

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 diseases are non-communicable disease it includes cardio vascular diseases, diabetes, chronic lung disease, cancer and obesity. Lifestyle diseases are also prone by the some microorganisms and lifestyle disease are also attracts the other disease like due to obesity there is more chances of heart disease because of hyperlipidaemia and hypertension. 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 also plays role in health. This life style disease is not curable but they can be maintaining by healthy lifestyle and dietary habits.

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 behaviour 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 behaviour 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 is a disease related to the change in people's behaviours 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 Communicable Disease (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, hyperlipidaemia, 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 behavioural risk factors ^[5]. The risk factors include the 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

Figures 1 and 2 ^[7].

LITERATURE REVIEW

Diseases and Risk Factors Related to Lifestyle Disorders

Life style diseases

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 [8,9]. Now-a-days 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 hyperlipidaemia by increased cholesterol levels or high sugar levels which is results in diabetes [10].

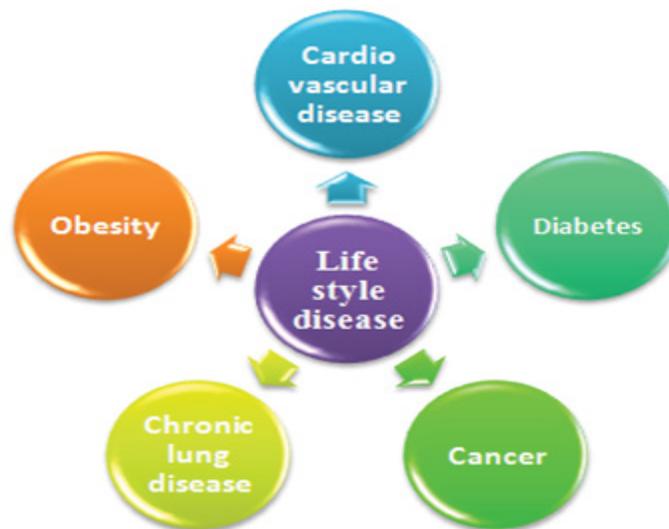


Figure 1. Lifestyle disease.

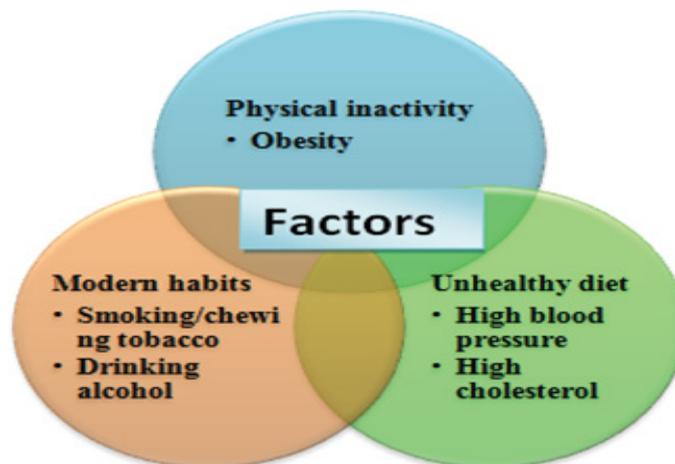


Figure 2. Factors related to lifestyle disease.

- 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 [141]. 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 [142]. 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, dyslipidaemia, glucose in tolerance and hypertension [143]. Pathophysiology of coronary heart disease includes the number of microbiological agents in which bacterial agents like *helicobacter pylori*, chlamydia pneumonia and viral agents like human immune deficiency virus, Epstein-bar virus, hepatitis virus, mycobacterium tuberculosis, human cytomegalovirus and dengue virus [144].
- 2. Diabetes:** Diabetes mellitus is chronic disorders that are characterized by high blood glucose levels (hyperglycaemia) as a result of insulin deficiency or cellular resistance to the action of insulin [145]. 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 hyperglycaemia 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 are increased due to the petroleum industries, pharmaceuticals, chemicals and dusts from the miles 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 inhaled the worsening of patient in breathing ^[21]. Haemophilus-influenzae is the most frequent bacterium isolated in all series followed by SARS covid-19, Streptococcus pneumonia, 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 (**Figure 3**) ^[22].

