

Modern lifestyle vs health

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ABSTRACT: Life style disease is disease which is related to living habits and eating habits. The lifestyle disease are noncommunicable disease it includes cardio vascular diseases, diabetes, chronic lung disease, cancer and obesity. Lifestyle diseases are also pron by the some micro organisms and lifestyle disease are also attracts the other disease like due to obesity there is more chances of heart disease because of hyperlipidemia and hypertention. Cancer is also a result of chewing tobacco and smoking . In chronic lung disease causative factors like pollution, dust and gases worsen the condition of person so the surrounding environment is alsoplays role in health. This life style disease arenot curable but they can be maintain by healthy lifestyle and dietary habits.

KEYWORDS: lifestyle disease, cardio vascular disease(CVDs),chronic obstructive pulmonary disease (COPD),human immune deficiency virus(HIV),

I. INTRODUCTION:

The 21st century is the era of modernization and the fast lifestyle where everyone is busy updating their modern lifestyle. Everyone is trying to look fashionable and remain updated; this sort of behavior leads to many problems. Nowadays, people are so busy in their life and routine work they don't focus on their physical activity and diet, leading to deterioration in health and behavior changes. Most people think that eating junk food (pizza, burger, hot-dog, etc.), drinking alcohol, smoking cigarette, etc. makes us modern and give status in society. This sort of lifestyle leads to the introduction of several diseases and complications, such as depression, heart-related problems, anxiety, etc. Due to the modernization of lifestyle, a new group of the disease and infection generates called lifestyle diseases and conditions in the world^[1,2]

Lifestyle disease isa disease related to the change in people's behaviors and the way they live there life. These are non-communicable diseases that occur due to lack of physical activity, unhealthy food habits, alcohol consumption, drugs, and smoking.Common examples of lifestyle

diseases are coronary heart diseases (CHD), type 2 diabetes, chronic obstructive pulmonary disease, and some types of cancer^[3].Lifestyle diseases are associated with a Non CommunicableDisease (NCDs) because they are the result of a combination of factors, including genetics, physiology, environment, and behaviours.An unhealthy lifestyle can contribute to the development of risk factors of non-communicable diseases (NCDs) such as overweight and obesity can lead to NCDs such as diabetes, hyperlipidemia, cardiovascular diseases (CVDs), and hypertension^[4].

WHO has identified four significant NCDs, i.e., diabetes, CVDs, cancer, and chronic lung disease/chronic obstructive pulmonary disease (COPD),that share common lifestyle-related behavioralrisk factors^[5].The risk factors includethe use of tobacco (smoking/chewing), physical inactivity, unhealthy diet, and consumption of alcohol leads to essential metabolic and physiological changes, raised blood pressure (BP), overweight/obesity, increased blood glucose, and cholesterol levels^[6].Heart diseases, cancer, diabetes, chronic pulmonary, and mental disorders area real burden for health systems in developed countries^[7].

Diseases and risk factor related to lifestyle disorders:



Figure-1



Figure-2

(figure -1:lifestyle disease and figure-2:factors related to lifestyle disease)^[8]

Life style disease:

Life style disease are all related to the physical and diet habits of the people due to that cardio vascular disease are generated like

consuming high salt in food results in high blood pressure and eating much fatty food increased the levels of cholesterol in body so the combination of this two are major reason for the CVDs^[9]. Nowadays people are consuming more amount of sugar in form of desserts, ice cream and soft drinks by not knowing how much they are consuming which is increased the risk of diabetes. Cancers is also included in lifestyle disease because causes of cancers are exposure of chemicals, fumes, uv radiation and also unhealthy food, smoking, drinking and chewing tobacco. The habits of smoking/chewing tobacco and the fumes of industrial areas also the dust of asbestos are causes difficulty in breathing which is worsen by the disease copd. Obesity is shown in every age group of the people due to unhealthy eating habits like fast food, sweets, ice cream and drinking soft drinks which is promoting the other disease like hyperlipidemia by increased cholesterol levels or high sugar levels which is results in diabetes^[10].

1. Cardiovascular disease:

Cardiovascular diseases (CVDs) are the primary cause of mortality globally, taking an estimated 17.9 million lives each year. CVDs are a group of disorders of the heart and blood vessels and include coronary heart disease, cerebrovascular disease, rheumatic heart disease and other conditions^[11]. CHD also known as coronary artery disease, is the narrowing of the blood vessels, as a result of atherosclerosis that supply blood and oxygen to the heart. CHD can lead to unstable angina, myocardial infarction (MI), and heart failure^[12]. In atherosclerosis, the atheromatous plaque is formed due to high lipid levels in blood vessels. The risk factors for atherosclerosis are smoking, obesity, sedentary habits, dyslipidemia, glucose intolerance and hypertension^[13]. Pathophysiology of coronary heart disease includes the number of microbiological agents in which bacterial agents like helicobacter pylori, chlamydia pneumoniae and viral agents like human immune deficiency virus, Epstein-bar virus, hepatitis virus, mycobacterium tuberculosis, human cytomegalovirus and dengue virus^[14]

2. Diabetes:

Diabetes mellitus is chronic disorders that are characterized by high blood glucose levels (hyperglycemia) as a result of insulin deficiency or cellular resistance to the action of insulin^[15]. Majorly the type 1 and type 2 diabetes occurs in patients. According to WHO, about 422 million

people worldwide have diabetes, particularly in low-and middle-income countries, and 1.6 million deaths are directly attributed to diabetes each year^[16]. In modernization the people are consuming more sugar by not knowing in diet like drinking more soft drink and energy drink also eating the desserts without limit which results in high glucose level increased the chances of hyperglycemia and insulin deficiency. People having the diabetes are more prone towards the infections because the glucose level in the blood is always high^[17]

3. Cancer:

Cancer is the second leading cause of death globally. Cancer is a large group of diseases that can start in almost any organ or tissue of the body when there is an abnormal growth of cells going beyond their usual boundaries to invade adjoining parts of the body or spread to other organs. The latter process is called metastasizing and is a significant cause of death from cancer. A neoplasm and malignant tumour are other common names for cancer^[18]. Nowadays pollution is increased due to the petroleum industries, pharmaceuticals, chemicals and dusts from the mines and also smoking habit it causes lung cancer. Hormonal changes due to unhealthy diet and obesity leads breast cancer and cervical cancer. Oral cancer is caused by chewing tobacco and drinking alcohol with having stomach infection enhances the chances of stomach and liver cancer. The Lung, prostate, colorectal, stomach, Oral and liver cancer are the most common types of cancer in men, while breast, colorectal, lung, cervical, and thyroid cancer are the most common among women^[19]

4. Chronic obstructive pulmonary disease:

Chronic obstructive pulmonary disease (COPD) is episodic increases in respiratory symptoms, which are called exacerbations^[20]. Each that has been defined as "Chronic obstructive pulmonary disease is group of lung disease (including emphysema and chronic bronchitis) that block the airflow in the lungs". The cause of Acute Exacerbation of COPD (AECOPD) is most often infectious and related to a viral and/or bacterial infection. In acute exacerbation of copd habit of smoking and the smoke and pollution of industries are enhanced the worsening of patient in breathing^[21]. Haemophilus-influenzae is the most frequent bacterium isolated in all series followed by SARS covid -19, Streptococcus pneumoniae,

and *Moraxella catarrhalis*. Others stated that the organism commonly play pathogenic role in acute exacerbations of COPD are *Pseudomonas* and *Klebsiella*, *Acinetobacter*, *M. Catarrhalis* and *Enterobacter*. Several recent studies have reported the presence of multidrug-resistant bacteria at hospital admission in patients with severe COPD exacerbations. Non-fermenting Gram-negative bacilli, including *Pseudomonas aeruginosa*, *Acinetobacter baumannii* and *Stenotrophomonas maltophilia*, are the most frequently isolated multidrug-resistant bacteria in severe COPD exacerbations [22].

