and PEF) were also significantly lower in WP smoker than in non-smokers (p<0.05) (Table 8). The result is consistent with a study conducted in Mashhad that identified and studied three groups, including 57 WP smokers, 81 cigarette smokers and 44 non-smokers as a control group. PFT (FVC, FEV1 and PEF) values were significantly lower in WP smokers than in non-smokers [101]. Similarly another study conducted in Kuwait that compared 75 subjects in cigarette smoking group, 77 in WP smokers and 16 subjects in control group. The spirometry finding showed the FEV1 as well as FEV1% among WP smokers was less than controls [41]. A study conducted in Tunisia found FEV1% and PEF were reduced when measured values were compared with the reference values (normal range) [103]. A study conducted in Izmir (Turkey) included four groups; water pipe smokers, water pipe smokers who had quit cigarette smoking, cigarette smokers and never smokers. When compared with non-smokers, statistically significant decreases in PEF of water-pipe smokers were observed which is in agreement with the present study [24]. In a study conducted in China, with regard to lung function, the Chinese water pipe smoking group had the lowest FEV1% than among all other groups [30]. The current study is also in agreement with a systematic review and meta-analysis studies on the effects of water pipe smoking on lung function that compared non-smoker group with WP smoking group. Compared with non-smokers, WP smokers were associated with a statistically significant reduction in FEV1 [29]. Similarly, study on pulmonary function test
results revealed that there were significant fall in \( \text{FEF}_{25\%-75\%} \) and \( \text{PEF} \) \( (p=0.045 \) and \( p=0.0004 \), respectively) [32].

PFT results also revealed WPS had significantly lower percent predicted value of \( \text{FEV}_1 \), \( \text{FVC} \) and \( \text{PEF} \); which is in agreement with the current study [100]. In a study conducted in Tunisia, the subjects were divided into three groups; cigarette smokers, WP smokers and non-smokers. The study found that WP smokers had significantly lower FVC than non-smokers. FVC values of WP smokers were lower than that of the non-smoker group. Regarding the PEF, WP smokers were significantly lower than non-smokers \( (p<0.001) \). Non-smokers subjects justified also \( \text{FEV}_1 \) values significantly higher than WP smokers. Additionally, WP smokers have \( \text{FEF}_{25\%-75\%} \) value, was significantly lower compared to the non-smokers group [94].

The measurement of Fractional Exhaled Nitric Oxide (FeNO) is emerging, non-invasive and widely used method for assessing lower respiratory tract for biomarkers of airway inflammation and oxidative stress. There was decreased fractional exhaled nitric oxide that is characteristics of air way inflammation and oxidative stress in WP smokers [86].Another mechanism for the effect of water pipe smoking on respiratory outcomes was found to be through the damage that it causes to the lung parenchyma and the associated inflammation of the airways [104].

Matter from the water pipe contained high levels of particulate matter, PAHs and heavy metals, which may be deposited effectively in the lungs rather than staying in the airways after inhalation of large volume smoking. Indeed, these Long-term exposure would inevitably cause damage in the alveolar epithelial cells and result in decrement in lung functions [30]. WPS resulted in increased airway resistance, lung inflammation, oxidative stress and catalase activity in animal lungs that provides a mechanistic explanation for the detrimental chronic respiratory effects of WPS [105].
SBP and PP were significantly different between the two groups but DBP, MAP, SpO₂ and PR were not significantly different between the two groups (Table 10). The number of studies on the chronic cardiovascular impact of WP smoke exposure is scarce. With respect to acute effects; it has been shown that WP smoking induced an increase in heart rate, systolic blood pressure and diastolic blood pressure [32, 33, 36, 38].

A study which was conducted in Northern Israel on the acute effects of water pipe smoking on the cardiorespiratory system included forty five volunteers. After one session of WPS, systolic and diastolic blood pressure levels were significantly higher after smoking (systolic, 119.52±12.07 mm Hg vs. 131.98±17.8 mm Hg; diastolic, 74.84±7.89 mm Hg vs. 82.98±12.52 mm Hg, respectively; \( p < 0.001 \)) and heart rates increased from 80.39±9.92 beats/min to 95.59±17.41 beats/min, \( p < 0.001 \) [32]. Another study conducted in Saudi also showed that acute short-term WP smoking elicits a modest increase in heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial blood pressure [106]. Similarly, systolic blood pressure and diastolic blood pressure were higher among water pipe smokers than non-smokers [100]. Furthermore, a study on the acute effects of WPS revealed that all cardiovascular indices (HR, DBP and SBP) were higher among WP smokers than those of non-smokers [95].