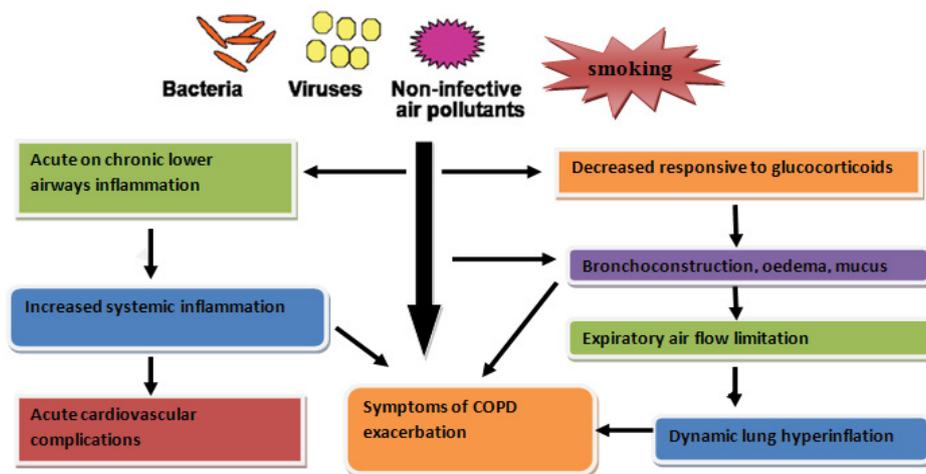


Figure 3. Pathophysiology of COPD ^[23].

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 is not described directly but the hypothesis are made which proves that they are enhancing 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**.

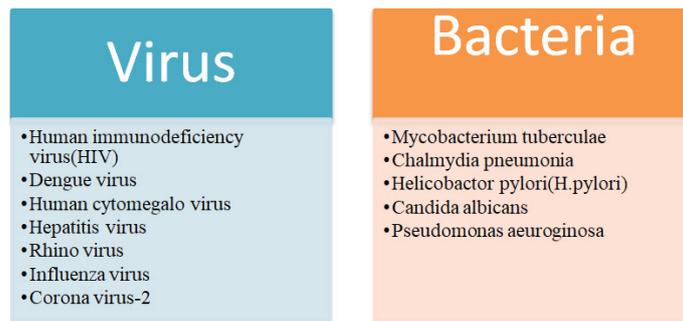


Figure 4. Causative micro-organism for life style disease ^[26].

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 cytokinins 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 were caused by the virus itself 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 (**Figure 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].

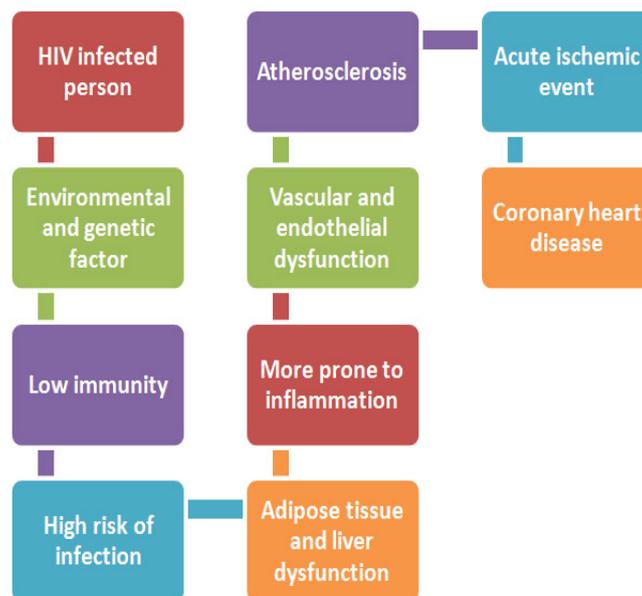


Figure 5. Pathophysiology of HIV in Cardiac Heart Disease.

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 six fold increase in the risk of cervical cancer caused by oncogenic subtypes of Human Papilloma Virus (HPV). There may be direct effects of HIV infection was such as insertional mutagenesis, upregulation of oncogenes, chronic antigenic stimulation or cytokines regulation (**Figure 6**).

