UNIT 2 PHYSIOLOGICAL ANTHROPOLOGY

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Learning Objective

After going through the unit, you will be able to know about:

- the relation of human physiological traits to their health status;
- the cardiorespiratory fitness to physiological traits; and
- the consequence of environmental factors on human physiology.

2.1 INTRODUCTION

In the past decade, the growing epidemic of health risks has led to health promotion strategies focus solely on behavioural change and toward ecological approaches that engage communities in fostering environment and behavioural changes. This general trend coincides with the increased popularity of interdisciplinary and participatory approaches to health promotion, creating new opportunities for anthropologists to play a role in community health promotion.

The health promotive capacity of an environment is understood, not simply in terms of the health effects of separate environmental features (e.g., air quality, seismic safety, or social climate), but more broadly as the cumulative impact of multiple environmental conditions or occupants' physical, emotional, and social well-being, over a specific time interval (Stokols, 1996). Anthropologists bring an important understanding of the interaction of physical and social environmental conditions and can be strong participants in such an "inherently interdisciplinary" approach.

In this unit we will review the interaction of human physiological fitness and environment with respect to their health & morbidity.

2.2 HUMAN CARDIOVASCULAR PHYSIOLOGY AND HEALTH STATUS

The cardiovascular system consists of an interconnected continuous vascular circuit containing a pump (heart), a high pressure distribution system (arteries), exchange vessels (capillaries) and a low pressure collection and return system (veins). Functionally, the heart consists of two separate pumps: the left heart receives blood from the body and pumps it to the lungs for aeration (pulmonary circulation) and the right heart accepts oxygenated blood from the lungs and pumps it throughout the body (systemic circulation). In other words, the heart provides the force to propel blood throughout the vascular circuit.

2.2.1 Blood Pressure

With each contraction of the left ventricle a surge of blood enters the aorta, distending the vessel and creating pressure within it. The force exerted by blood against the arterial walls during the cardiac cycle is known as blood pressure and reflect combined effect of arterial blood flow per minute (cardiac output) and resistance to that flow in the peripheral vasculature expressed as blood pressure = cardiac output × total peripheral resistance.

The highest pressure generated during left ventricular contraction termed systole, reflects the systolic blood pressure. While during ventricular relaxation termed diastole, the aortic valve close and the natural elastic arterial recoil maintains a continuous pressure providing a steady blood flow into the periphery. The arterial pressure continually declines as blood flows. The lowest pressure reached during ventricular relaxation represents diastolic blood pressure. In a normal individual, systolic blood pressure varies between 110 and 130 mmHg and diastolic blood pressure between 60 and 80 mmHg. However, arteries "hardened" by mineral and fatty deposits within their walls or arteries with excessive peripheral resistance to blood flow from kidney malfunction induce systolic pressures as high as 300 mmHg and diastolic pressures above 120 mmHg. Although there is a continuum of cardiovascular risk across levels of blood pressure, the classification of adults according to blood pressure provides a framework for differentiating levels of risk associated with various blood-pressure categories and for defining treatment thresholds and therapeutic goals (Vasan et al. 2001). According to the classification approaches developed by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI) the adults may be categorised as per given in table 2.1.

Table 2.1: Classification of blood pressure for adults

Blood Pressure Classification	SBP (mmHg)	DBP (mmHg)
Normal	<120	and <80
Prehypertension	120–139	or 80–89
Stage 1 Hypertension	140–159	or 90–99
Stage 2 Hypertension	>160	or >100

SBP, systolic blood pressure; DBP, diastolic blood pressure

In children and adolescents, hypertension is defined as BP that is, on repeated measurement, at the 95th percentile or greater adjusted for age, height, and gender (1).