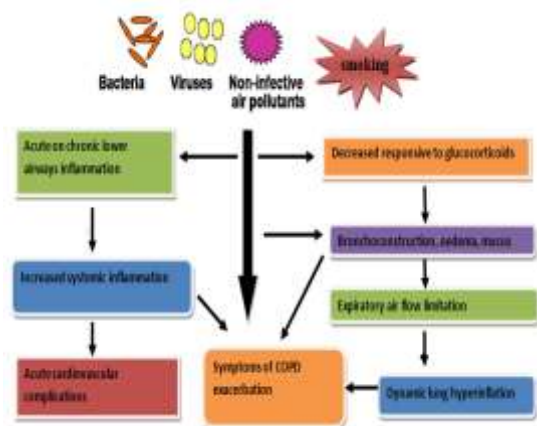


Figure-3 pathophysiology of COPD [23]

5. Obesity :

Obesity is a complex disease involving an excessive amount of body fat. Obesity isn't just a cosmetic concern. It is a medical problem that increases your risk of other diseases and health problems, such as heart disease, diabetes, high blood pressure and certain cancers [24]. In modern life style the rather than healthy and nutritious diet, people are go for the junk food which is having the lots of fat and it is unhealthy due to that children are also obese nowadays [25].

Relationship of Lifestyle disease and bacteria/virus

Lifestyle disease and bacteria/viruses relationship are not described directly but the hypothesis are made which proves that they are enhance the chances of life style disease. Some causative viruses and bacteria which are directly or indirectly caused the life style are depicted in the figure 4

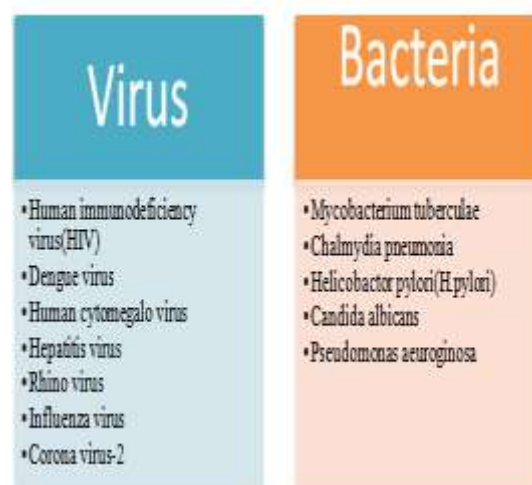


Figure -4 causative micro-organism for life style disease [26]

1. Human immunodeficiency virus:

HIV (human immunodeficiency virus) causes Acquired Immuno Deficiency Syndrome (AIDS). AIDS is a slow, progressive, and degenerative disease of the human immune system. HIV virus decreases the activity of helper t-cells and cytokines production that decreases the immunity which makes the person more susceptible for various other infection and diseases. HIV infection is caused by unprotected intercourse with multiple partners and the syringe which is infected by hiv patient [27]. Highly Active Anti Retroviral Therapy (HAART) is used for the treatment of AIDS, in which the combination of Nucleoside Reverse Transcriptase Inhibitors (NRTIs) and a protease or integrase inhibitor. Cardiovascular complications was caused by the virus it self or by the opportunistic infections [28]. Adipose tissue dysfunction, immune activation, and chronic inflammation could result in vascular and endothelial dysfunction, leading to atherosclerosis and acute ischemic events (fig-5). The physicians and patients have to take into account a higher risk of CHD in HIV-infected patients, mainly when treated with HAART [29].

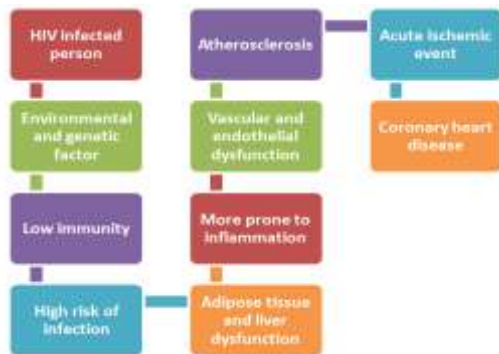


Fig-5 pathphysiology of HIV in cardiac heart disease

People with HIV infection and AIDS have a high risk of cancer as compared to normal people. HIV-infected individuals have an increased risk of Kaposi sarcoma (KS) caused by human herpesvirus 8 (HHV8), Non-Hodgkin Lymphomas (NHL), some of which are caused by Epstein-Barr virus. A sixfold increase in the risk of cervical cancer caused by oncogenic subtypes of Human Papilloma Virus (HPV) [30]. There may be direct effects of HIV infection such as insertional mutagenesis, upregulation of oncogenes, chronic antigenic stimulation or cytokine dysregulation (fig-6) [31]

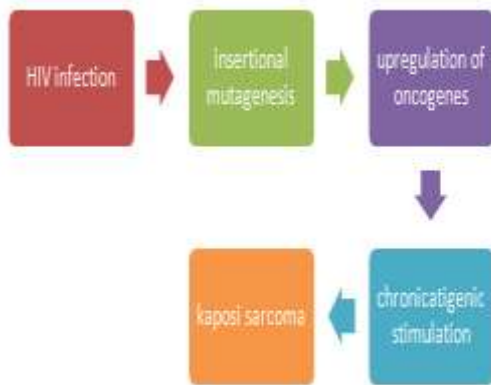


Fig - 6: Kaposi sarcoma by HIV infection

Hepatitis virus:

Correlation with the Hepatitis virus and coronary heart disease remains unclear. Still, Hepatitis-C Virus (HCV) infected subjects were at a significantly higher risk of developing CHD in comparison with HCV-uninfected subjects [32]. The markers of coronary heart disease by hepatitis are high C-reactive protein (CRP) rates as a marker of inflammation, the most significant relative risk factor for coronary artery disease [33] and differential level of cytokines, which are markers of

inflammation, thrombosis, and endothelial dysfunction; behavioral and social risk profile; malnutrition and/or inflammation pathway activation; or liver injury. Combination of these factors acts in a favorable risk profile and increases the overall risk of CAD [34]

Individuals with type II diabetes have an increased prevalence of cirrhosis, and a proportion of patients with acute and chronic liver disease develop diabetes mellitus, patients with various forms of liver disease can be predisposed to impaired glucose tolerance because of corticosteroid and hydrochlorothiazide therapy or hemochromatosis. In addition to hepatitis C virus (HCV) infection may also contribute to the development of diabetes. For example, glucose intolerance is observed more often in patients with HCV infection compared with controls with liver disease [35]. The present findings suggest that the association between HCV infection and type 2 diabetes may be stronger for persons defined as high risk on the basis of their age and BMI [36]. Hypothesized mechanism is that the virus might directly damage insulin-secreting cells. HCV may be present in human pancreatic B-cells and demonstrates that islet cells from HCV-positive patients have morphological and functional defects [37]

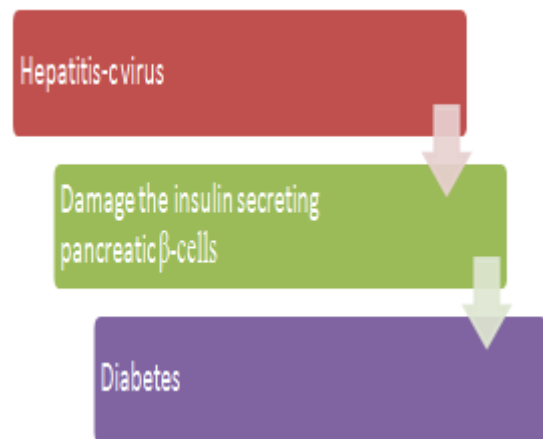


Fig -7: Hypothetical mechanism of diabetes by hepatitis C virus

Hepatocellular carcinoma (HCC) are associated with cirrhosis related to chronic hepatitis B virus (HBV) or hepatitis C virus (HCV) infection. Environmental, host genetic, and viral factors can affect the risk of HCC in individuals with HBV or HCV infection. The risk of HCC is increased in patients with higher levels of HBV replication, determined by tests for HBeAg and levels of HBV DNA [38].