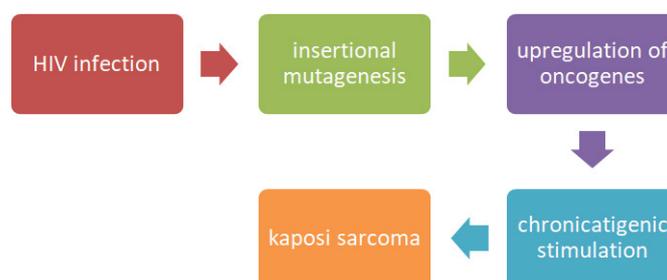


Figure 6. Kapsomi sarcoma by HIV infection.

2. 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. 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 and differential level of cytokines, which are markers of inflammation, thrombosis, and endothelial dysfunction; behavioural and social risk profile; malnutrition and/or inflammation pathway activation; or liver injury. Combination of these factors acts in a favourable risk profile and increases the overall risk of CAD.

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. 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. 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 (**Figure 7**).

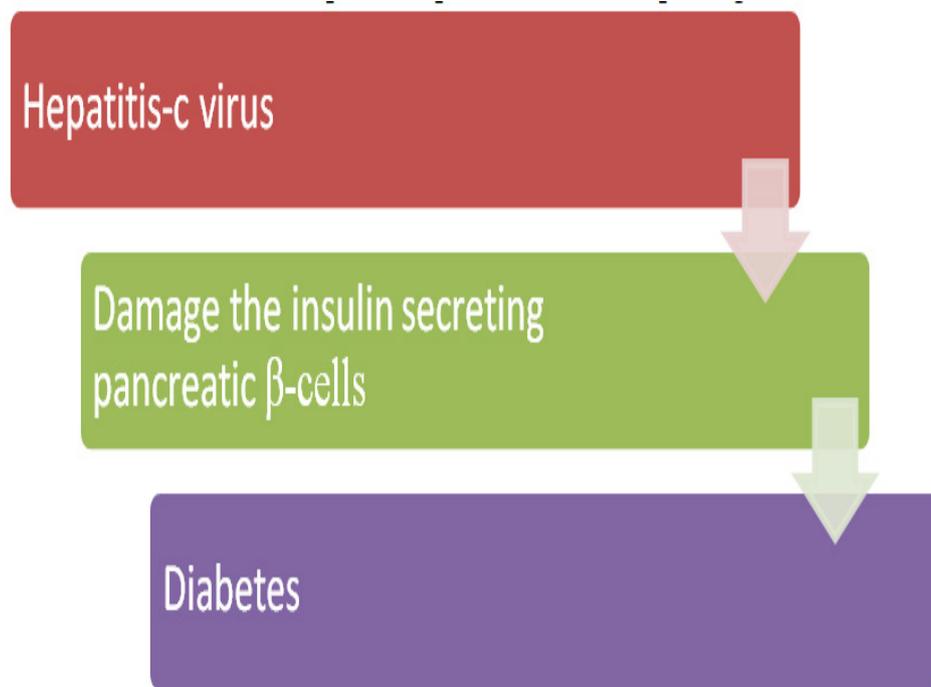


Figure 7. Hypothetical mechanism of diabetes by hepatitis-C virus.

Hepato cellular carcinomas (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.

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. 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 (**Figure 8**).