A study that was conducted in Lebanon on the acute and chronic effects of WP smoking comprised of three groups: non-smokers, WP smokers and cigarette smokers. The measurement was done before the beginning of WP smoking (for chronic effect) and 45 minute after start of WP smoking (for acute effect). The chronic effect result showed that SBP (118±16.9 in non-smokers vs. 125.9±17.3 in WP smokers; \( p = 0.05 \)), DBP (73±12.2 in non-smokers vs. 78.1±11.5 in WP smokers; \( p = 0.05 \)) and HR (76.19±8.87 in non-smokers vs. 82.3±12.42 in WP smokers; \( p = 0.01 \)) were observed. On the other hand the results revealed that all cardiovascular indices (HR, DBP and SBP) were higher among WP smokers than non-smokers [95].

Chronic effects was also demonstrated in a study conducted in Tunisia where SBP and HR were significantly higher in WP smokers than those of non-smokers group (\( p < 0.001 \)), but there were no significant difference between WP smokers and non-smokers group in the DBP values (\( p > 0.05 \)) [94].
The effect of WP smoke exposure on SBP in mice at 1, 2, 4, and 6 month, were compared with their respective air exposed groups, the SBP in animals exposed to WP smoke was significantly higher at 2 month ($p<0.005$), 4 month ($p<0.0005$), and 6 month ($p<0.0001$) [96]. It was reported that a significant increase in SBP (+13 mmHg) at the end of 1 month exposure to WP smoke compared with air-exposed group, significant increase in SBP at 2 month (+18 mmHg), 4 month (+49 mmHg), and 6 month (+43 mmHg) of exposure to WP smoke compared with their respective control groups [96].

The central hypotheses in the pathophysiology of WPS related cardiovascular disease include instigation of proinflammatory responses, alterations in systemic ANS activity, and oxidative stress that leads to compromised cardiac ANS regulation [107-108]. It was reported that WPS showed impaired vasodilation of the brachial artery that was suggestive of endothelial dysfunction when compared with that of cigarette smokers and non-smokers [35]. This impaired vasodilation could potentially lead to vascular remodeling and dysfunction [109]

WPS has been shown to acutely and unfavorably regulate vascular resistance, blood flow, venous outflow and BP parameters, leading to altered peripheral and central vasomotor function [35, 110-112]. WPS have been shown to result in significant increase in the biomarkers for oxidative stress and inflammatory marker high-sensitivity C reactive protein [110]. Considering the negative biological effects of oxidative stress - such as increased endothelin release [113], vasoconstriction [114] and the fact that in smokers increased oxidative stress biomarkers are found in peripheral [115] and coronary vascular tissue [116], water pipe consumption is believed that it increases the likelihood of cardiovascular diseases.
7. CONCLUSION

The results showed higher prevalence of respiratory symptoms, history of respiratory diseases and reduction of all pulmonary function tests in WP smokers compared to the non-smokers. The increased respiratory symptoms and reduction of PFT values in WP smokers showed the negative impact of traditional WP smoking on the respiratory system.

Except SpO\textsubscript{2} all cardiovascular parameters including SBP, DBP, MAP, PP and PR were higher among WP smokers than non-smokers. SBP and PP were significantly higher among WP smokers than non-smokers. To minimize the effects of confounding factors like job, age, BMI and ethnicity on cardiovascular indices, WP smokers and non-smokers were recruited from the same area with similar job, age range, BMI and ethnicity. Therefore, it seems that these factors have minor effect on cardiovascular indices.
8. STRENGTH AND LIMITATIONS OF THE STUDY

This research investigated the effects of water pipe smoking on pulmonary functions and cardiovascular indices for the first time in the country and can be used as a base for further researches in this particular area.

