Physiological Anthropology
Unit d

3. Impact of air pollution on cardio-respiratory functions –

- Air pollution is one of the biggest environmental threats to human health and is estimated to contribute to 2.9 million annual deaths globally, of which more than 85% occur in low-income and middle-income countries (LMICs).

- Particulate matter (PM), a heterogeneous mixture of suspended solid and liquid particles from different sources and varying in size, mass, and chemical composition, is often acknowledged as the most damaging element of air pollution to human health, with its ability to penetrate deeply into the human respiratory and circulatory systems and cause direct localized and systemic damage.

- Both short-term (days) and long-term (years) exposure to PM has been independently associated with increased risks for mortality and morbidity, particularly cardiorespiratory outcomes.

3.1 EFFECTS OF AIR POLLUTION ON CARDIOVASCULAR FUNCTIONS:

- **Cardiovascular mortality:** In various studies, short-term PM2.5 exposure increased the relative risk for acute myocardial infarction (MI) by 2.5%. Given the fact that air pollution exposure occurs over a lifetime, the repetitive, near-continuous exposure to air pollution has been hypothesized to promote atherosclerosis and recurrent events.

- Indeed, longer-term exposures over several years appear to pose amplified risks. There are now many studies across the world demonstrating an increase in acute Cardiovascular mortality due to air pollution.

- **MYOCARDIAL INFARCTION (MI):** Various studies across the globe have explored the association between short-term changes in air pollution and daily changes in MI. A systematic review and meta-analysis of studies of short-term air pollution exposures and MI showed that PM2.5, along with nitrogen dioxide (NO2), and sulfur dioxide and carbon monoxide were associated with increased risk of MI.
- **CEREBROVASCULAR DISEASE:** In a systematic review and meta-analysis of 94 studies until 2014, involving 28 countries, a 10 mg/m^3 increase in PM2.5 and PM10 concentration was associated with a 1% increase in relative risk for admission to the hospital with stroke and stroke mortality. Also, living close to the roadway and poverty appear to be positively associated with ischemic stroke and stroke severity

- **HEART FAILURE:** In a systematic review and meta-analysis of 35 studies, a short-term increase in gaseous components and PM (both PM10 and PM2.5) was associated with increased risk for heart failure hospitalization or death.
- **HYPERTENSION:** The association between air pollution and hypertension has been reviewed extensively and has been the subject of at least 4 recent meta-analyses. Increases in ambient PM2.5 by 10 mg/m3 are consistently associated with 1 to 3 mm Hg elevations in systolic and diastolic blood pressure over the ensuing few days.

Longer-term exposures have been linked chronic elevations in blood pressure and with an increased prevalence or incidence of hypertension in many studies as well

MECHANISMS OF AIR POLLUTION–MEDIATED CARDIOMETABOLIC DISEASE: The knowledge of mechanisms underlying air pollution–mediated systemic Cadiovascular risk is still evolving but can be encapsulated into two broad pathways:

i) **Prothrombotic pathways**: In human studies, inhalation of diluted diesel exhaust particulate matter increased thrombotic response, as assessed by ex vivo flow chamber perfusion studies, and increased platelet–leukocyte aggregates with exposure. Rapid platelet sensitization could occur due to direct contact in the lung or translocation of ultrafine particulate matters (PM). Activation of platelets as well as alteration of the plasminogen activator inhibitor/tissue plasminogen activator balance in patients with other risk factors may heighten their susceptibility to cardiovascular events.

ii) Epigenomic changes: Extensive human and animal model data indicate that environmental influences during critical periods of prenatal and postnatal development influence developmental trajectories and chronic disease susceptibility. A common finding in environmental changes such air pollution can change leads to epigenetic changes. Further, even small changes in methylation can have a strong effect on transcriptional activity.

3.2 EFFECT OF AIR POLLUTION ON RESPIRATORY FUNCTIONS:

- Adverse effects of air pollutants on the lungs include disruption of airway epithelial barrier and cellular signalling pathways, parenchymal destruction, oxidative stress, impairment of phagocytosis, inflammatory cell infiltration, dysregulated cell immunity, epigenetic modifications and autophagy.
- Following are the major respiratory heath complications occurs due to air pollution:

i) **Asthma-** Air pollution contributes to increased asthma prevalence and symptom onset. Many developing countries (e.g., rural China, India, and Malawi) still use open fires for cooking. Despite mixed findings for biomass smoke exposure and asthma risk, coal combustion for heating and cooking conferred higher risks of childhood asthma. Environmental tobacco smoke and chemical emissions from new furniture were risk factors for asthma.

ii) **Chronic obstructive pulmonary disease (COPD)- c**hronic obstructive pulmonary disease (COPD) is a progressive, irreversible inflammatory disease of the lungs that makes it hard to breathe. Common symptoms include a persistent cough, wheezing, production of phlegm, shortness of breath, and a feeling of tightness in your chest, though these symptoms may not be noticeable until you're in the later stages of the disease. COPD is not curable, but it's a preventable and treatable illness. Long-term exposure to both indoor and outdoor air pollution can have severe consequences on lung health that are generally irreversible and research supports a correlation between air pollution and chronic obstructive pulmonary disease (COPD). In addition, both indoor and outdoor air pollution can exacerbate lung disease which is already present.

iii) **Lung cancer-** Smoking and solid-fuel (eg, coal and wood) combustion predispose to lung cancer, with spatial and gender variation. Time-based multiple risk factor models have shown smoking and solid-fuel use collectively contributed to 75% of lung cancer deaths in world. Other studies also suggest that both indoor and outdoor air pollution play crucial roles in lung cancer pathogenesis. Indeed, improved indoor ventilation, which increases air quality, has been associated with a lower incidence of lung cancer.