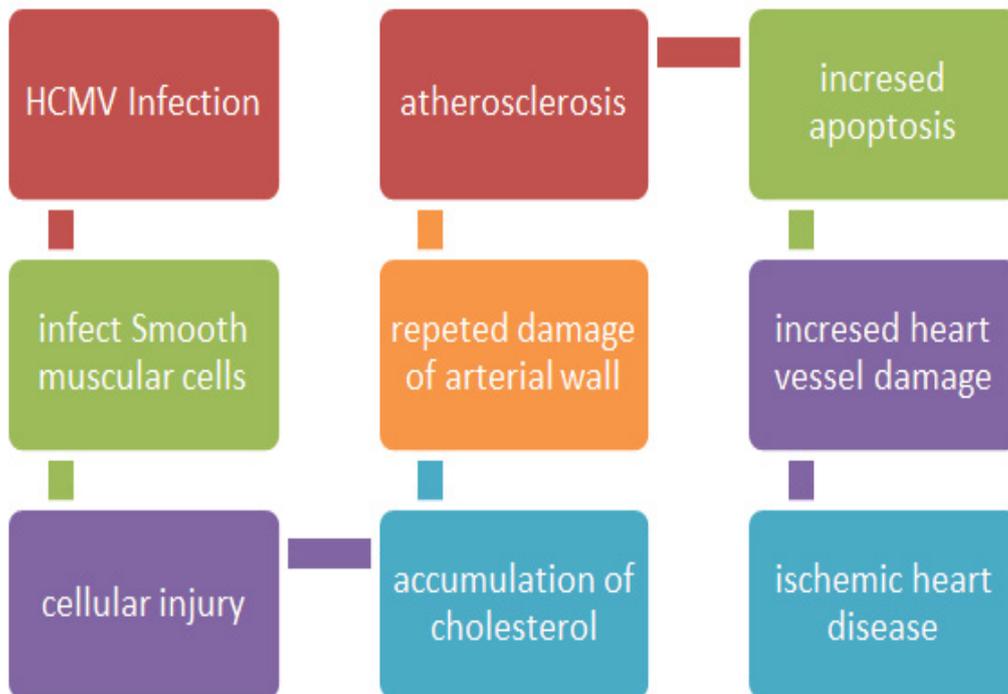


Figure 8. Mechanism of HCMV for atherosclerosis and Ischemic Heart Disease.

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. 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 (Figure 9).

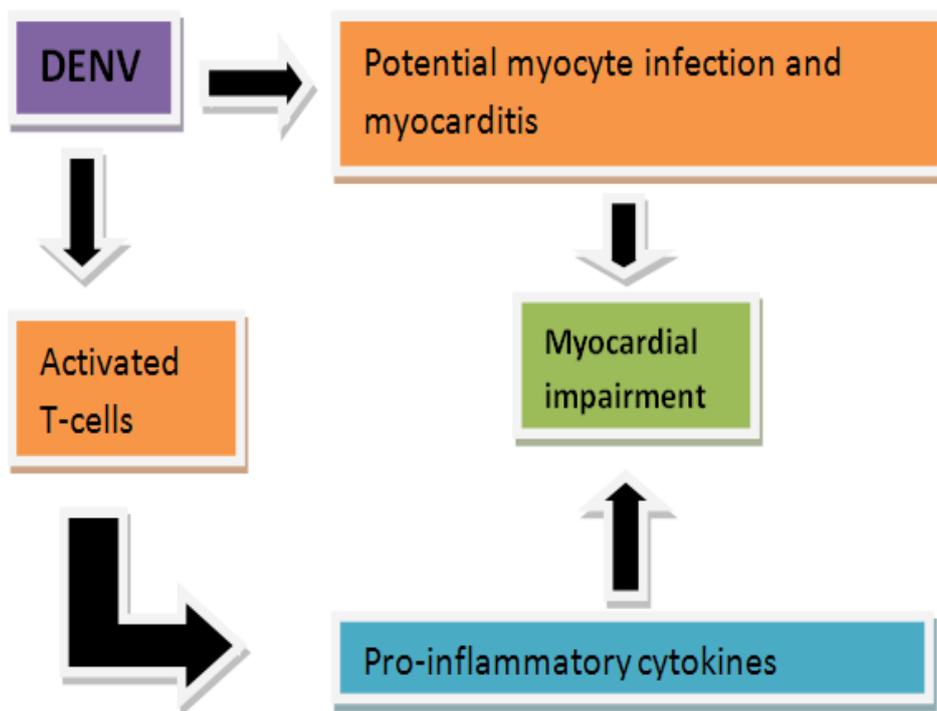


Figure 9. Mechanisms involved in the cardiac and vascular manifestations of dengue.

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 up regulation of homeostatic compensatory mechanisms can take place.

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. Non-encapsulated Haemophilus influenza often causes chronic infections of the lower respiratory tract in both non-obstructive and obstructive chronic bronchitis. 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. 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.

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.

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. 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 [28].

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 intra epithelial neoplasia (CIN) grade 3 (CIN3); 18 by contrast, HPV6 and HPV11 are responsible for about 90% of genital warts (Figure 10) [29].