Decades of epidemiologic research has established elevated blood pressure as a major contributor of cardiovascular diseases (O'Donnell et al., 2002). Elevations in both systolic and diastolic blood pressure are associated with strong, continuous increases in risk of stroke, coronary heart disease, congestive heart failure, peripheral vascular disease, and renal disease across a very broad range of blood pressure levels (Carolyn E. Barlow et al. 2004). Framingham Heart Study has indicated that blood pressure values between 130–139/85–89 mmHg are associated with a more than twofold increase in relative risk from cardiovascular disease (CVD) as compared with those with BP levels below 120/80mmHg (JNC, 2003). Worldwide, 7·6 million premature deaths (about 13·5% of the global total) and 92 million disability-adjusted life years (DALYs, 6·0% of the global total) were attributed to high blood pressure. Furthermore, it has become a common health problem globally as a consequence of increased longevity and prevalence of contributing factors such as obesity, physical inactivity and an unhealthy diet (WHO, 1983). High blood pressure imposes a chronic strain on normal cardiovascular function. If left untreated, severe hypertension leads to congestive heart failure as the heart muscle weakens and is unable to maintain its normal pumping ability. Degenerating, brittle vessels can obstruct blood flow, or can burst, cutting off vital blood flow to brain tissue and precipitate a stroke. Thereby, hypertension plays a major etiologic role in the development of cerebrovascular disease, ischemic heart disease, cardiac and renal failure. This burden is distributed over different economic regions, age groups, and blood-pressure levels and is certainly not limited to people with hypertension (Vasan et al. 2001). In addition, hypertension often coexists with other cardiovascular risk factors, such as tobacco use, diabetes, hyperlipidemia and obesity, which compound the cardiovascular risk attributable to hypertension. Worldwide, these coexistent risk factors are inadequately addressed in patients with hypertension, resulting in high morbidity and mortality (WHO, 1983).

Absolute risk of cardiovascular disease for any given level of blood pressure rises with age. The SBP continues to rise throughout life in contrast to DBP which rises until approximately age 50 and tends to level off over the next decade, and may remain the same or fall later in life. Thus, DBP is a more potent cardiovascular risk factor than SBP until age 50; thereafter, SBP is more important (JNC 7, 2003). An elevated systolic blood pressure provides a more reliable and accurate predictor of the risk associated with hypertension than diastolic blood pressure (Katch, 2007).

In the context of this large and growing disease burden, strategies to improve population health require consistent and comprehensive measures of the contribution of major risk factors to premature mortality and disability.6,7 These estimates can elucidate the potential for prevention and provide an important input into health planning and other cost-utility decisions. Therefore, the importance of modifiable cardiovascular health risks, such as blood pressure, should not be overestimated or under estimated (Vasan et al. 2001).

Effective prevention strategies include lifestyle changes-regular physical activity, modest weight loss, stress management, smoking cessation, reduced sodium and alcohol consumption and adequate potassium, calcium and magnesium intake.

Table 2.2: Lifestyle modifications to prevent and manage hypertension

Modification	Recommendation	Approximate SBP Reduction†
Weight reduction	Maintain normal body weight (body mass index 18.5–24.9 kg/m2).	5–20 mmHg/10kg
Adopt DASH eating plan	Consume a diet rich in fruits,vegetables, vegetables, and low-fat dairy products with a reduced content of saturated and total fat.	8–14 mmHg
Dietary sodium reduction	Reduce dietary sodium intake to no more than 100 mmol per day (2.4 g sodium or 6 g sodium chloride).	2–8 mmHg
Physical activity	Engage in regular aerobic physical activity such as brisk walking (at least 30 min per day, most days of the week).	4–9 mmHg
Moderation of alcohol consumption	Limit consumption to no more than 2 drinks (e.g., 24 oz beer, 10 oz wine, or 3 oz 80-proof whiskey)per day in most men, and to no more than 1 drink per day in women and lighter weight persons.	2–4 mmHg

DASH, Dietary Approaches to Stop Hypertension; SBP, systolic blood pressure

From The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure

High levels of leisure-time physical activity have been associated with a reduced risk of hypertension. Exercise promotes weight reduction, decrease "bad" cholesterol levels in the blood (the low-density lipoprotein, LDL), as well as total cholesterol, and can raise the "good" cholesterol (the high-density lipoprotein level, HDL). When combined with other lifestyle modifications such as proper nutrition, smoking cessation, and medication use, it can dramatically reduce the risk. However, genetic factors contribute significantly to the inter-individual differences in endurance training-induced changes in blood pressure. Regular exercise controls the tendency for blood pressure to increase over time and relates more to prevent early mortality than to extending overall life span. During rhythmic muscular activity vasodilatation in the active muscles reduces total peripheral resistance to enhance blood flow through large portions of the peripheral vasculature. Increased blood flow rapidly increased systolic blood pressure during first few minutes of exercise and then levels off at 140 to 160 mmHg for healthy individual. As exercise continues, systolic blood pressure decline as the arterioles in the active muscle dilate, further reducing peripheral resistance to blood flow. Diastolic blood pressure remains unchanged throughout exercise. During maximum exercise, systolic blood pressure may increase to

200 mmHg or higher among healthy individual despite reduced total peripheral resistance. This level of blood pressure most likely reflects heart large cardiac output during maximal exercise by individuals with high aerobic capacity.

