

The major risk factors of cardiovascular diseases health and social care essay



INTRODUCTION : According to the World Health a considerable number of deaths would be prevented and tobacco related deaths would be halved if most of the smokers quit smoking over the next 20 years. People who begin smoking early have a greater risk of lung cancer compared to those who begin smoking at a later age as a result of the cumulative exposure. (Colditz & Hunter 2000). Tobacco is known to be the only legal consumer product that can cause harm to everyone exposed to it and kills most of those who use it as intended. Tobacco is also considered as the single most preventable cause of death in the world. The use of tobacco is widespread due to low prices, strong marketing, lack of education about its negative effects, and poor public policies against its use. (WHO 2008). Tobacco contains many chemicals which are known to cause cancers. (Report on Carcinogens 2005). Tobacco kills more than the combination of AIDS, legal drugs, illegal drugs, road accidents, murder and suicide. (Mackay & Eriksen 2002). The easiest way to stop the effect of tobacco is to prevent its initiation. (Robin & Sugarman 2001). Nicotine is like heroine because it is a psychoactive drug. It is also considered as a reinforcing drug, which is the reason why the smoker finds it hard to quit. Due to the reinforcing effect, most smokers may find it difficult to stop smoking. Cigarette smoking can be described as a physical and psychological addiction. (Becket 2004,). Some smokers claim that the smell, and the mere sight of a cigarette and the ritual of obtaining, lighting, handling and smoking the cigarette are all linked to the pleasurable effects of smoking and may worsen withdrawal or cravings. (National Institute on Drug Abuse 2009). Nicotine causes reliance by providing centrally mediated reinforcing effects, by controlling elements such as body weight and mood in a manner that is considered useful or pleasing by the tobacco user and by <https://assignbuster.com/the-major-risk-factors-of-cardiovascular-diseases-health-and-social-care-essay/>

causing a physical reliance such that abstinence may result in unpleasant symptoms (Jiloha 2008). Health Effects of Smoking prevalence is estimated at around 28. 6% (40% among males and 18. 2% among females) in the WHO European Region. Smoking rates in men and women has stabilized or is reducing in most western European countries. The rate of smoking in some eastern European countries is declining although in general it is rising in women and stabilizing in men. The rates of weekly smoking in fifteen-year old girls in many western European countries surpasses that of boys of the same age. The rates of weekly smoking in boys in eastern European countries is higher than that of girls. (European Tobacco Control Report 2007). Studies have shown that the smoker does not only harm him or herself but also puts the life of others at risk. Research has firmly established that an environment devoid of smoke is the only effective way to protect the population from the detrimental effects of secondhand smoke exposure. (WHO 2007). Tobacco has been found to cause many chronic diseases, acute respiratory diseases such as pneumonia and influenza, and a number of persistent respiratory symptoms such as wheezing and cough (Colditz & Hunter 2000). Research suggest that the risk of death from lung cancer may rise with amount of cigarettes smoked and duration of smoking. (Report on Carcinogens 2011). Many are of the belief that young people are picking up the habit of smoking due to ignorance about risks associated with smoking and also that it isa part of adolescent rebelliousness (Dawn 2006). Cigarrete smokers been found to have lower level of lung function compared to nonsmokers. The rate of lung growth can also be reduced by smoking. Smoking is known to affect the athletic capabilities of young people. Regular smoking may lead to coughs and worsen respiratory diseases among young

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people. Adolescent smokers experience shortness of breath at higher rates compared to nonsmoking adolescents and produce phlegm more often than those who do not smoke. Tobacco products contains elements and compounds such as arsenic, carbon monoxide, nicotine and formaldehyde which may cause wrinkles, discolouration of skin complexion and yellowing of fingernails when they enter the bloodstream. Secondhand smoke may also result in cosmetic damage when it gets in contact with the body. The harm caused by smoking on a person's appearance may be cumulative and may take several years of smoking to create observable effects. Smoking makes people seem older than they actually are. (McCay et al. 2009). Women who are smokers or who stay with a smoker are prone to health effects associated with reproductive health such as problems associated with pregnancy, use of oral contraceptive, menstrual function, and cancers of the cervix and bladder. Smoking may also lead to irregular menstrual cycles and increased menstrual discomfort. Women who are smokers may also have earlier menopause, which increases chances of getting osteoporosis, heart disease and other conditions for which estrogen provides a protective effect. The risk of sudden infant death syndrome may also increase when a pregnant woman smokes. Tobacco smoking can have negative effects on the oral cavity such as bad breath, stained teeth and life threatening conditions such as oral cancer. Also tobacco users often develop gum disease (Alters & Schiff 2009). According to Alters & Schiff (2009), since cigarette smoking has negative effects on the respiratory system, a smoker who quits may notice that it is easier to breathe. Smoking cessation reduces the occurrence of symptoms such as cough, mucus production, and wheezing. Smoking