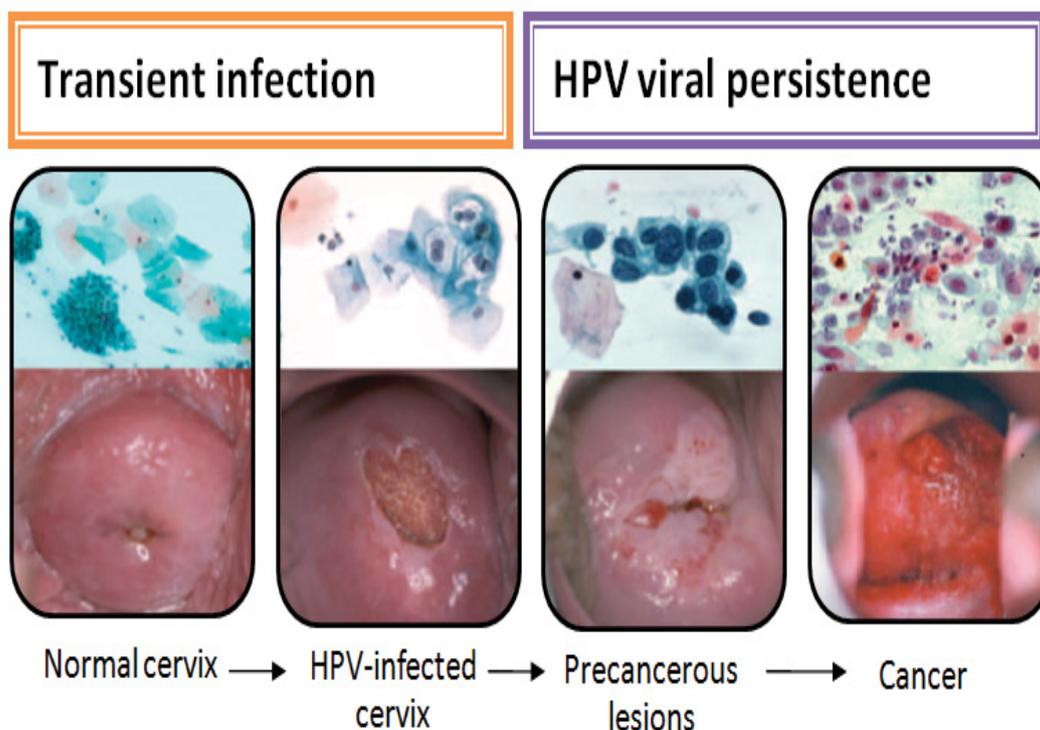


Figure 10. Development of cervical cancer.

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 appear 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. 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.

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 tumours 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 in dispute. 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.

9. Human Rhino Virus: Viral infections are associated with more severe exacerbations in terms of symptoms, resulting in longer recovery times and a greater likelihood of hospitalization. Human Rhino Virus (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). 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. A proposed mechanism of increased viral susceptibility is intracellular adhesion molecule (ICAM)-1 on respiratory epithelial cells. ICAM-1 showed up regulation in the bronchial mucosa of patients with chronic bronchitis leading to increases in HRV infection in these patients (**Figure 11**)^[30].