3. Human cytomegalovirus :

Human cytomegalovirus (HCMV) is a herpes virus. HCMV is related to accelerated Atherosclerosis(AS) and the development of Ischemic Heart Disease (IHD) among recipients of heart transplants^[39]. The biological characteristics of HCMV are consistent with the pathogenesis of AS; systemic HCMV infection leads to sub-clinical inflammation; and HCMV infects epithelial cells(EC), leading to cellular injury and metabolic changes^[40]

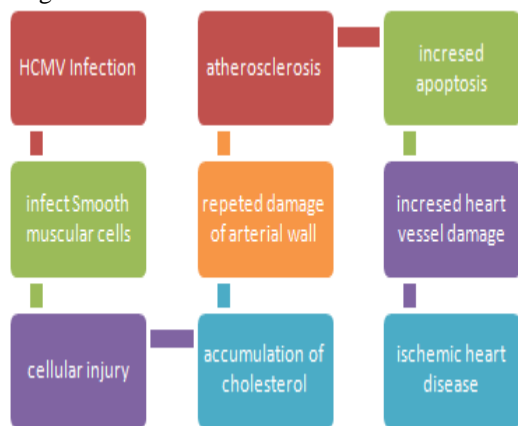


Figure -8: mechanism of HCMV for atherosclerosis and Ischemic Heart Disease

4. Dengue virus:

Aedes aegypti is the major urban vector of dengue viruses worldwide. The dengue virus (DENV), a member of the genus *Flavivirus* in the family *Flaviviridae*, is a single-stranded enveloped RNA virus, of which four distinct, but related, serotypes exist (DENV1-4). *Aedes aegypti* Mosquitoes, a host for chikungunya, zika fever, mayaro, and yellow fever^[41]. The spectrum of cardiovascular manifestations in dengue is broad, ranging from myocardial impairment and arrhythmias to vascular barrier dysfunction causing plasma leakage and hemodynamic compromise. Myocardial impairment can contribute to haemodynamic instability during the critical phase of capillary leakage.

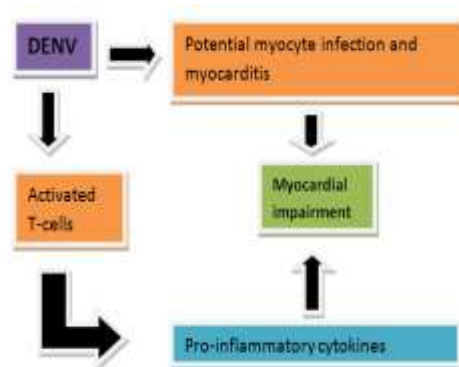


Fig:9 Mechanisms involved in the cardiac and vascular manifestations of dengue.

The capillary leak in DENV infection is slow and persistent, contrary to the leak associated with bacterial septic shock, which is sudden and rapid and leads to cardiovascular collapse within hours. Slow leakage over several days, such that upregulation of homeostatic compensatory mechanisms can take place^[42]

5. Influenza:

The disease burden of influenza is particularly heavy in the elders. The impact of smoking on mortality risks specifically associated with influenza, despite the fact that the increased mortality risk of pneumonia and influenza among smokers has been widely reported. It is necessary to encourage the elders to adopt other prevention approaches such as improving personal hygiene and maintaining healthy life style that can be enhanced before or at the very early stage of pandemics^[43]. Non-encapsulated Haemophilus influenzae often causes chronic infections of the lower respiratory tract in both non-obstructive and obstructive chronic bronchitis^[44]. The H. Influenza strain may persist in the respiratory tract in the presence of negative sputum cultures. Elucidating the mechanism of this colonization pattern of H. Influenza has important implications in understanding the potential role of H. Influenza in the chronic airway inflammation observed in COPD. The H. Influenza in causing exacerbations of COPD, characterizing the host response to H. Influenza in the respiratory tract, and interpreting the results of sputum cultures from adults with COPD^[45]. The incubation of cultured human bronchial epithelial cells with endotoxin from NTHi leads to markedly increased expression and release of pro-inflammatory mediators, including IL-6, IL-8, and TNF- α . Together, these findings suggest that persistent or repetitive exposure of the airway

to NTHi products may contribute to airway inflammation in COPD^[46].

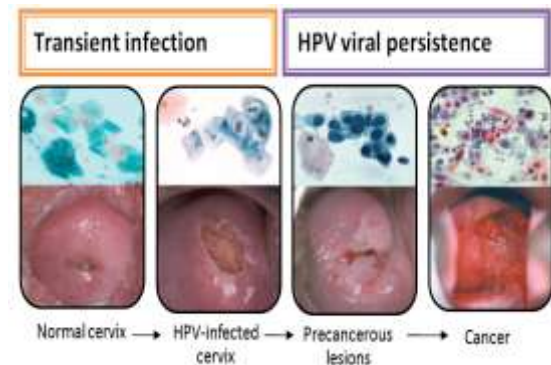
Estimates the risk of influenza A(H1N1)p infection in persons with diabetes would complement estimates of the risk of hospitalization and ICU care after the illness. Risk estimates for seasonal influenza could strengthen the basis for recommendations that persons with diabetes be regularly immunized against influenza^[47]

6. Enterovirus:

Enteroviruses are one of the primary candidates because traces of this viral infection have been found more frequently in patients with Type-1 Diabetes than in individuals without diabetes^[48]. Higher rates of enterovirus infection, defined by detection of enterovirus IgM or IgG, or both, viral RNA with reverse transcription polymerase chain reaction (RT PCR), and viral capsid protein, have been found in patients with diabetes at diagnosis compared with controls. Based on the hypothesis that enterovirus infection increases the risk of pancreatic islet autoimmunity or type 1 diabetes, or both^[49]

7 Human papilloma virus:

Cervical cancer is the second most common cancer in women worldwide, and knowledge regarding its cause and pathogenesis is expanding rapidly. There are four major steps in cervical cancer development: infection of metaplastic epithelium at the cervical transformation zone, viral persistence, progression of persistently infected epithelium to cervical precancer, and invasion through the basement membrane of the epithelium. Infection with a carcinogenic HPV is a necessary cause of both squamous cell carcinoma and adenocarcinoma. HPV16 and HPV18 are the two most carcinogenic HPV types, and are responsible for 70% of cervical cancer and about 50% of cervical intraepithelial neoplasia (CIN) grade 3 (CIN3); by contrast, HPV6 and HPV11 are responsible for about 90% of genital warts^[50]



(figure 10: Development of cervical cancer:).