Upon completion of exercise blood pressure temporarily falls below pre-exercise levels which may last upto 12 hours. Such post exercise hypotension could be explained by the significant quantity of blood in visceral organs or skeletal muscle during recovery. Consequently, venous pooling reduces central blood volume and decreases arterial filling pressure and hence arterial blood pressure. Systolic and diastolic blood pressure decreases by 6 to 10 mmHg with aerobic exercise training in previously sedentary individuals. Relatively prolonged reductions in post-exercise blood pressure justify recommending multiple periods of physical activity interspersed throughout the day (McArdle et al., 2007).

2.2.2 Heart Rate

The number of times a human heart beats per minute is known as the heart rate. Resting heart rate (RHR) is one of the simplest cardiovascular parameters, which usually averages 60 to 80 beats per minute (bpm), but can occasionally exceed 100 bpm in unconditioned, sedentary individuals and be as low as 30 bpm in highly trained endurance athletes. However, rates below 60 bpm referred to as bradycardia and rates above 100 bpm referred to as tachycardia are clinical heart disorders. Elevated heart rate (>80-85 beats/min) measured under resting conditions is directly associated with risk of developing hypertension and atherosclerosis, and is a potent predictor of cardiovascular morbidity and mortality (Palatini P. 2007). Epidemiological evidences demonstrate that RHR, or its corollaries, namely post-exercise heart rate recovery, which is mediated primarily by vagal tone, and heart rate variability (HRV, beat-to-beat variability also mediated by autonomic nervous system, especially parasympathetic) correlates with cardiovascular morbidity and suggests that RHR determines life expectancy (Cook et al, 2006). Heart rate proves to be the best predictor after myocardial infarction, in patients with congestive heart failure, as well as in patients with diabetes mellitus or hypertension (Disegni et al, 1995; Hathaway et al, 1998).

Resting heart rate both contributes to and reflects cardiac pathology. Increased heart rate, due to imbalances of the autonomic nervous system with increased sympathetic activity or reduced vagal tone, has an impact on perfusion-contraction matching, which is the dynamic that regulates myocardial blood supply and function. In the healthy heart, increased metabolism as a result of increased contractile function results in increased myocardial blood flow and, to a lesser degree, increased oxygen extraction. In the presence of coronary artery disease, perfusion-contraction mismatching is localised to areas of inadequate supply. When coronary artery inflow is inadequate to meet demands, contractile and diastolic functions in the affected area are correspondingly reduced. An increase in heart rate results not only in an increase in myocardial oxygen demands, but also a potential impairment of supply resulting from a reduction of collateral perfusion pressure and collateral flow. This imbalance may promote ischemia, arrhythmias and ventricular dysfunction, as well as acute coronary syndromes, heart failure or sudden death (Arnold et al. 2008)

2.2.3 Pulse Rate

The stretch and subsequent recoil of the aorta wall propagates as a wave through the entire arterial system. The pressure wave readily appears as pulse in the

following are: the superficial radial artery on the thumb side of the wrist, the temporal artery and carotid artery. In healthy persons, pulse rate equals heart rate.

2.3 CARDIO-RESPIRATORY FITNESS

It is generally considered the most important component of health related fitness (Giam Choo Keong, 1981) and remains a significant predictor of hypertension risk (McArdle et al., 2007). It is defined as the ability of the cardiorespiratory system to respond adequately and safely to the blood, oxygen and other nutritional requirements of the body organ and tissues, particularly the working muscle during physical activity. Higher levels of cardiorespiratory fitness (CRF) often neutralises increased mortality associated with elevated blood pressure and reduce risk of developing hypertension among healthy normotensive persons. In other words, higher incidences of hypertension have been demonstrated among individuals with low fitness compared with fit individuals (Aina Emaus et al. 2011).

2.4 PHYSICAL FITNESS AND PHYSICAL PERFORMANCE TESTS

Physical Fitness: It is the ability to meet the demands of life with vigour and alertness without undergoing fatigue and to have sufficient energy beyond this to enjoy leisure time's activities.

Physical performance tests are conducted to analyse a person's fitness level.

2.4.1 Methods to Access Physical Fitness

i) Direct Calorimeter

ii) Treadmill Concept

iii) Ergometer Test

iv) Step Test

i) **Direct Calorimeter:** The calorie is the basic unit of heat measurement and the term calorimetry defines the measurement of heat transfer. There are 2 types of approaches of calorimetry:

   a) Direct

   b) Indirect:

      i) Open Circuit Spirometry

      ii) Closed Circuit Spirometry: This method has the ability to directly measure oxygen consumption. The subject breathes 100% oxygen from a purified container. The equipment is closed system because the subject-breathes in only the gas in the spirometer, soda lime removes the expired air's carbon di oxide.

ii) **Treadmill Test:** The objective of the test is to monitor the development of athlete's general endurance & to calculate vo_2 max through vo_2 submax. Vo_2 Max: It is defined as the maximum capacity of an individual body to transport

& utilise oxygen during incremental exercise which reflects the physical fitness of the individuals. This indicates an individual's capacity for aerobically resynthesising ATP.