Cessation may also reduce the occurrence of respiratory infections such as <https://assignbuster.com/the-major-risk-factors-of-cardiovascular-diseases-health-and-social-care-essay/>

bronchitis and pneumonia. (Alters & Schiff 2009.) LITERATURE REVIEW :

Smoking is one of the major risk factors of cardiovascular diseases. Amongst the thousands of identified components present in tobacco smoke, nicotine is thought to be responsible for most acute effects of smoking (Greenhoff and Svensson, 1989; Miller et. al., 1989). Current knowledge on the acute effects of smoking on the cardiovascular system is based on laboratory studies, and it is unclear whether laboratory results are compared to data gathered in natural settings when smokers are engaged in everyday activities. Under laboratory conditions, the acute action of nicotine on the cardiovascular system is similar to sympathetic activation (Greenhoff and Svensson, 1989; Miller et. al., 1989). After overnight abstinence, the short term changes elicited by one cigarette are numerous and include an increase in blood pressure of about 5 to 10 Hg, an increase in heart rate of about 10 to 20 beats per minute, and increase in cardiac output, stroke volume, and peripheral resistance (Benowitz et. al., 1986; Robertson et. al., 1988). Changes are most likely caused by an enhanced transmitter release from sympathetic nerve endings and sympathetic ganglia (Pomerleau and Pomerleau, 1984; Bassenge, 1988; Robertson et. al., 1988). Few studies have focused on the short term effects of smoking under natural condition. The effects of smoking on ambulatory hr and intra arterial bp in a group of subjects with cardiovascular problems were increases in systolic blood pressure and diastolic blood pressure 5-min after smoking when compared to pre-smoking levels (Cline et. al., 1975). hr remained unchanged after smoking . another study utilizing radiotelemetric monitoring of psychiatry patients found no significant changes in hr after smoking (Erwin, 1971). for healthy subjects, Jacobson and colleagues (1993) found a significant increase

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in hr after smoking. the acute effect of smoking on ambulatory bp in healthy individuals are yet unreported. after overnight deprivation, the activating action of one cigarette on hr and bp is an acute effect that seems to last for about 30 to 45 min under laboratory conditions(Koch et. al., 1980). so far, however research in laboratory and natural settings investigating the development of acute intraday tolerance to the cardiovascular effects of nicotine has yield mixed results(perkin et al., 1989). different administration modes of nicotine in different studies, e. g. nasal spray versus smoking cigarette make it difficult to clarify this matter . in natural settings, cellina and colleagues(1975) found no indications of acute tolerance to the bp increase found after smoking. however another study (jacobson et al. 1993) found smaller increase in hr after smoking towards the end of the day of measurement in everyday settings. Few studies have investigated the possibility that nicotine might affect men and women differently. however, there is some evidence that points in that direction. benowitz and Jacob (1984)found that the metabolic clearance of nicotine is lower in women and consequently women have tendency to be exposed to higher plasma nicotine levels. it has been suggested that women regulate nicotine intake more precisely than men(battig et al., 1982). cardiovascular reactivity to smoking has been shown to be somewhat increased in women(macdougall et al., 1988). Smoking is just one of the many factors known to affect bp and hr. laboratory and ambulatory studies have shown that posture, activity , and location influence the cardiovascular system complicating the assessment of smoking effects. bbp and hr are increased when subjects are standing compared to sitting (suter et al ., 1986). activities have also been found to affect bp and hr. for example, walking and talking increase bp and