HPV16 and related types are most likely to produce high-grade squamous intraepithelial lesions; by contrast HPV18 (the second most common type in cancers) causes a disproportionately low fraction of such lesions. In addition to cancer of the cervix, a major proportion of anal, perianal, vulvar, and penile cancers appears to be linked to the same HPV infections. Recent evidence also points to a possible role of other HPV infections in squamous cell carcinomas of the skin^[51]. Viral persistence leads to clonal progression of the persistently-infected epithelium and cervical intraepithelial neoplasia (CIN)-3/precancers arise; events which remain unknown lead infected cells to cervical invasion^[52]

8 Epstein-Barr Virus :

Epstein-Barr virus (EBV) was the first human virus to implicate in carcinogenesis. It infects >90% of the world's population. Although most humans coexist with the virus without serious sequelae, a small proportion will develop tumors. EBV has been implicated in the pathogenesis of Burkitt's lymphoma, Hodgkin's disease, non-Hodgkin's lymphoma, nasopharyngeal carcinoma, and lymphomas, as well as leiomyosarcomas arising in immune-compromised individuals. The presence of this virus has also been associated with epithelial malignancies arising in the gastric region and the breast, although some of this work remains indisputable. EBV uses its viral proteins, the actions of which mimic several growth factors, transcription factors, and antiapoptotic factors, to usurp control of the cellular pathways that regulate diverse homeostatic cellular functions. Recent advances in antiviral therapeutics, application of monoclonal antibodies, and generation of EBV-specific CTLs are beginning to show promise in treating EBV-related disorders^[53]

9. Human rhinovirus:

Viral infections are associated with more severe exacerbations in terms of symptoms, resulting in longer recovery times and a greater likelihood of hospitalization. Human rhinovirus (HRV) is one of the causes of the common cold. It is the major viral pathogen detected in COPD exacerbation identified in 60% of the virus using quantitative PCR (qPCR)^[54]. It is shown that experimental HRV infection triggers COPD exacerbations, although these episodes were mild events that did not require increased systemic therapy. COPD exacerbations are complex events that can last for prolonged periods. There is little information on the course of HRV infection during and after naturally occurring COPD exacerbations. Information from HRV presence and load during the onset and recovery of exacerbation may allow appropriate targeting of therapeutic interventions, and thus help reduce exacerbation severity^[55]. A proposed mechanism of increased viral susceptibility is intracellular adhesion molecule (ICAM)-1 on respiratory epithelial cells. ICAM-1 showed upregulation in the bronchial mucosa of patients with chronic bronchitis leading to increases in HRV infection in these patients^[56]

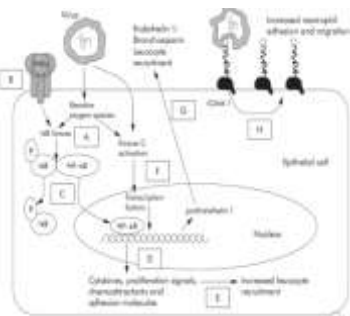


Figure 11 Key inflammatory effects of viral infection^[57]

1. Mycobacterium tuberculosis:

Mycobacterium tuberculosis can persist within the human host for years without causing disease, in a syndrome known as latent tuberculosis (TB). As one-third of the world population has latent TB, placing them at risk for active TB, the mechanisms by which M. tuberculosis establishes a latent metabolic state, eludes immune surveillance and responds to triggers that stimulate reactivation are a high priority for the future control of TB^[58]. The burden of tuberculosis and cardiovascular disease (CVD) is enormous worldwide and rapidly increasing in low- and middle-income countries.

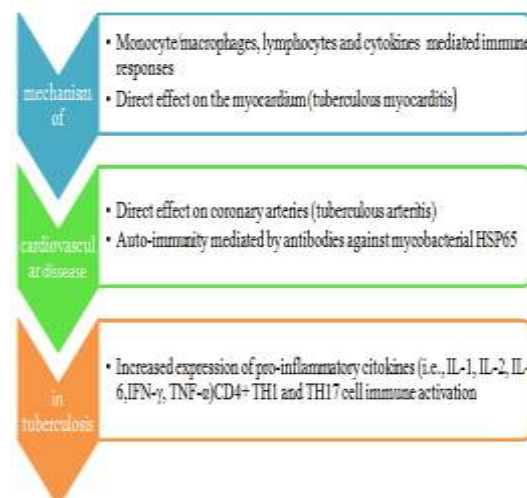


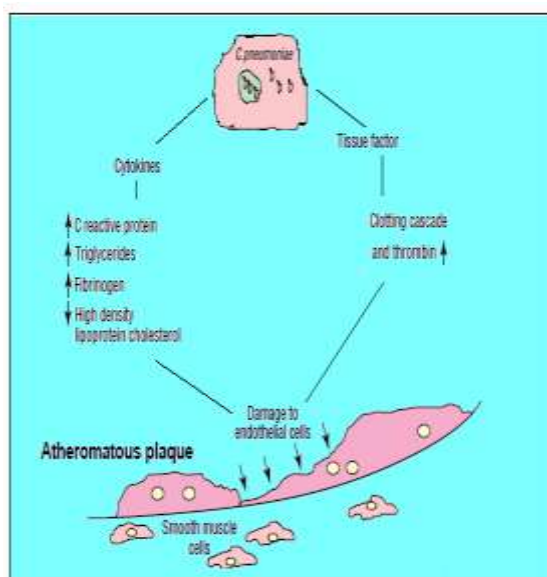
Figure – 12 Possible Mechanism of cardiovascular disease in TB

According to Studies, a pro-atherogenic effect of antibody-mediated responses against mycobacterial heat shock protein-65 through cross-reaction with self-antigens in human vessels. Furthermore, subsets of mycobacteria actively replicate during latent tuberculosis infection (LTBI), and recent studies suggest that LTBI is associated with persistent chronic inflammation that may lead to CVD.^[59]

Diabetes increases the risk of tuberculosis incidence and the risk of adverse treatment outcomes in patients with tuberculosis^[60]. Many studies have explored the relationship between DM and TB, including a recent systematic review, which showed that the risk of TB among people with DM is three times higher than in people without DM^[61]. Patients with DM were four times more likely to develop relapse of TB disease than patients without DM. These individuals were considered cured or treatment complete but the patients could have relapsed through two possible routes which are as mention: 1. They may have been fixed but experienced a recurrence of the former infection 2. They may have been re-infected with a new strain of TB.^[62]

2. Chlamydia pneumoniae:

Chlamydia pneumoniae infection participates in the development of CHD, and there are several mechanisms by which it is associated with CHD risk factors. Chronic conditions can lead to elevated levels of C-Reactive Protein, leukocytes, and several cytokines, all associated with arteriosclerosis (figure-13).^[63]



(Figure:13 atherosclerosis formation by chlamydia pneumonia)^[64]

Pathogenetic mechanisms by which *C.pneumoniae* infection could affect the development of atherosclerosis and coronary heart disease. A high C-reactive protein (CRP) rates as a marker of inflammation—the most significant relative risk factor for coronary artery disease. Pathogenetic mechanisms for the generation of atherogenesis, thrombosis, and plaque rupture are as follow:

Chlamydia pneumoniae is an intracellular bacterium associated with acute respiratory diseases and tends to cause chronic infections. Infection with *Chlamydia pneumoniae* could lead to the stimulation of a continuous inflammatory response resulting in an increased production of IL-6 and essential fibroblast growth factor (bFGF). This increased inflammatory factors could contribute to subepithelial fibrosis in small airways analogous to the scarring observed in chronic *Chlamydia trachomatis* infection of the eye (trachoma) or genital tract (tubal infertility). Thus, *Chlamydia pneumoniae* theoretically can cause tissue remodeling and “the disease of the small airways” seen in COPD. This could trigger the development of COPD by initiating the release of cytokines and chemokines and thereby sustaining an inflammatory response. *Chlamydia pneumoniae* (Cpn) is an established cause of acute and chronic upper and lower respiratory tract infections^[65]. The more substantial prevalence recorded in our patient group could be due to either chronic disease by *Chlamydia pneumoniae*, as suggested by the

increase of specific Ig-G prevalence and geometric mean titer with age, or to a higher rate of acute infection in such patients^[66]

3. *Helicobacter pylori*:

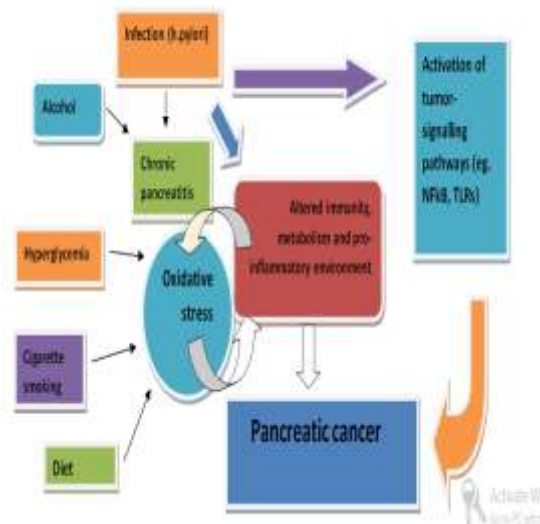
H. pylori the typical bacterial pathogen found worldwide that attaches to mucus-secreting cells in the gastric mucosa and initiates inflammation leading to gastritis and peptic ulcer. The sources of the *h.pylori* is drinking contaminated water, people have to be taken the care of hygiene and diet^[67]. A relationship may exist between *H. pylori* infection and atherosclerotic vascular disease. *H. pylori*-specific IgG serum levels among CAD patients. *H. pylori* infection seems to link the presence of CAD through the ability of modification of serum lipids and induction of inflammation^[68]. Monocytes and macrophages have long components of atheromatous plaques. Elevated levels of the acute phase proteins, fibrinogen, C-reactive protein (CRP), and pro-inflammatory cytokines are associated with an increased risk of cardiovascular events. The possibility that an undetected chronic infection may be behind these changes in inflammatory markers is an attractive hypothesis and has led to the spotlight falling on microorganisms, which is known to be commonly detectable in asymptomatic individuals.^[69]

Prevalence of *H. pylori* infection was significantly higher in Type-2 Diabetes Mellitus obese subjects than non-diabetic subjects^[70]. The mechanism by which *H. pylori* infection increases the risk of diabetes remains to be elucidated but may involve inflammation or dyspepsia. Infection with *H. pylori* was found in previous studies to be correlated with elevated levels of CRP, IL-6, and tumor necrosis factor- α (TNF α), which are markers of inflammation implicated in insulin resistance and development of diabetes. Furthermore, the presence of Gram-negative bacteria, such as *H. pylori*, in the gut microbiota leads to increased production of lipopolysaccharide, which also activates innate inflammatory processes^[71]

H. pylori were designated as a class I carcinogen by the World Health Organization (WHO) in 1994. Two different cancers are associated with *H. pylori* infection—gastric lymphoma and adenocarcinoma.

The most likely mechanism includes oxidative DNA damage that eventually escapes repair within the host cell. In addition, oxidative stress regulates the expression of several genes that govern epithelial cell-turnover, which is consistent with the increased rate of malignancy associated with other

forms of chronic inflammatory disease in the digestive tract, including celiac disease and ulcerative colitis^[72]



(figure:14 Role of bacterial infections in pancreatic cancer^[73]

4.candidiasis:

Candidiasis is an infection caused by a yeast (a type of fungus) called Candida. Candida normally lives inside the body (in places such as the mouth, throat, gut, and vagina) and on skin without causing any problems. Personal hygiene is necessary for women and diabetic person^[74] Pseudomembranous candidiasis (thrush) is characterized by extensive white pseudo membranes consisting of desquamated epithelial cells, fibrin, and fungal hyphae. Oral candidiasis is an opportunistic infection of the oral cavity and can also be a mark of systemic disease, such as diabetes mellitus which is a common problem among the immune compromised. Oral candidiasis is caused by an overgrowth or infection of the oral cavity by a yeast-like fungus, candida^[75]. Vulvovaginal candidiasis affects about 75% of all adult women at some stage in their lives. Several related factors, including diabetes mellitus, have been identified. Uncontrolled diabetes with glycosuria and increased glucose concentrations in vaginal secretions may precipitate symptomatic vaginitis presumed to be due to colonization with *Candida albicans*^[76]

5. Pseudomonas aeruginosa:

Pseudomonas aeruginosa is an opportunistic human pathogen associated with a wide range of 27 infections affecting, among others, skin, ear, eye, urinary tract, heart, airway

and lung tissues. Self medication promotes the drug resistance in the *pseudomonas aeruginosa* strain^[77]. *Pseudomonas Aeruginosa* may cause chronic infections in patients with COPD that are similar to those seen in patients with Cystic fibrosis. COPD isolates generally showed an increased mutation rate, increased antibiotic resistance, reduced production of proteases, less cytotoxicity, less motility, and greater biofilm production in in-vitro assay^[78]. Exacerbations caused by *P. Aeruginosa* are more likely to be seen in patients with more-advanced COPD, those who have received recent antibiotic therapy, and those who require mechanical ventilation for an exacerbation^[79]. An unusual ‘mucoid’ phenotype of *P. Aeruginosa* chronically infects approximately 70–80% of adolescents and adults with cystic fibrosis. The high levels of elastase produced by this pathogen damage the lungs and have a cumulative, deleterious effect on pulmonary function over years or even decades, resulting in death. Mucoid phenotypes of *P. Aeruginosa* are occasionally seen causing pulmonary infections in individuals with another chronic lung disease^[80]

5. Moraxella catarrhalis:

Moraxella (Branhamella) catarrhalis is a gram-negative, aerobic diplococcus frequently found as a commensal of the upper respiratory tract. *M. catarrhalis* is an essential cause of lower respiratory tract infections, particularly in adults with chronic obstructive pulmonary disease (COPD)^[81]. Cigarette smoking can lead to worsening the condition of the patient^[82]. *M. catarrhalis* induces activation of the mitogen-activated protein kinase and nuclear factor- κ B signalling systems in bronchial epithelial cells, with the release of interleukin-8 and granulocyte-macrophage colony-stimulating element from the cells. Three serotypes of *M. Catarrhalis* have been identified based on structural differences in lipooligosaccharide^[83]. Serotype A is the predominant type among clinical isolates. The distribution of serotypes appears to differ by patients age, with isolates from adults having a somewhat more significant proportion of serotype A, compared with those from children. Lipooligosaccharide is likely a vital inducer of the host inflammatory response^[84]

6. Gram-positive/negative bacteria:

A broad range of gram-positive/negative bacteria cause serious infections in the cancer patient with the greatest burden of disease being due

to staphylococci, streptococci, enterococci and in *Escherichia coli*, *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Stenotrophomonas maltophilia*.^[85] The evolution of cancer therapy and the changing epidemiology of major gram-positive/negative pathogens mean that ongoing efforts are needed to understand and mitigate the impact of these bacteria in patients with malignancy. Among cancer patients, GBS predominantly affects those with breast cancer in which recurrent bouts of postsurgical cellulitis are problematic. Malignancy is also a risk factor for invasive disease due to *S. pneumoniae*, with persons having active leukemia, lymphoma, or myeloma, or those having undergone stem cell transplantation having the highest incidence.