Vo_2 Submax: Vo_2 max has many risks as a person is not aware that he may have heart problem, Vo_2 submax are those levels where one does not reach the maximum of respiratory and cardiovascular system. Example: Balke's modified Treadmill Test.

iv) **Step Test:** It is used to measure the energy output of the given subject by checking the response of heart rate and to find the rapid fitness index.

Example: Harvard step test- it was developed by Broucher et al in 1943 at Harvard fatigue laboratory during World War II. The fitness is accessed with the help of Rapid Fitness Index (RFI) which reads as:

$$RFI = \frac{\text{Test duration in seconds}}{5.5 \times \text{pulse count between 1 – 1.5 mins}} \times 100$$

Depending upon the RFI (Rapid Fitness Test) value, a person's physical fitness can be categorised into the following:

Age Group	Physical Fitness
Below 55	Poor
56-64	Average
65-79	Average
80-89	Good
Above 90	Excellent

Recovery heart rate from a standardised stepping exercise can classify individuals according to level of cardiovascular fitness assessed on the basis of VO_{2max} with a reasonable degree of accuracy. The individual steps to a four-step cadence, up-up-down-down continuously for 3 minutes on a wooden bench 16¼ inch high. Metronome was used to monitor the stepping cadence, which was set at 88 beats per minute (22 complete steps per minute) for females and 96 beats per minute (24 complete steps per minute) for males. The step test begins after a brief demonstration and practice period. Prior to the test, subjects warm up by stretching lower limb muscles and brisk walking. After completion of test, subjects remains standing while pulse rate was measured for 15 seconds period, 5 to 20 seconds into recovery. 15 seconds recovery heart rate is converted to beats per minute (15 s HR x 4) and estimates VO_{2max} using equation:

Males: VO 2max = 111.33 – [0.42 × Step-test pulse rate (b.min-1)]

Females: VO2max = 65.81 – [0.1847 × Step-test pulse rate (b.min-1)]

Sports performance is based in a complex and intricate diversity of variables, which include range of factors including physiological, anthropometric dimensions, reflecting body shape, proportionality and composition, biomechanical and skill traits within different sports. For example in swimming

body fat gives greater bounce to swimmers and contributes to improved efficiency by decreasing hydrodynamic drag. Fat layer acts as thermal insulator to preserve body heat in the water, despite high rate of heat production during competition (Norton et al., 1996). Regardless of the sport discipline, on average, athletes are less fat and more muscular than non-athletes are.

2.5 ENVIRONMENT AND HUMAN CARDIO-RESPIRATORY FUNCTION TRAITS

In physiological anthropology, we study the adaptation of human cardio-respiratory function with different environmental condition i.e. heat, cold and high altitude. i) Adaptation to Heat or Hot Climate: when temperature of environment is more than the body, then it is called heat stress. Heat loss occurs by 4 processes-

1) Radiation
2) Conduction
3) Convection
4) Evaporation

1) **Radiation –** All objects including humans continuously emit electromagnetic heat waves (radiant energy). Our body usually remain warmer than the environment, making the exchange of radiant heat energy, move through the air to solid and cools down objects in the environment. This form of heat transfer does not require molecular contact between objects. The body absorbs radiant heat energy from the surroundings when a person's temperature exceeds skin temperature.

2) **Conduction –** Heat exchange by conduction involves direct heat transfer from one molecule to another through a liquid, solid or gas. The circulation transports most body heat to shell, but a small amount continually moves by conduction directly through the deep tissues to the cooler surfaces. The rate of conductive heat loss depends on two factors-

   a) Temperature gradient between skin and the surroundings surface.
   b) Thermal qualities of the surfaces.

3) **Convection –** The convection depends on how rapidly the air (adjacent to the body) exchanges once it warms. If air movement or convection proceeds slowly, the air next to the skin warms and acts as a zone of insulation, which minimises further conductive heat loss. Heat loss through convection increases because it continuously replaces the zone of insulation.

   Another mechanism of radiating body heat is vasodilation. In vasodilation, capillaries near the skin's surface widen to permit increased blood flow to skin. The visible effect is increased redness of the skin, particularly of the face.