This study was based on a small group of subjects and studied only the chronic effects of WP smoking. Studies of the acute and long-term effects, as well as studies based on larger populations that include smokers from different parts of the country are needed. Such studies would further confirm the risks of water pipe smoking. Additionally this study lacks cigarette smoking group due to lack of exclusive cigarette smokers in the study area. Future studies that take in to consideration these confounding variables are recommended.
9. RECOMMENDATIONS

- Evidences of pulmonary function tests and cardiovascular indices suggest that WP smoking is associated with acute and chronic negative health outcomes. Health care providers should assess patients for WP smoking in regions it is commonly encountered; should advise that abstaining from WP smoking will limit their risks of negative health outcomes.
- Dita Woreda Health Office should give regular training to the community on the health impacts of WP smoking in order to create awareness among the public.
- Concerned governmental and non-governmental bodies in the region should bring the issue to their attention and play a leading role in the prevention of WP smoking.
10. REFERENCES


64. Daniels KE, Roman NV. A descriptive study of the perceptions and behaviors of water pipe use by university students in the Western Cape, South Africa. TobInduc Dis 2013;11(1):4


66. WHO advisory note, Water pipe tobacco smoking: health effects, research needs and recommended actions for regulators, 2nd edition, 2015


110. Diab OA, Abdelrahim EM, Esmail M. Effect of water pipe tobacco smoking on plasma high sensitivity C reactive protein level and endothelial function compared to cigarette smoking. Egypt Heart J. 2015; 67:233–241


115. Gniwotta C., Morrow J.D., Robert II L.J. Prostaglandin F2-like compounds, F2-isoprostanes, are present in increased amounts of human atherosclerotic lesions. Arteriosclerosis, Thrombosis and Vascular Biology. 1997; 17, 3236–3241

11. APPENDIX

Appendix 1. Questionnaire (English Version)

Part I: Sociodemographic Characteristics

1. How old are you? ____________

2. Have you ever attended school? Yes □ No □

3. If your answer is yes for Q2, what is the highest level of education you attended?
   - Primary
   - Secondary
   - College diploma
   - Bachelor degree
   - Others/specify…….

4. What is your main work?
   - Farmer
   - Government employee
   - Student
   - Private employee
   - Not working
   - Others/specify…….

5. What is your current marital status?
   - Unmarried
   - Married
   - Divorced
   - Widowed
6. Monthly income (Birr)
   - Less than 600
   - 601 up to 1500
   - 1501 up to 3000
   - 3001 up to 5000
   - Above 5000

**Part II: Respiratory Symptoms**

**Cough**

1. Do you usually cough first thing in the morning in the winter?  
   - Yes  
   - No

2. Do you usually cough during the day or at night in the winter?  
   - Yes  
   - No

3. If Yes to Q1 or Q2, do you cough like this on most days for as much as three months each year?  
   - Yes  
   - No

**Phlegm**

4. Do you usually bring up any phlegm from your chest first thing in the morning in the winter?  
   - Yes  
   - No

5. Do you usually bring up any phlegm from your chest during the day or at night in the winter?  
   - Yes  
   - No

6. If Yes to 4 or 5, Do you bring up phlegm like this on most days for as much as three months each year?  
   - Yes  
   - No

**Period of cough and phlegm**

7. In the past three years have you had a period of increased cough and phlegm lasting for three weeks or more?  
   - Yes  
   - No

8. If Yes for Q7, have you had more than one such period?  
   - Yes  
   - No
9. Are you troubled by any shortness of breath when hurrying on level ground or walking up a slight hill?  Yes ☐ No ☐

10. If Yes for Q9, do you get shortness of breath while walking with other people of your own age on level ground? Yes ☐ No ☐

11. If Yes for Q10, do you have to stop for breath when walking at your own pace on level ground? Yes ☐ No ☐

Wheezing

12. Have you had attacks of wheezing in your chest at any time in the last 12 months?  Yes ☐ No ☐

13. Have you ever had attacks of shortness of breath with wheezing? Yes ☐ No ☐

14. Have you at any time in the last 12 months been woken at night by an attack of shortness of breath? Yes ☐ No ☐

Chest illness

15. During the past three years have you had any chest illness which has kept you from your usual activities for as much as a week? Yes ☐ No ☐

16. If Yes for Q 15, did you bring up more phlegm than usual in any of these illnesses? Yes ☐ No ☐
Past illnesses

17. Have you ever had, or been told that you had:

- Heart trouble
- Bronchitis
- Pneumonia
- Pulmonary tuberculosis
- Bronchial asthma
- Other/specify…….

18. How old were you when you started water pipe smoking? ______________

19. For how long you have been smoked water pipe? ______________

20. How often do you smoke water pipe in a week?

- Every day
- Two days in a week
- Three days in a week
- Four days in a week
- Five days in a week
- Six days in a week
- Others/specify…….