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hr. lower levels of bp and hr are measured during reading and relaxing conditions, and the lowest bp and hr levels are seen during sleep (Pickering et al 1983). However, other studies have failed to find difference in bp levels between home and work (Kennedy et al., 1983). One study on 6 volunteers to investigate the immediate effects of smoking (and inhaling) three cigarettes separated by a 2 hr gap, upon heart rate, blood pressure (BP) and CBF assessed by xenon inhalation and flow velocities in the middle cerebral artery by transcranial Doppler ultrasound found that Smoking caused significant increases in heart rate, BP and middle cerebral artery flow velocities, and a significant fall in CBF (individual falls in hemispherical flow could be as great as 40%) (Cruickshank JM et al., 1989). E. Arosio et al (2006), found that smoking one cigarette increase resistance, impairs baroreflex and increases carotid wall tension in mild smokers. In healthy subjects cardiopulmonary baroreceptor stimulation causes humeral artery dilation and a reduction of peripheral resistance owing to an increased vagal tone. Furthermore, cardiopulmonary receptors exert inhibitory effects on the vasomotor centre of the oblongata, reducing the sympathetic tone. Smoking one cigarette causes a reduction in humeral calibre and impairs humeral dilation. Increased sympathetic tone can enhance vasoconstriction at rest. Furthermore, we can evoke an impairment of cardiopulmonary baroreceptors. Endothelial dysfunction, caused acutely by smoking, could play a role in this mechanism, producing a paradoxical response of brachial artery to the stimulus of baroreceptors. On the other hand, a slight increase of circulating catecholamines could counteract vagal function. Smoking cessation may cause a resetting of the autonomic function toward a decrease of sympathetic activation and a restored vagal tone.

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Baroreceptor activity should be carefully studied, as it could take time to reset. Duration of smoking could influence the recovery of the baroreceptor function. Probably, a transitory vagal hyperactivity could be hypothesized, contributing to the symptoms occurring after smoking cessation. In conclusion, smoking one cigarette acts significantly on vascular regulation by increasing resistance, impairing baroreflex function and increasing carotid wall tension in mild smokers. All these changes may take part in the atherosclerotic damage and could explain the higher rate of cardiovascular events affecting smokers. (E. Arosio et al., 2006) COPD is currently one of the major causes of death in the world, and further increases in the prevalence and mortality of this disease are predicted in the coming decades. 1, 2 Murray and Lopez estimated that COPD will become the third most common cause of death by 2020. 3 The major cause of this increase is previous smoking behaviour and population aging A survey conducted by the Japanese Ministry of Health and Welfare in 1987 reported the number of patients with emphysema to be 32 000. 5 A further survey conducted in 1996, reported approximately 220 000 diagnosed cases of COPD (prevalence of 0. 2%)⁵ and 65 000 with emphysema, a twofold or more increase in less than 10 years. It is expected that in the near future the high prevalence of cigarette smoking and demographic changes in Japan will result in a further substantial increase in mortality due to COPD. 5 According to data from the Nippon COPD epidemiology (NICE) study, 6 the prevalence of airflow limitation was 10. 9% overall (males, 16. 4%; females, 5. 0%). Based on this study, it is estimated that over 5 million Japanese individuals over the age of 40 have airflow limitation consistent with a diagnosis of COPD, 6 some 20 times greater than rates previously reported in Japan. The prevalence also

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appears to be higher than those described for North America and Europe. 7 This may reflect the fact that smoking prevalence among men in Japan is considerably higher than in North America and Europe. Damage to the respiratory system from cigarette smoking is slow, progressive, and deadly. A healthy respiratory system is continuously cleansed. The mucus produced by the respiratory tubules traps dirt and disease-causing organisms, which cilia sweep toward the mouth, where it can be eliminated. Smoking greatly impairs this housekeeping. With the very first inhalation of smoke, the beating of the cilia slows. With time, the cilia become paralyzed and, eventually, disappear altogether. The loss of cilia leads to the development of smoker's cough. The cilia no longer effectively remove mucus, so the individual must cough it up. Coughing is usually worse in the morning because mucus has accumulated during sleep. To make matters worse, excess mucus is produced and accumulates, clogging the air passageways. Pathogenic organisms that are normally removed now have easier access to the respiratory surfaces and the resulting lung congestion favors their growth. This is why smokers are sick more often than nonsmokers. In addition, a lethal chain reaction begins. Smoker's cough leads to chronic bronchitis, caused by destroyed respiratory cilia. Mucus production increases and the lining of the bronchioles thickens, making breathing difficult. The bronchioles lose elasticity and are no longer able to absorb the pressure within the alveoli (microscopic air sacs) enough to rupture the delicate alveolar walls; this condition is the hallmark of smoking-induced emphysema. The burst alveoli cause worsening of the cough, fatigue, wheezing, and impaired breathing. Emphysema is fifteen times more common among individuals who smoke a pack of cigarettes a day than