4) **Evaporation –** Water vaporising from the respiratory passages and skin surface continuously transfers heat to the environment. For example- the body's surface contains 2-4 million sweat glands. During heat stress, these

eccrine glands secrete large quantities of hypotonic saline solution (Nacl). Evaporation of sweat from the skin exerts a cooling effect. The cooled peripheral blood flow then flows to the deeper tissues to absorb additional heat on its return to the heart.

ii) **Adaptation to Cold Climate:** Body adapts in cold climate by combined factors i.e. increase heat retention & enhance heat production. Out of the two, heat retention is more efficient because it requires less energy and this is derived from dietary sources.

a) Increased metabolic rate and shivering- These are short term responses to cold climate, both of which generate body heat for a short time.

b) Vasoconstriction- It restricts heat loss and conserves energy. In addition, human process a subcutaneous fat layer (beneath skin) that provides an insulative layer throughout the body and conserves heat within the body. It also restricts capillary blood flow to the surface of the skin, thus reducing heat loss at the body surface.

c) Vasoconstriction and vasodilation- The compromise provides periodic warmth to the skin that helps in preventing frostbite in below freezing temperatures. At the same time, because vasodilation is intermittent, energy loss is restricted with more heat retained at the body's core.

iii) **Adaptation to High Altitude**- High altitudes are defined between 3048 meters (m) (10,000 ft) to 5486 m (18,000ft) above sea level. It is a multi stressor environment including hypoxia, nutritional stress and cold radiation. The adaptive responses that improve one's tolerance to altitude hypoxia are broadly termed acclimatisation. The longer term acclimatisation process involves physiologic and metabolic adjustments that greatly improve tolerance to altitude hypoxia. The main adjustments involve (1) restabilising the acid- base balance of the body fluids, (2) increased formation of haemoglobin and red blood cells and, (3) changes in local circulation and cellular function. (Mcardle & Katch, 2007)

2.6 IMPACT OF AIR POLLUTION ON CARDIO-RESPIRATORY FUNCTIONS

Over the last decade, accumulating epidemiological and clinical evidence has led to a heightened concern about the potential deleterious effects of ambient air pollution on pulmonary and cardiovascular system depending on the physical and chemical properties of contaminants, time and frequency of exposure. The concerned environmental air pollutants include carbon monoxide, oxides of nitrogen, sulfur dioxide, ozone, lead, and particulate matter ("thoracic particles" [PM_{10}], "fine particles" [$PM_{2.5}$] "coarse particles" [PM_{10} to $_{2.5}$]). These pollutants are associated with increased hospitalization and mortality due to cardiovascular disease, especially in persons with congestive heart failure, frequent arrhythmias, or both (Brook et al., 2004). The cardiac effects are a consequence of inflammation in the lung, leading to the release of cytokines with secondary effects on blood constituents interfering with coagulability and stability of atheromatous plaques (Seaton et al, 1995). The lung inflammation is a consequence not of the mass but of the number of particles, particularly those in the ultrafine (<100 nm) size

range. It has the potential to explain both short-term morbidity and also longer-term atherogenesis. Other hypothesis, suggests that inhalation of air pollutants might trigger reflex changes in the control of the heart (Ayres, 2006). Increased parasympathetic activity normally leads to coronary vasodilation, in the presence of coronary artery disease, parasympathetic stimulation may lead to net coronary constriction. (Pope et al., 2004). Accelerated heart rate, diminished heart rate variability (HRV), and increased incidence of arrhythmias on exposure to air pollution suggest the primary effects on myocardial excitability or autonomic regulation of the heart or both.