22. If you are smoking every day, how many times do you smoke per day?

- One time
- Two times
- Three times
- Four times
- Others/specify…..
Part III: Physical Activity

1. Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like [carrying or lifting heavy loads, digging or construction work] for at least 10 minutes continuously? Yes ☐ No ☐

2. In a typical week, on how many days do you do vigorous intensity activities as part of your work? Number of days_____________

3. How much time do you spend doing vigorous-intensity activities at work on a typical day? Hours____________

4. Does your work involve moderate-intensity activity that causes small increases in breathing or heart rate such as brisk walking [or carrying light loads] for at least 10 minutes continuously? Yes ☐ No ☐

5. In a typical week, on how many days do you do moderate intensity activities as part of your work? Number of days_____________

6. How much time do you spend doing moderate intensity activities at work on a typical day? Hours____________
Part IV: Respiratory and Cardiovascular indices to be measured

- Weight_________________
- Height_________________

- Respiratory functions to be measured
  - FVC
  - FEV₁
  - FEV₁%
  - PEF
  - FEF25% - 75%

- Cardiovascular indices to be measured
  - SBP
  - DBP
    - SpO₂ (Oxygen Saturation)
  - PR (Pulse Rate)
  - PP (Pulse Pressure)
  - MAP (Mean Arterial Pressure)
Appendix 2. Questionnaire (Amharic Version)

መጠይቅ

ከስል ከወ: ይሉ እንወ

1. እድሜ? _____________

2. መደበኛ እ/ት እንወ? እም □ □ እሪሚም □ □

3. አንቀፋ ከፋር 2 መልም እም እንወ፣ ከ ከም ይወ ይት እንወ?
   o ከም እርን ይወ
   o ከም እርን ይወ
   o ከም እርን ይወ
   o ከም እርን ይወ
   o ከም እርን ይወ
   o ከም እርን ይወ

4. መደበኛ ከራ?
   o ያካ
   o ያካ ብ ያካ ያካ ብ ያካ ብ ያካ
   o ብካ
   o ብካ ብ ያካ
   o ብካ ብ ያካ
   o ብካ ብ ያካ
   o ብካ ብ ያካ

5. ረካድ እንወ?
   o ያካ
   o ያካ
   o ያካ
   o ያካ ብ ያካ
   o ያካ ብ ያካ

6. መድፍ ውስ, መስን (አ-ር)
   o እንወ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያለ ያlation

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የሳማ በሽታ የምልክቶች

1. በክሱት ቍቃው ንትት እና ወቅት መቅት ይሆኔ? እወ እደለም

2. በክሱት ቍቃው ንትት እና ወቅት መቅት ይሆኔ? እወ እደለም

3. አንቀፍ አጥር 1 ወይም 2 መልስ እወ ከምስክር ይህ ሌሎቹ ሲሆን መር ይሆኔ? እወ እደለም

እስከ

4. በክሱት ቍቃው ንትት እና ወቅት መቅት እስከ ከርስጥ ይሆኔ? እወ እደለም

5. በክሱት ቍቃው ንትት እና ወቅት መቅት እስከ ከርስጥ ይሆኔ? እወ እደለም

6. አንቀፍ አጥር 4 ወይም 5 መልስ እወ ከምስክር ይህ ሌሎቹ ሲሆን መር ይሆኔ? እወ እደለም

ማል እና እስከ ውስጥ

7. በክሱት ቍቃው ንትት እና ወቅት መቅት እስከ ከርስጥ ይሆኔ? እወ እደለም

8. አንቀፍ አጥር 7 መልስ እወ ከምስክር ይህ ሌሎቹ ሲሆን መር ይሆኔ? እወ እደለም

9. በክሱት ቍቃው ንትት እና ወቅት መቅት ይሆኔ? እወ እደለም
10. አንቀጽ ዓትር 9 ወወን እም ከሆነ፣ ከሆነ ከር መማሪ እውነት ዓትር የተወርከ በምርሱ?

አወ እወላ ባንክ

11. አንቀጽ ዓትር 9 ወወን እም ከሆነ፣ ከሆነ ከር መማሪ እውነት ዓትር የተወርከ በምርሱ?

አወ እወላ ባንክ

ስርስርታ

12. ኢትዮጵያ በ8 መድስት ወሃ መሆን ያቀር ያሃ እንወ ከመጋ በምርሱ ያስጡ ያሌዩ ያወርህ?