Identified risk factors predisposing cancer patients to enterococcal infections have included nosocomial infection onset, prior antibiotic exposure, prolonged neutropenia, and stem cell transplantation.^[86] Effects of bacterial infection that contribute to carcinogenesis: (1) Cancer-associated bacteria provoke chronic inflammatory responses, (2) directly manipulate host cell biology (3) and might alter tissue stem cell homeostasis. The overlap of these effects in the correct cellular context might promote the accumulation of genetic defects that result in the emergence of malignant cells.^[87]

Conclusion : Life style diseases are sometimes curable because if they are treated in the beginning stage. Life style diseases are showing the result of unhealthy diet and bad habits. Life style diseases differ by age and gender also the daily routine and eating pattern. Chronic life style diseases like cardiovascular disease, diabetes, cancer and chronic lung disease are not curable but they can be maintained by taking care of health and diet. Obesity is also attracting several diseases like hyperlipidemia and high glucose levels so by losing weight it is also treated or maintained. Life style diseases are the outcome of the health of a person so healthy food and physical activity are needed in every person's daily routine for living a good quality life.

REFERENCES:

- [1]. Vallgård S. Why the concept "lifestyle diseases" should be avoided. *Scandinavian journal of public health*. 2011 Nov;39(7):773-5. doi:10.1177/1403494811421978
- [2]. Tabish SA (2017) Lifestyle Diseases: Consequences, Characteristics, Causes and Control. *J Cardiol Curr Res* 9(3): 00326. DOI: 10.15406/jccr.2017.09.00326
- [3]. Sharma M, Majumdar PK. Occupational lifestyle diseases: An emerging issue. *Indian journal of occupational and environmental medicine*. 2009 Dec;13(3):109.
- [4]. Gluckman P, Hanson M. *Mismatch: The lifestyle diseases timebomb*. Oxford University Press on Demand; 2008 Feb 14.
- [5]. Pappachan MJ. Increasing prevalence of lifestyle diseases: high time for action. *The Indian journal of medical research*. 2011 Aug;134(2):143.
- [6]. Chakma JK, Gupta S. Lifestyle practice and associated risk factors of non-communicable diseases among the students of Delhi University. *International Journal of Health & Allied Sciences*. 2017 Jan 1;6(1):20.
- [7]. Boutayeb A, Boutayeb S. The burden of non-communicable diseases in developing countries. *International journal for equity in health*. 2005 Dec 1;4(1):2
- [8]. **Camp to Screen Most Common Non Communicable (lifestyle) disease by HT team**
from: <https://www.techplusmedia.com/health/2016/07/13/camp-to-screen-most-common-non-communicable-lifestyle-disease/>
- [9]. Lucas A, Murray E, Kinra S. Health beliefs of UK South Asians related to lifestyle diseases: a review of qualitative literature. *Journal of obesity*. 2013 Jan 1;2013.
- [10]. Tabish SA. Lifestyle diseases: consequences, characteristics, causes and control. *Journal of Cardiology & Current Research*. 2017 Jul;9(3):1-4.
- [11]. Cardiovascular disease by world health organisation
from: https://www.who.int/health-topics/cardiovascular-diseases#tab=tab_1
- [12]. Shrivastava AK, Singh HV, Raizada A, Singh SK. C-reactive protein, inflammation and coronary heart disease. *The Egyptian Heart Journal*. 2015 Jun 1;67(2):89-97.
- [13]. Roger Walker and Catherine Whittlesea - *Clinical pharmacy and therapeutics*. Fifth edition. Edinburgh: Churchill Livingstone. 2007.
- [14]. Ellis RW. Infection and coronary heart disease. *Journal of Medical Microbiology*. 1997 Jul 1;46(7):535-9.

- [15]. Arcangelo VP, Peterson AM, editors. *Pharmacotherapeutics for advanced practice: a practical approach*. Lippincott Williams & Wilkins; 2006.
- [16]. WHO.diabetes.available from <https://www.who.int/diabetes/en/>
- [17]. Saha S, Gerdtham UG, Johansson P. Economic evaluation of lifestyle interventions for preventing diabetes and cardiovascular diseases. *International journal of environmental research and public health*. 2010 Aug;7(8):3150-95.
- [18]. Jemal A, Bray F, Center MM, Ferlay J, Ward E, Forman D. Global cancer statistics. *CA: a cancer journal for clinicians*. 2011 Mar;61(2):69-90.
- [19]. WHO.cancer. available from https://www.who.int/health-topics/cancer#tab=tab_1
- [20]. Perera WR, Hurst JR, Wilkinson TM, Sapsford RJ, Müllerova H, Donaldson GC, Wedzicha JA. Inflammatory changes, recovery and recurrence at COPD exacerbation. *European Respiratory Journal*. 2007 Mar 1;29(3):527-34.
- [21]. Rodriguez-Roisin R. Toward a consensus definition for COPD exacerbations. *Chest*. 2000 May 1;117(5):398S-401S.
- [22]. Gram negative organisms as a cause of acute exacerbation of COPD:ElKorashy RI, El-Sherif RH. Gram negative organisms as a cause of acute exacerbation of COPD. *Egyptian Journal of Chest Diseases and Tuberculosis*. 2014 Apr 1;63(2):345-9.
- [23]. COPD immunopathology:Caramori G, Casolari P, Barczyk A, Durham AL, Di Stefano A, Adcock I. COPD immunopathology. In *Seminars in immunopathology 2016 Jul 1 (Vol. 38, No. 4, pp. 497-515)*. Springer Berlin Heidelberg.)
- [24]. Hu F. *Obesity epidemiology*. Oxford University Press; 2008 Mar 21
- [25]. James PT, Leach R, Kalamara E, Shayeghi M. The worldwide obesity epidemic. *Obesity research*. 2001 Nov;9(S11):228S-33S
- [26]. Ehlers S, Kaufmann SH. Infection, inflammation, and chronic diseases: consequences of a modern lifestyle. *Trends in Immunology*. 2010 May 1;31(5):184-90.
- [27]. Centers for Disease Control (US), Center for Infectious Diseases (US). Division of HIV/AIDS., National Center for Infectious Diseases (US) Division of HIV/AIDS.. HIV/AIDS surveillance. US Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Infectious Diseases, Division of HIV/AIDS; 1990.
- [28]. Sani MU, Okeahialam BN, Aliyu SH, Enoch DA. Human immunodeficiency virus (HIV) related heart disease: a review. *Wiener KlinischeWochenschrift*. 2005 Feb 1;117(3):73-81.
- [29]. Boccara F, Lang S, Meuleman C, Ederhy S, Mary-Krause M, Costagliola D, Capeau J, Cohen A. HIV and coronary heart disease: time for a better understanding. *Journal of the American College of Cardiology*. 2013 Feb 5;61(5):511-23.
- [30]. Cancer Burden in the HIV-Infected Population in the United States:Shiels MS, Pfeiffer RM, Gail MH, Hall HI, Li J, Chaturvedi AK, Bhatia K, Uldrick TS, Yarchoan R, Goedert JJ, Engels EA. Cancer burden in the HIV-infected population in the United States. *Journal of the National Cancer Institute*. 2011 May 4;103(9):753-62.
- [31]. Silverberg MJ, Lau B, Justice AC, Engels E, Gill MJ, Goedert JJ, Kirk GD, D'Souza G, Bosch RJ, Brooks JT, Napravnik S. Risk of anal cancer in HIV-infected and HIV-uninfected individuals in North America. *Clinical infectious diseases*. 2012 Apr 1;54(7):1026-34.
- [32]. Butt AA, Xiaoqiang W, Budoff M, Leaf D, Kuller LH, Justice AC. Hepatitis C virus infection and the risk of coronary disease. *Clinical Infectious Diseases*. 2009 Jul 15;49(2):225-32.
- [33]. [33]Ishizaka N, Ishizaka Y, Takahashi E, Toda EI, Hashimoto H, Ohno M, Nagai R, Yamakado M. Increased prevalence of carotid atherosclerosis in hepatitis B virus carriers. *Circulation*. 2002 Mar 5;105(9):1028-30.
- [34]. Butt AA, Xiaoqiang W, Budoff M, Leaf D, Kuller LH, Justice AC. Hepatitis C virus infection and the risk of coronary disease. *Clinical Infectious Diseases*. 2009 Jul 15;49(2):225-32.
- [35]. Mason AL, Lau JY, Hoang N, Qian K, Alexander GJ, Xu L, Guo L, Jacob S, Regenstein FG, Zimmerman R, Everhart JE. Association of diabetes mellitus and chronic hepatitis C virus infection. *Hepatology*. 1999 Feb;29(2):328-33.