Lung function, as measured by spirometry is an excellent operative marker of the effects of air pollution. It is objective and quantitative, an early predictor of cardio-respiratory morbidity and mortality, able to describe trajectories to the occurrence of chronic obstructive pulmonary disease (COPD) and coherent with experimental data on deposition and accumulation of pollutants in airways and lungs and the resulting systemic inflammation and oxidative stress (Sunyer, 2009). Understanding and quantifying the contributions of environmental exposures to lung disease is difficult because individuals respond differently to the same factors. The variations in response arise from different susceptibilities, including genetic predisposition, developmental stages of life, presence of co-existing diseases, other exposures, and lifestyle differences such as varying nutritional status and physical activity levels.Long-term exposure to smoke or dust damages the lung airways and air sacs, and may eventually cause chronic obstructive pulmonary disease (COPD) indicated by FEV_1 less than 0.80. COPD is characterised by chronic inflammation throughout the airways, parenchyma, and pulmonary vasculature. People with COPD usually have a combination of two conditions: chronic bronchitis and emphysema. In chronic bronchitis the airways become inflamed and their walls thicken, so that the air passage narrows down (Figure 2.1). The damaged airways also produce a lot of thick, sticky mucus which causes frequent coughing. In emphysema, destruction of lung parenchyma leads to the loss of elastic recoil and alveolar septa which increases the tendency for airway collapse. The combination of these two conditions obstructs airflow through the lungs and the individual becomes increasingly breathless and fatigued. The damage caused to the lungs is irreversible, and the condition is progressive; that is, the damage gradually accumulates and the symptoms worsen. Indoor air pollution from biomass fuel, burned for cooking and heating in poorly vented dwellings, has also been implicated as a risk factor for the development of COPD. The long term adverse effect of air pollution on lungs has been evident among children during their development. Cumulative pollution related deficits in the average growth in lung function results in a strong association between exposure to air pollution and a clinically low FEV at the age of 18 years. FVC is largely a function of the number and size of alveoli, with differences in volume primarily attributable to differences in the number of alveoli, since their size is relatively constant. However, since the postnatal increase in the number of alveoli is complete by the age of 10 years, pollution related deficits in the growth of FVC and FEV_1 during adolescence may, in part, reflect a reduction in the growth of alveoli. Another plausible mechanism of the effect of air pollution on lung development is airway inflammation, such as occurs in bronchiolitis; such changes have been observed in the airways of smokers and of subjects who lived in polluted environments (Gauderman et al. 2004).

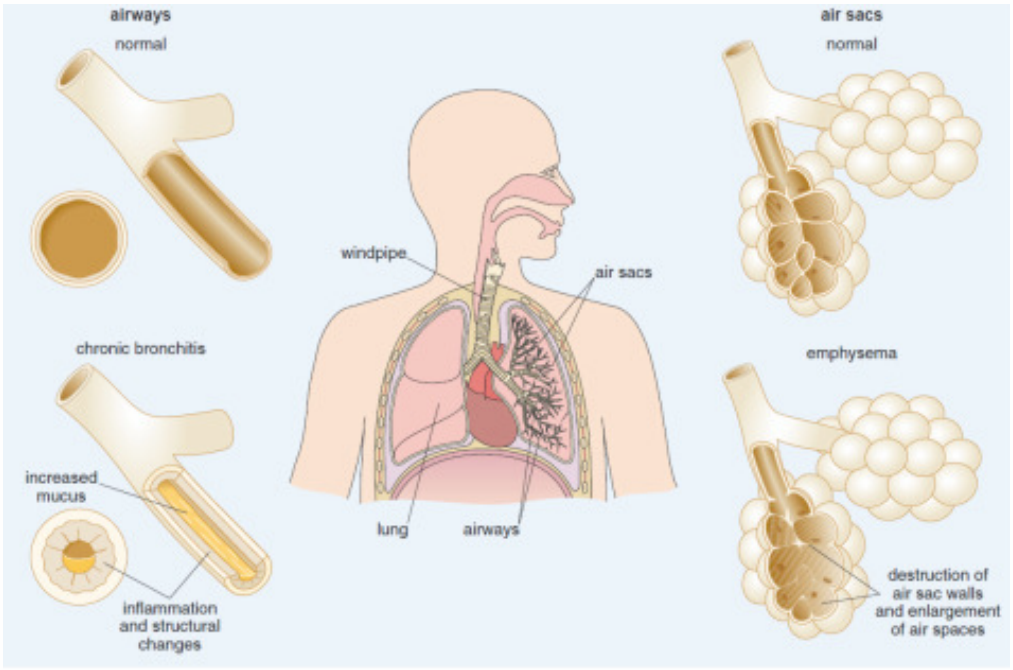

Fig. 2.1: The lungs are filled with a network of airways and air sacs

In COPD, damage to the lungs results in narrowing of the airways and destruction of the walls of the air sacs.

There is negative dose-dependent association between various outdoor exposures and lower levels of the forced expiratory volume in one second (FEV_1), forced vital capacity, and maximal mid-expiratory flow rate.

The short-term negative impact of exposure to air pollutants on respiratory volume and flow is limited to individuals with already impaired respiratory function. Individuals with chronic obstructive pulmonary disease (COPD) including asthma, ischemic heart diseases (IHD), congestive heart failure, heart rhythm disorders, and diabetes are "frail" population susceptible to the acute effects of air pollution. Decrements in lung function indices (FVC and/or FEV_1) associated with increasing concentrations.