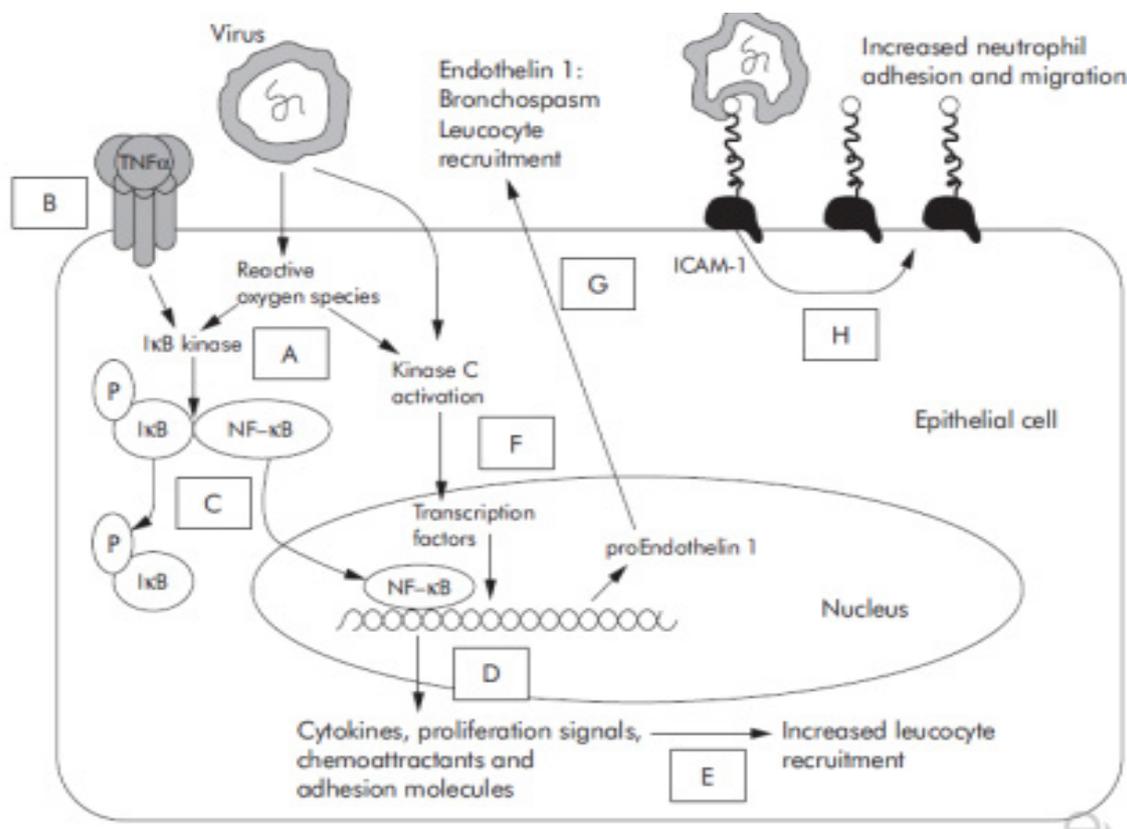


Figure 11. Key inflammatory effects of viral infection.

a) 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. The burden of tuberculosis and cardiovascular disease (CVD) is enormous worldwide and rapidly increasing in low and middle income countries (**Figure 12**).