- [36]. Mehta SH, Brancati FL, Strathdee SA, Pankow JS, Netski D, Coresh J, Szklo M, Thomas DL. Hepatitis C virus infection and incident type 2 diabetes. *Hepatology*. 2003 Jul 1;38(1):50-6.
- [37]. Masini M, Campani D, Boggi U, Menicagli M, Funel N, Pollera M, Lupi R, Del Guerra S, Bugliani M, Torri S, Del Prato S. Hepatitis C virus infection and human pancreatic β -cell dysfunction. *Diabetes care*. 2005 Apr 1;28(4):940-1.
- [38]. Kuper H, Adami HO, Trichopoulos D. Infections as a major preventable cause of human cancer. *Journal of internal medicine*. 2001 Feb;249(S741):61-74.
- [39]. Vercellotti GM. Overview of infections and cardiovascular diseases. *Journal of allergy and clinical immunology*. 2001 Oct 1;108(4):S117-20.
- [40]. Tuberculosis and cardiovascular disease: linking the epidemics: Huaman MA, Henson D, Ticona E, Sterling TR, Garvy BA. Tuberculosis and cardiovascular disease: linking the epidemics. *Tropical diseases, travel medicine and vaccines*. 2015 Dec;1(1):1-7.
- [41]. Jansen CC, Beebe NW. The dengue vector *Aedes aegypti*: what comes next. *Microbes and infection*. 2010 Apr 1;12(4):272-9.
- [42]. Yacoub S, Wertheim H, Simmons CP, Sreaton G, Wills B. Cardiovascular manifestations of the emerging dengue pandemic. *Nature reviews cardiology*. 2014 Jun;11(6):335.
- [43]. Wong CM, Yang L, Chan KP, Chan WM, Song L, Lai HK, Thach TQ, Ho LM, Chan KH, Lam TH, Peiris JS. Cigarette smoking as a risk factor for influenza-associated mortality: evidence from an elderly cohort. *Influenza and other respiratory viruses*. 2013 Jul;7(4):531-9.
- [44]. BRESSER P, OUT TA, van ALPHEN LO, JANSEN HM, LUTTER R. Airway inflammation in nonobstructive and obstructive chronic bronchitis with chronic *Haemophilus influenzae* airway infection: comparison with noninfected patients with chronic obstructive pulmonary disease. *American journal of respiratory and critical care medicine*. 2000 Sep 1;162(3):947-52.
- [45]. Murphy TF, Brauer AL, Schiffmacher AT, Sethi S. Persistent colonization by *Haemophilus influenzae* in chronic obstructive pulmonary disease. *American journal of respiratory and critical care medicine*. 2004 Aug 1;170(3):266-72.
- [46]. Moghaddam SJ, Clement CG, De la Garza MM, Zou X, Travis EL, Young HW, Evans CM, Tuvim MJ, Dickey BF. *Haemophilus influenzae* Lysate Induces Aspects of the Chronic Obstructive Pulmonary Disease Phenotype. *American journal of respiratory cell and molecular biology*. 2008 Jun;38(6):629-38.
- [47]. Allard R, Leclerc P, Tremblay C, Tannenbaum TN. Diabetes and the severity of pandemic influenza A (H1N1) infection. *Diabetes care*. 2010 Jul 1;33(7):1491-3.
- [48]. Rodriguez-Calvo T, von Herrath MG. Enterovirus infection and type 1 diabetes: closing in on a link?. *Diabetes*. 2015 May 1;64(5):1503-5.
- [49]. Yeung WC, Rawlinson WD, Craig ME. Enterovirus infection and type 1 diabetes mellitus: systematic review and meta-analysis of observational molecular studies. *Bmj*. 2011 Feb 3;342:d35.
- [50]. Mark Schiffman, Philip E Castle, Jose Jeronimo, Ana C Rodriguez, Sholom Wacholder: Schiffman M, Castle PE, Jeronimo J, Rodriguez AC, Wacholder S. Human papillomavirus and cervical cancer. *The Lancet*. 2007 Sep 8;370(9590):890-907.
- [51]. *J Natl Cancer Inst* 2000;92:690-8. Papillomaviruses causing cancer: evasion from host-cell control in early events in carcinogenesis. *Journal of the National Cancer Institute*. 2000 May 3;92(9):690-8.
- [52]. Moscicki AB, Schiffman M, Kjaer S, Villa LL. Chapter 5: updating the natural history of HPV and anogenital cancer. *Vaccine* 24 (Suppl): S3, S42-S51.
- [53]. Thompson MP, Kurzrock R. Epstein-Barr virus and cancer. *Clinical Cancer Research*. 2004 Feb 1;10(3):803-21. (Glenn WK, Heng B, Delprado W, Iacopetta B, Whitaker NJ. Epstein-Barr Virus. *Human Papillomavirus and Mouse Mammary Tumour Virus* as. 2012.) (Glaser SL, Hsu JL, Gulley ML. Epstein-Barr virus and breast cancer: state of the evidence for viral carcinogenesis. *Cancer Epidemiology and Prevention Biomarkers*. 2004 May 1;13(5):688-97.
- [54]. Wong CM, Yang L, Chan KP, Chan WM, Song L, Lai HK, Thach TQ, Ho LM, Chan KH, Lam TH, Peiris JS. Cigarette smoking as a risk factor for influenza-associated