2.6.1 Impact of Smoking on Cardio-respiratory Functions

Of all inhalational exposures, cigarette smoking is the major factor which contributes to the risk of pulmonary diseases in most countries. When the tobacco leaf is burnt, the smoker is exposed to over 4,000 chemicals, a number of them being carcinogenic affecting almost all organs. Many other organic and inorganic chemicals in the gaseous, volatile, and particulate phases of cigarette smoke appear to contribute to smoke's toxicity to the respiratory system, including hydrocarbons, aldehydes, ketones, organic acids, phenols, cyanides, acrolein, and nitrogen oxides.

Cigarette smokers have a higher prevalence of lung-function abnormalities and respiratory symptoms, a greater annual rate of decline in FEV_1, and higher death rates for COPD than nonsmokers. The patient will have a reduced FEV1 and FEV1/FVC ratio.Cigarette smoke is toxic to the cilia that line the central breathing passages. Some components contribute to the development of chronic mucus hypersecretion in the central airways, whereas others play a greater role in the

production of emphysematous injury to the peripheral air sacs. Smoking also induces abnormalities in the inflammatory and immune systems within the causing inflammatory cells to produce an enzyme called elastase, which in turn breaks down elastin, an important protein that lines the elastic walls of the air sacs (Fera et al. 1986; U.S. Department of Health and Human Services, 1984). Moreover, oxidants present in cigarette smoke can inactivate a separate protective enzyme called alpha,-antitrypsin, which inhibits the destructive action of elastase. Smoking during pregnancy may also pose a risk for the fetus, by affecting lung growth and development *in utero* and possibly the priming of the immune system.

Cigarette smoking is a major contributing cause to coronary heart disease (CHD), stroke and other atherosclerotic diseases of the circulatory system. The relationship between cigarettes consumption and relative risk of coronary heart disease appears to be independent of other factors such as raised serum cholesterol, hypertension, obesity and physical inactivity. Smokers are at two fold increased risk of CHD than non-smokers. Nicotine, the major psychoactive component of smoke, increases heart rate and blood pressure via stimulation of autonomic nerves. It affects cholesterol metabolism by lowering the level of protective high-density lipoprotein (HDL) cholesterol and increasing bad cholesterol low density lipoprotein (LDL). Nicotine damages the inner lining of blood vessels, thus enhancing the transfer of low-density lipoprotein (LDL) cholesterol particles across the arterial wall and development of cholesterol-laden plaque. Furthermore, smokers have elevated levels of thrombin, an enzyme that causes the blood to clot. The adherence of blood platelets to the lining of arterial blood vessels and the formation of blood clots narrow down artery (Willett et al., 1983; Pittilo et al., 1984; Penn et al., 1994). Carbon monoxide in cigarette smoke binds to the haemoglobin in red blood cells, thereby reducing the oxygen-carrying capacity of the blood.

2.6.2 Impact of Occupation on Cardio-respiratory Function

Occupational lifetime exposure to dust, fumes, endotoxin, organic dusts, and sensitising agents evaluated in the general population have found to be associated with airway hyper-responsiveness, chronic bronchitis and airflow obstruction. The effect of occupational exposure on lung function is related with the duration of exposure. When the exposures are sufficiently intense or prolonged, occupational dusts and chemicals (vapours, irritants, fumes) can cause COPD independently of cigarette smoking and increase the risk of the disease in the presence of concurrent cigarette smoking.

The airways, from nares to alveoli, come into contact with 14,000 litres of air in the workplace during a 40-hour work week. Physical activity can increase ventilation, and thus exposure to contaminants, up to 12 times the levels at rest. As ventilation increases, breathing shifts from nasal to a combination of oral and nasal, allowing a greater volume of air to bypass the cleansing nasopharynx and further increasing the exposure of the lower airways to inhaled materials. Strong irritants (such as ammonia) produce an aversive response, whereas materials with little sensory effect (such as asbestos) can be inhaled for prolonged periods and result in serious injury (Beckett, 2000).

It has also been suggested that the occurrence of respiratory symptoms represents the earliest response and a risk factor for subsequent loss of pulmonary function. Cumulative exposure to dust and increasing working years in specific jobs have

been associated with a steeper decline in FEV1 (Jaén et al., 2006). The spirometric parameters estimate among individuals exposed to ambient levels of particulate matter/dust shows dose-response relationships more pronounced for forced vital capacity (FVC) compared to forced expiratory volume in first second (FEV1). Forced expiratory volume in first second (FEV1) is the volume of air (expressed in litres) exhaled in the first second of the FVC manoeuvre, and it is decreased in obstructive lung diseases while restrictive lung diseases decrease FVC. Forced mid-expiratory flow rate (FMEF or FEF25-75%) is the rate of flow of air between 25% and 75% of the FVC. It is a sensitive measurement and is determined from forced expiratory spirogram. It is reduced in early obstruction involving the smaller airways, which are the primary site of deposition of inhaled pollutants (Jafary et al. 2007). Such relationship between occupational exposure and over-shift changes in lung function has been documented in different occupational set up such as coal mine, textile factory, grain processing and animal feed industry etc.