አወ እወላ ባንክ

13. ይግባኝ ያሃ እንወ እንወ ከመጋ ባወቅ ያስጡ ያሌዩ ያወርህ?

አወ እወላ ባንክ

14. ኢትዮጵያ በ8 መድስት ወሃ መሆን ያቀር ያሃ እንወ ከመጋ በምርሱ ያስጡ ያሌዩ ያወርህ?

አወ እወላ ባንክ

የደረት ከማም

15. ኢትዮጵያ በ8 መድስት ወሃ መሆን ያቀር ያሃ እንወ ከመጋ ባወቅ ያስጡ ያሌዩ ያወርህ ይህ ከማም?

አወ እወላ ባንክ

16. አንቀጽ ዓትር 15 ወወን እም ከሆነ፣ ከሆነ ከር መማሪ ያስጡ ያሌዩ ያወርህ ይህ ከማም ያሃ ያሌዩ ያውርስ ያሃ ያሌዩ ያወርህ ይህ ከማም?
17. የትኛዉ በሽታ / ከአሇብሁ ተብሇህ ተቃሇህ ይ哉?  
   o ውስ ትርር  
   o ዩርር ፩ች  
   o ዩሇም ፩ች  
   o ገል  
   o ይም አስም  
   o እል/ ያን ይች

18. ይች ይወላል ያወራደው ከአሇብ እውﻠቱ ይሆ?  

19. ወሳኔ ለሆ ቤ የሚችል?  

20. ወሳምንት ሉገ ት የሚችል?  
   o እም  
   o ለአምኝ እ-ም  
   o ለአምኝ እ ለም  
   o ለአምኝ እራት ለም  
   o ለአምኝ እም ለም  
   o ለአምኝ እም ለም  
   o እል/ ያን ይች

21. ወሳምንት ይሮፋል ከአምኝ ከአ ት የሚችል?  
   o እና እና  
   o እ-ክ እና  
   o እና እና  
   o እራት እና  
   o እ-ክ እና  
   o እል/ ያን ይች
ክፍል ሶስት: የሰዉነት እንቅስቃሴ

1. በወንወን ይል እንቁነት፣ ለስከ መልሮች የምጠይቅ ይመለስ ቤት፣ የምጨምር፣ የምሳሌ፣ ከባድ ይወስከም፣ የምጠይቅ ወይም የምሳሌ ይስር ይችሉ ከው?

2. በወንወን ይል እንቁነት ያስ መልሮች ይህ ላይ ይህ ይህ ይህ ይህ ይህ ይህ ይህ ይህ ከው?

3. በወንወን ይል እንቁነት ያስ መልሮች ይህ ያስ ላይ ይህ ይህ ይህ ይህ ይህ ይህ ይህ ይህ ከው?

4. በወንወን ይል እንቁነት ያስ መልሮች ይህ ያስ ላይ ይህ ይህ ይህ ይህ ይህ ይህ ይህ ይህ ከው?

5. በወንወን ይል እንቁነት ያስ መልሮች ይህ ያስ ላይ ይህ ይህ ይህ ይህ ይህ ይህ ይህ ይህ ከው?

6. በወንወን ይል እንቁነት ያስ መልሮች ይህ ያስ ላይ ይህ ይህ ይህ ይህ ይህ ይህ ይህ ይህ ከው?
Part IV: Respiratory and Cardiovascular indices to be measured

- Weight________________
- Height________________

➤ Respiratory functions to be measured

<table>
<thead>
<tr>
<th></th>
<th>Actual</th>
<th>Predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC</td>
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<td></td>
</tr>
<tr>
<td>FEV₁</td>
<td>_______</td>
<td></td>
</tr>
<tr>
<td>FEV₁%</td>
<td>_______</td>
<td></td>
</tr>
<tr>
<td>PEF</td>
<td>_______</td>
<td></td>
</tr>
<tr>
<td>FEF₂₅%-₇₅%</td>
<td>_______</td>
<td></td>
</tr>
</tbody>
</table>

➤ Cardiovascular indices to be measured

- SBP________________________
- DBP________________________
- \(\text{SpO}_₂\) (Oxygen saturation)_______________
- PR (Pulse Rate)________________
- PP (Pulse Pressure)_____________
- MAP (Mean Arterial Pressure)_______