Targets for intervention:

- Environmental health cannot be achieved by government agencies alone. Research institutions should be actively engaged in the development of efficient technologies to reduce air pollution.
- Strict observance of laws and regulations is a principal duty for individual corporations.
- Finally, education in schools regarding precautions to take to limit the detrimental effects of air pollution should be instituted.
- At a personal level, wearing face masks is the simplest approach to minimize pollutant exposure.
- Antioxidant-rich diets (leafy vegetables and fruits) might help to mitigate adverse impacts of air pollution by counteracting oxidative stress. Studies shows that antioxidant supplementation with vitamin C and E above the minimum dietary requirement led to attenuated nasal inflammation and partially restored antioxidant levels in asthmatic patients exposed to high levels of ozone. Hence, daily intake of fresh vegetables and fruits or antioxidant medication and vitamins C and E should be promoted as part of healthy living.

4. EFFECTS OF OCCUPATION ON CARDIO-RESPIRATORY FUNCTIONS:

4.1 Effects of Occupation on Cardiovascular Functions:

- Cardiovascular diseases (CVD) are diseases that involve the heart or blood vessels. Among the many conditions that make up CVD are coronary heart disease, stroke, arrhythmia, cardiomyopathy, and heart valve problems. CVD is the leading cause of death worldwide.
- While it has been shown that more than half of those with CVD (53%) are less than 60 years old, and circulatory diseases are a leading cause of death and permanent disability among workers, much is unknown about how occupational risk factors contribute to CVD.

OCCUPATIONAL FACTORS THAT EFFECT CARDIOVASCULAR FUNCTIONS:

i) **Physical Demands of Work:** The development of mechanized and automated work processes has reduced the prevalence and severity of highly metabolically demanding work, however such demands remain a required component to some types of work.

- Metabolic demand exceeding approximately 35% of aerobic capacity has been proposed as a source of work related fatigue.

- A recent population-based longitudinal study, demonstrated an association between demands and progression of atherosclerosis

ii) **Work Organization:** Substantial research now exists relating work organization to cardiovascular disease, with several recent reviews and large prospective studies concluding that there is a relationship between features of work organization and CVD and its major risk factors such as hypertension.

iii) **Job control:** The most consistent work organization feature associated with CVD is low job control. In occupations with low control, a worker has limited decision making authority over how, when, or where their work tasks are performed–which includes the skills used, pace, intensity, and loads. In addition to an association between low control and CVD, there has been consistent evidence that low worker control is associated with increased rates of fatigue as well as physiological markers of fatigue.

PROPSED MECHANISM PATHWAY: The mechanistic pathway suggests that an intermediate step in the pathway from increased occupational metabolic demands to CVD includes the development of fatigue and is based on 3 assumptions: (1) occupational metabolic demands exceeding ~35% of worker aerobic capacity [metabolic demand/aerobic capacity index (MD/AC index)] leads to fatigue; (2) low worker control increases the likelihood that high MD/AC index will result in fatigue; and (3) fatigue increases risk for CVD.

- Physical therapists are uniquely qualified to evaluate and intervene with the interacting concepts of the proposed pathway: decreasing environmental demands, improving individual capacity, and fostering control over action as a potent means to decrease fatigue.

4.2 Effects of Occupation on Respiratory Functions:

- Respiratory diseases due to occupational exposure are mostly related to dust inhalation and the deposition of inhaled particles, depending on the size, physical properties and chemical properties of the aerosol, frequency and duration of exposure, and individual response to dust particles in the lungs.

- The available evidence corroborates the relationships between the exposure to dust produced by traffic and respiratory disorders, reduced pulmonary function, cardiovascular disease and lung cancer.

- For example- Chronic obstructive pulmonary disease (COPD) is the third leading cause of death in worldwide. Although the primary cause of COPD is tobacco smoking, increasing evidence implicates occupational and environmental exposures as additional etiologic factors. The long and continuous inhalation of non-industrial dust has been reported to be one of the critical factors in the development of COPD, resulting in obstructive ventilatory patterns.

- Occupational exposures may influence the course of COPD in several ways:

(1) Causing COPD

(2) Modifying the effect of tobacco smoke in causing COPD, such as amplifying its adverse impact

(3) Creating greater disability by adding exposure-related impairment to that due to tobacco smoking

(4) Accelerating the rate of decline of respiratory function in persons with established COPD.