Barometric pressure is elevated in deep underground mines and reduced at high altitude mines. Chronic intermittent hypoxia at altitude has been reported to induce physiological adaptations and symptoms of benign acute mountain sickness (AMS) in mine workers while increased barometric pressures in deep mines increase air temperatures, increase convective heat exchange and reduce sweat evaporation rates.Ambient exposure to particulate matter air pollution such as silica dust, asbestos or welding fumes is a risk factor for cardiovascular disease. It has been proposed that inhalation of small particles induces an inflammatory reaction in the airways and subsequent induction of systemic inflammation and coagulation disturbances (Seaton et al. 1999). Intermittent exposure to magnetic field leads to reduction in heart rate variability and arrhythmia. Heart rate variability is a marker of autonomic cardiac control, and reductions in heart rate variability have been shown to predict sudden death, all-cause mortality, and heart disease in prospective epidemiologic studies (Håkanssonet al. 2003).

2.7 SUMMARY

The increasing epidemic of health risks has paved way for health promotion policies which focus purely on behavioural change and towards ecological approaches that involve communities in nurturing environment and behavioural changes. It is because of this trend there is an increased popularity of interdisciplinary and participatory approaches to health related issues. Anthropologists facilitate in the understanding of the interaction of physical and social environmental conditions and can be strong participants in such an integrally interdisciplinary approach. In this unit we learn how various physiological dimensions are used in practicing anthropology. It is well understood that the heart provides the force to push blood throughout the vascular circuit. The epidemiological research over the years established elevated blood pressure as a major contributor of cardiovascular diseases and that increased both systolic and diastolic blood pressure are associated with strong, continuous increases in risk of stroke, coronary heart disease, congestive heart failure, peripheral vascular disease, and renal disease across a very broad range of blood pressure levels. Heart rate measured by monitoring one‘s pulse differs significantly between individuals based on fitness, age and genetics. Cardiovascular fitness is designated as the most significant component of health related fitness and remains a

significant predictor of hypertension. **Step test** classifies individuals according to level of cardiovascular fitness. Eepidemiological and clinical signals has led to a heightened concern about the potential harmful effects of ambient air pollution on pulmonary and cardiovascular system depending on the physical and chemical properties of contaminants, time and frequency of exposure. Cigarette smoking is the major factor which contributes to the risk of pulmonary diseases in most countries whereas ooccupational lifetime exposure to dust, fumes, endotoxin, organic dusts, and sensitising agents evaluated in the general population have found to be associated with chronic bronchitis and airflow obstruction.

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Suggested Reading

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Sample Questions

1) Describe blood pressure, heart rate, pulse rate and associated health risks.

2) Explain cardiorespiratory fitness in relation to maximum oxygen consumption.

3) Elaborate applied aspects of anthropometric measurements in sports.

4) Explain the physiological changes experienced by human on exposure to air pollutant, smoke and occupation.

Paleo-anthropology is a subspecialty in physical anthropology which is interested in the search for fossil remains from prehistoric times to trace the development of outstanding human physical, social and cultural characteristics. Primatology: studies the animals that most closely resemble human beings in terms of physiological and. 12. Introduction to Anthropology. Unit Writers Dr Rashmi Sinha (Units 1,2 & 3) Reader, Faculty of Anthropology SOSS, IGNOU, New Delhi. Mrs. Narinder Jit Kaur Retired, Associate Professor in English Government Mohindra College, Patiala. Dr. P. Venkatramana (Unit 4) Assistant Professor, Faculty of Anthropology SOSS, IGNOU, New Delhi.Â Unit 2 concerns with the interdisciplinary and trandisciplinary approaches in relation to physical anthropology. There are some strong connections between physical anthropology and other disciplines like forensic science, life sciences, medical sciences, earth sciences, human biology, environmental sciences, social sciences, human engineering and technology, and physical sciences. Learn about physical anthropology chapter 2 with free interactive flashcards. Choose from 500 different sets of flashcards about physical anthropology chapter 2 on Quizlet.Â Physical anthropologists seek to undersâ€¦ What is anthropology? evolutionary or nonevolutionary. the changes resulted in genetic changes.Â An anatomical, physiological, or behavioral response of organiâ€¦ The field of inquiry that studies human culture and evolutionaâ€¦ The practical application of anthropological and archaeologicaâ€¦