- mortality: evidence from an elderly cohort. *Influenza and other respiratory viruses*. 2013 Jul;7(4):531-9.
- [55]. George SN, Garcha DS, Mackay AJ, Patel AR, Singh R, Sapsford RJ, Donaldson GC, Wedzicha JA. Human rhinovirus infection during naturally occurring COPD exacerbations. *European Respiratory Journal*. 2014 Jul 1;44(1):87-96.
- [56]. George SN, Garcha DS, Mackay AJ, Patel AR, Singh R, Sapsford RJ, Donaldson GC, Wedzicha JA. Human rhinovirus infection during naturally occurring COPD exacerbations. *European Respiratory Journal*. 2014 Jul 1;44(1):87-96.
- [57]. Caramori G, Casolari P, Barczyk A, Durham AL, Di Stefano A, Adcock I. COPD immunopathology. *In Seminars in immunopathology 2016 Jul 1 (Vol. 38, No. 4, pp. 497-515)*. Springer Berlin Heidelberg.
- [58]. Pavan FR, Leite CQ. What is "Mycobacterium tuberculosis"? *Tuberculosis*. 2009 Jul;27:28.
- [59]. Human MA, Henson D, Ticona E, Sterling TR, Garvy BA. Tuberculosis and cardiovascular disease: linking the epidemics. *Tropical diseases, travel medicine and vaccines*. 2015 Dec;1(1):1-7.
- [60]. Pan SC, Ku CC, Kao D, Ezzati M, Fang CT, Lin HH. Effect of diabetes on tuberculosis control in 13 countries with high tuberculosis: a modelling study. *The lancet Diabetes & endocrinology*. 2015 May 1;3(5):323-30.
- [61]. Jiménez-Corona ME, Cruz-Hervert LP, García-García L, Ferreyra-Reyes L, Delgado-Sánchez G, Bobadilla-del-Valle M, Canizales-Quintero S, Ferreira-Guerrero E, Báez-Saldaña R, Téllez-Vázquez N, Montero-Campos R. Association of diabetes and tuberculosis: impact on treatment and post-treatment outcomes. *Thorax*. 2013 Mar 1;68(3):214-20.
- [62]. Baker MA, Harries AD, Jeon CY, Hart JE, Kapur A, Lönnroth K, Ottmani SE, Goonesekera SD, Murray MB. The impact of diabetes on tuberculosis treatment outcomes: a systematic review. *BMC medicine*. 2011 Dec 1;9(1):81.
- [63]. Leinonen M. Chlamydia pneumoniae and other risk factors for atherosclerosis. *The Journal of infectious diseases*. 2000 Jun 1;181(Supplement_3):S414-6.
- [64]. Gupta S, Camm AJ. Chlamydia pneumoniae and coronary heart disease: coincidence, association, or causation
- [65]. Brandén E, Koyi H, Gnarpe J, Gnarpe H, Tornling G. Chronic Chlamydia pneumoniae infection is a risk factor for the development of COPD. *Respiratory medicine*. 2005 Jan 1;99(1):20-6.
- [66]. Blasi F, Legnani D, Lombardo VM, Negretto GG, Magliano E, Pozzoli R, Chiodo F, Fasoli A, Allegra L. Chlamydia pneumoniae infection in acute exacerbations of COPD. *European Respiratory Journal*. 1993 Jan 1;6(1):19-22.
- [67]. Farhadkhani M, Nikaeen M, Hassanzadeh A, Nikmanesh B. Potential transmission sources of Helicobacter pylori infection: detection of H. pylori in various environmental samples. *Journal of Environmental Health Science and Engineering*. 2019 Jun 1;17(1):129-34
- [68]. Al-Ghamdi A, Jiman-Fatani AA, El-Banna H. Role of Chlamydia pneumoniae, helicobacter pylori and cytomegalovirus in coronary artery disease. *Pak J Pharm Sci*. 2011 Apr 1;24(2):95-101.
- [69]. Kowalski M, Pawlik M, Konturek JW, Konturek SJ. Helicobacter pylori infection in coronary artery disease. *Journal of physiology and pharmacology*. 2006 Sep 1;57:101.)
- [70]. Bener A, Micallef R, Afifi M, Derbala M, Al-Mulla HM, Usmani MA. Association between type 2 diabetes mellitus and Helicobacter pylori infection. *Turk J Gastroenterol*. 2007 Dec 1;18(4):225-9.
- [71]. Jeon CY, Haan MN, Cheng C, Clayton ER, Mayeda ER, Miller JW, Aiello AE. Helicobacter pylori infection is associated with an increased rate of diabetes. *Diabetes care*. 2012 Mar 1;35(3):520-5.
- [72]. Ernst PB, Gold BD. The disease spectrum of Helicobacter pylori: the immunopathogenesis of gastroduodenal ulcer and gastric cancer. *Annual Reviews in Microbiology*. 2000 Oct;54(1):615-40.
- [73]. Michaud DS. Role of bacterial infections in pancreatic cancer. *Carcinogenesis*. 2013 Oct 1;34(10):2193-7.
- [74]. Domer JE, Carrow EW. Candidiasis. *In Immunology of the fungal diseases 2020 Jul 25 (pp. 57-92)*. CRC Press
- [75]. Akpan A, Morgan R. Oral candidiasis. *Postgraduate medical journal*. 2002 Aug 1;78(922):455-9.

- [76]. Rowe BR, Logan MN, Farrell I, Barnett AH. Is candidiasis the true cause of vulvovaginal irritation in women with diabetes mellitus?. *Journal of clinical pathology*. 1990 Aug 1;43(8):644-5.
- [77]. Valentini M, Gonzalez D, Mavridou DA, Filloux A. Lifestyle transitions and adaptive pathogenesis of *Pseudomonas aeruginosa*. *Current opinion in microbiology*. 2018 Feb 1;41:15-20
- [78]. Parameswaran GI, Sethi S. *Pseudomonas* infection in chronic obstructive pulmonary disease. *Future microbiology*. 2012 Oct;7(10):1129-32
- [79]. Murphy TF. The many faces of *Pseudomonas aeruginosa* in chronic obstructive pulmonary disease.
- [80]. Lieberman D, Lieberman D. Pseudomonas infections in patients with COPD. *American Journal of Respiratory Medicine*. 2003 Dec 1;2(6):459-68.
- [81]. Verduin CM, Hol C, Fleer A, van Dijk H, van Belkum A. *Moraxella catarrhalis*: from emerging to established pathogen. *Clinical microbiology reviews*. 2002 Jan 1;15(1):125-44.
- [82]. Zhang W, Case S, Bowler RP, Martin RJ, Jiang DI, Chu HW. Cigarette smoke modulates PGE2 and host defence against *Moraxella catarrhalis* infection in human airway epithelial cells. *Respirology*. 2011 Apr;16(3):508-16.
- [83]. Richards SJ, Greening AP, Enright MC, Morgan MG, McKenzie H. Outbreak of *Moraxella catarrhalis* in a respiratory unit. *Thorax*. 1993 Jan 1;48(1):91-2
- [84]. Goldstein EJ, Murphy TF, Parameswaran GI. *Moraxella catarrhalis*, a human respiratory tract pathogen. *Clinical Infectious Diseases*. 2009 Jul 1;49(1):124-31.
- [85]. Perez F, Adachi J, Bonomo RA. Antibiotic-resistant gram-negative bacterial infections in patients with cancer. *Clinical infectious diseases*. 2014 Nov 15;59(suppl_5):S335-9.
- [86]. Holland T, Fowler Jr VG, Shelburne III SA. Invasive gram-positive bacterial infection in cancer patients. *Clinical infectious diseases*. 2014 Nov 15;59(suppl_5):S331-4.
- [87]. Vogelmann R, Amieva MR. The role of bacterial pathogens in cancer. *Current opinion in microbiology*. 2007 Feb 1;10(1):76-81.