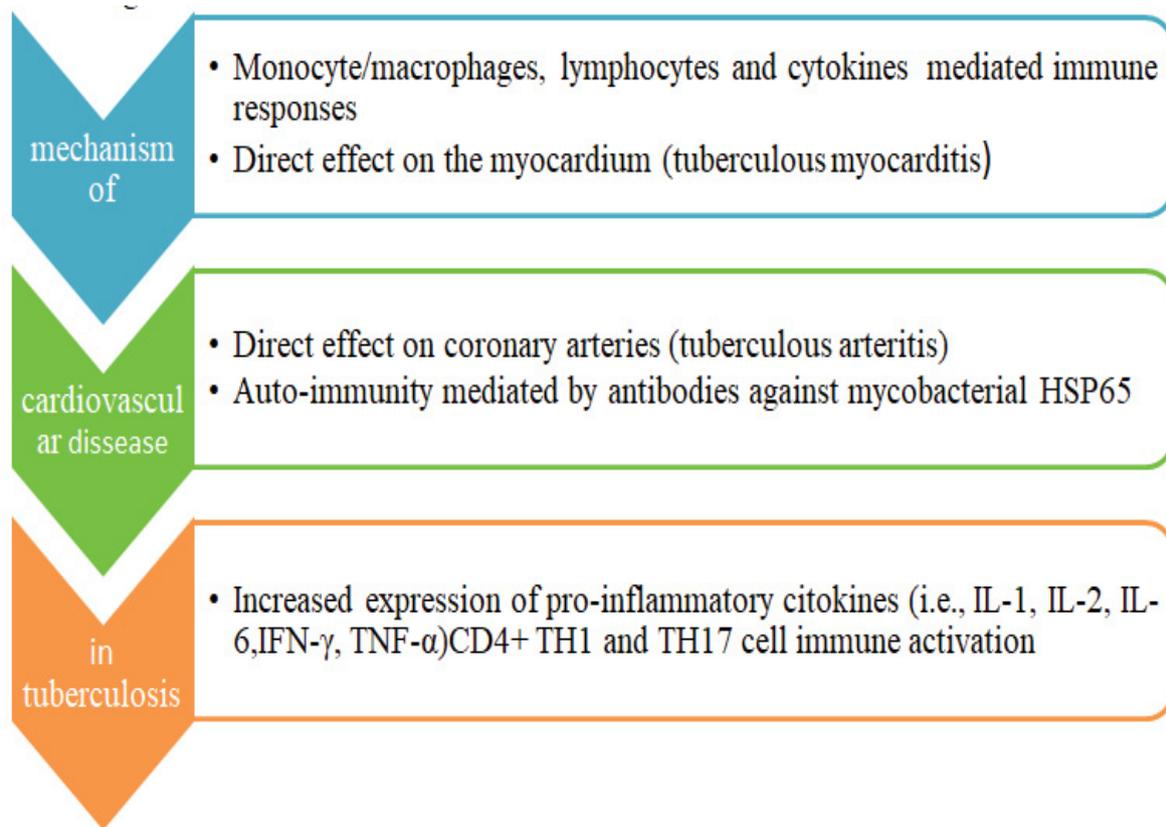


Figure 12. Possible mechanism of cardiovascular disease in TB.

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.

Diabetes increases the risk of tuberculosis incidence and the risk of adverse treatment outcomes in patients with tuberculosis. 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. 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 are occurrence of the former infection
2. They may have been re-infected with a new strain of TB.

b) *Chlamydia pneumoniae*: *Chlamydia pneumoniae* infection participates in the development of CHD, and there are several mechanisms by which are 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**).

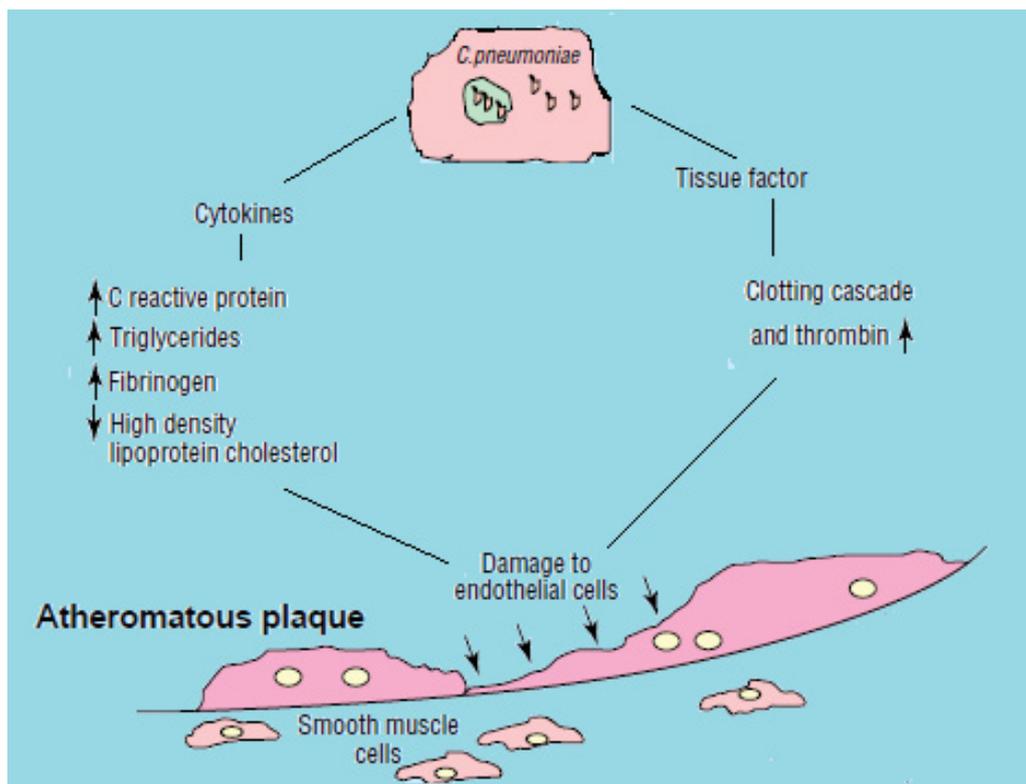


Figure 13. Atherosclerosis formation by chlamydia pneumonia.