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among nonsmokers. Simultaneous with the structural changes progressing to emphysema may be cellular changes leading to lung cancer. First, cells in the outer border of the bronchial lining begin to divide more rapidly than usual. Eventually, these displace the ciliated cells. Their nuclei begin to resemble those of cancerous cells--large and distorted with abnormal numbers of chromosomes. Up to this point, the damage can be repaired if smoking ceases. However, if smoking continues, these cells may eventually break through the basement membrane and begin dividing within the lung tissue, forming a tumor with the potential of spreading throughout lung tissue. Eighty percent of lung cancer cases are due to cigarette smoking. Only 13% of lung cancer patients live as long as 5 years after the initial diagnosis. Hisamitsu OMORI, 1 Yoshio NONAMI1 AND Yasuo MORIMOTO2 2005 performed cross-sectional study, pulmonary function data from 11 875 healthy asymptomatic males, aged between 35 and 74 and analysed, correlated with their smoking history and age. In the study, changes in pulmonary function were monitored over a 5-year period in 1888 healthy males. The study showed that the difference in FEV1 between male never smokers and current smokers was small at a younger age but increased with age. A beneficial effect on FEV1 decline was observed in those who ceased smoking, even within the previous 12 months. Longitudinally, current smokers showed a more rapid decline in FEV1 over the 5-year period than nonsmokers. Those who ceased smoking had lower rates of decline in FEV1 than those who continued to smoke. the results of the study had showed that cigarette smoking is associated with a reduction in pulmonary function, and that smoking cessation has a beneficial effect on FEV1 decline. Mohan Sopori (2002) Although the health risks of tobacco smoking are well <https://assignbuster.com/the-major-risk-factors-of-cardiovascular-diseases-health-and-social-care-essay/>

documented, there is increasing evidence that smokers have a lower incidence of some inflammatory and neurodegenerative diseases. Many of the adverse and beneficial effects of smoking might result from the ability of cigarette smoke to suppress the immune system. Nicotine, which is one of the main constituents of cigarette smoke, suppresses the immune system but might have therapeutic potential as a neuroprotective and anti-inflammatory agent. Smoking has substantial local and systemic adverse effects on the immune system, respiratory tract and skin and soft tissues. Smokers are at increased risk of invasive pneumococcal disease, pneumonia, periodontitis, surgical infections, tuberculosis, influenza and meningococcal disease. The results of several studies indicate that smokers with periodontitis or tuberculosis suffer more severe disease (Huttunen R et al ., 2010) The World Health Organization recently reported that smoking causes 9% of all deaths worldwide . The US Centers for Disease Control and Prevention's online computer program SAMMEC (Smoking-attributable Mortality, Morbidity, and Economic Costs) is widely used to produce estimates of tobacco-related mortality. Based on estimates using SAMMEC, the smoking-attributable risk ratios for influenza and pneumonia (together) mortality were 2. 0 for men and 2. 2 for women in the USA Several large case-control studies [23, 24, 37-39], large longitudinal cohort studies [25, 26] and meta-analyses [7, 8, 40] have been undertaken to analyse the impact of smoking on the outcome of tuberculosis. These studies, most of which have been conducted in countries with a high prevalence of tuberculosis, almost invariably indicate significantly increased mortality for smokers in tuberculosis Abundant data indicate that smoking is a major factor associated with the outcome of periodontal disease [5]. Smoking has

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been associated with indicators of severe periodontal disease such as periodontal loss of attachment, bone loss and premature tooth loss in cross-sectional [45–52] and longitudinal studies [53–56], and the outcome of periodontal treatment has been shown to be less favourable or indeed unfavourable in smokers compared with nonsmokers [57, 58]. Several studies have also explored the relation between exposure level and disease severity and found a correlation between increasing exposure to smoking and severe periodontal disease. Smoking has been shown to be a risk factor for complications and mortality after surgery [60–63]. The results of randomized clinical studies indicate that abstinence from smoking prior to surgery may reduce postoperative infections and other wound-related complications [64, 65]. Few studies have specifically addressed the impact of smoking on postsurgical infection. Some cohort studies have indicated that smoking may alter the disease course of influenza infection. One cross-sectional cohort study showed that smoking predisposes young men to more severe influenza and that the effect of smoking on disease severity is dose dependent [4]. A cohort study of female military recruits showed that smoking was a risk factor for severe influenza-like illness during an outbreak of influenza A (H1N1) subtype infection [68]. In a 16-year mortality follow-up of nearly 300 000 US veterans, influenza-related mortality (pneumonia and influenza deaths combined) was higher in cigarette smokers than in nonsmokers. The relation between cigarette smoking and the frequency and duration of colds was analysed in the Women's Health Study. This large longitudinal cohort study showed that women who are currently heavy smokers are at increased risk of contracting colds of longer duration compared with nonsmokers. However, the number of days confined to home