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 factor could contribute to sub epithelial 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. 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.

c) *Helicobacter pylori*: *H. pylori* is 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 source of the *H. pylori* is drinking contaminated water, people have to be taken the care of hygiene and diet. 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. 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.

Prevalence of *H. pylori* infection was significantly higher in Type-2 Diabetes Mellitus obese subjects than non-diabetic subjects. 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 tumour 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.

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 adeno carcinoma.

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 (**Figure 14**)^[31].

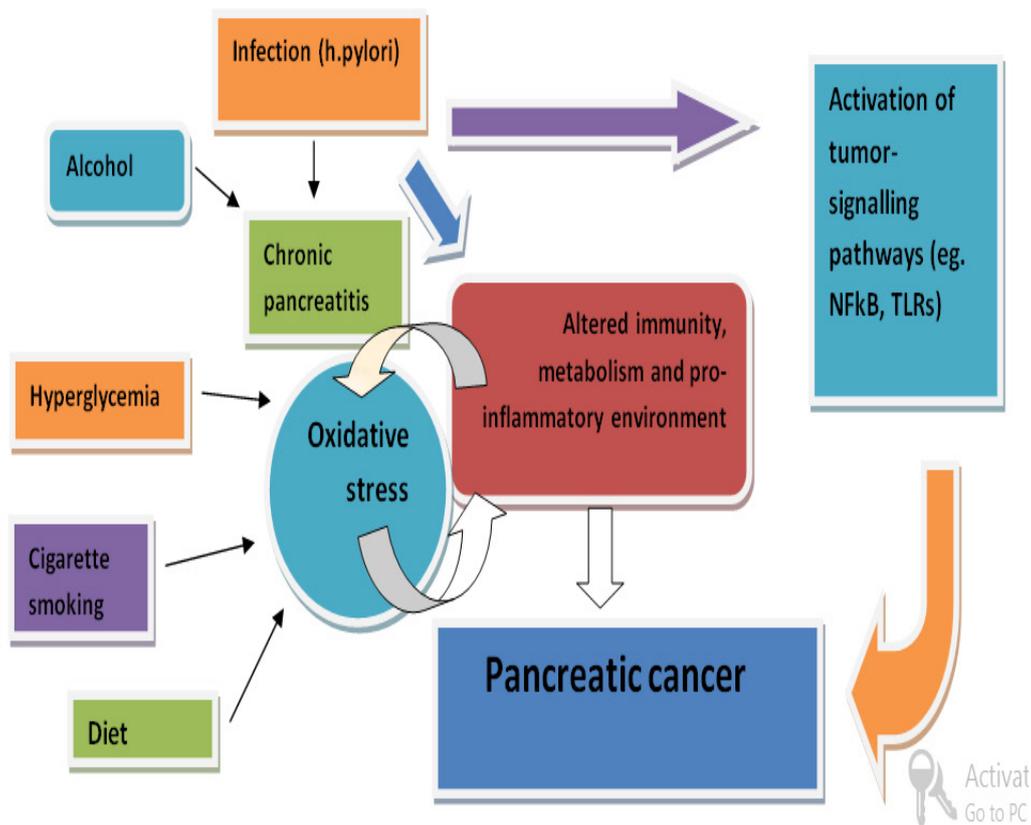


Figure 14. Role of bacterial infections in pancreatic cancer.

d) 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's and diabetic person. *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*. 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*.

e) 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. *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. 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. 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^[32].

f) Moraxella catarrhalis: *Moraxella (Branhamella) catarrhalis* is a gram-negative, aerobic diplococcus frequently found as a commensal of the upper respiratory tract. *M. catarrhalis* an essential cause of lower respiratory tract infections, particularly in adults with chronic obstructive pulmonary disease (COPD)^[33]. Cigarette smoking can leads worsening the condition of the patient. *M. catarrhalis* induces activation of the mitogen-activated protein kinase and nuclear factor-

kB 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. Serotype A is the predominant type among clinical isolates. The distribution of serotypes appears to differ by patient's 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.

g) 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*. 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. Effects of bacterial infection that contribute to carcinogenesis:

- (1) Cancer-associated bacteria provoke chronic inflammatory responses,
- (2) Directly manipulate host cell biology,
- (3) 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 malignant cells^[33].

CONCLUSION

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

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