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with a cold was not affected by smoking status. The effect of smoking on the course of fungal infections is unclear. Smoking emerged as a risk factor for symptomatic coccidioidomycosis in one case-control study [97] and constituted an independent risk factor for increased disease severity in pulmonary coccidioidomycosis in another [98]. An association between smoking and poor outcome in disseminated cryptococcosis was shown in one small cohort study [99]. Smoking has substantial local and systemic effects on the respiratory tract, the immune system and the skin and soft tissues, and the changes induced may alter both susceptibility to contract infection and the course of an infectious disease [108, 109] (Table 3). Tobacco smoke contains more than 4500 compounds, many of which have toxic, irritant or even directly carcinogenic properties [109, 110]. Studies of the effects of smoking on the immune system may experience similar difficulties to those of outcome studies; differences in smoking history and genetic and socio-economic factors within studied populations may cause heterogeneity between different studies. In addition, in animal models, parameters of smoke exposure such as duration, mode and frequency vary between studies [109]. Given the huge number of compounds in cigarette smoke, studies using individual components of smoke to assess the overall effect of smoking on the immune system should be considered with caution [109].

The compromised ability to withstand an infectious assault has been attributed to the suppressed innate and acquired immunity in smokers (6). This effect of smoking on immune defense has been studied extensively, but unfortunately, many controversies regarding characteristic alterations and underlying processes remain unresolved. Previous studies have described decreased natural killers (NK) number and cytolytic activity (7) and

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depressed phagocytosis by macrophages in smokers (8). Reduced production of the proinflammatory cytokines IL-1b, IL-6 and TNFa by alveolar macrophages has also been demonstrated As a mechanism for the immune impairment the stimulatory effect of nicotine on sympathetic dis-charge H-thymidine incorporation and NK cell cytotoxicity by 51Cr release assay. Results concluded that Mononuclear cells from smokers showed increased production of the pro-inflammatory cytokines IL-1b, IL-6 and TNFa and enhanced proliferative response to mitogens as compared to non-smoking population. The secretion of IL-2 and the anti-inflammatory cytokines IL-1ra and IL-10 was similar in both groups. NK cell cytotoxic activity was suppressed in the smokers. Cigarette smokers without chronic obstructive pulmonary disease (COPD) exhibit impaired NK cytotoxic activity in peripheral blood and unbalanced systemic production of proand anti-inflammatory cytokines. These changes may serve as predisposing factors for respiratory and systemic infections in the postoperative period and should alert an anesthetist during perioperative management. c Acta Anaesthesiologica Scandinavica 46 (2002) charge and hypothalamic-pituitary axis, known for their immunosuppressive action, has been suggested Previous studies have described decreased natural killers (NK) number and cytolytic activity (7) and depressed phagocytosis by macrophages in smokers (8). Reduced production of the proinflammatory cytokines IL-1b, IL-6 and TNFa by alveolar macrophages has also been demonstrated (9, 10). As a mechanism for the immune impairment the stimulatory effect of nicotine on sympathetic dis- 959 by 3 H-thymidine incorporation and NK cell cytotoxicity by 51Cr release assay. Results: Mononuclear cells from smokers showed increased production of the pro-inflammatory cytokines IL-1b, IL-6 <https://assignbuster.com/the-major-risk-factors-of-cardiovascular-diseases-health-and-social-care-essay/>

and TNF α and enhanced proliferative response to mitogens as compared to non-smoking population. The secretion of IL-2 and the anti-inflammatory cytokines IL-1 α and IL-10 was similar in both groups. NK cell cytotoxic activity was suppressed in the smokers. Conclusion: Cigarette smokers without chronic obstructive pulmonary disease (COPD) exhibit impaired NK cytotoxic activity in peripheral blood and unbalanced systemic production of proand anti-inflammatory cytokines. These changes may serve as predisposing factors for respiratory and systemic infections in the postoperative period and should alert an anesthetist during perioperative management. Received 18 December 2001, accepted for publication 25 March 2002 Key words: Cytokines; immune response; NK cell cytotoxicity; smoking. c Acta Anaesthesiologica Scandinavica 46 (2002) charge and hypothalamic-pituitary axis, known for their immunosuppressive action, has been suggested An impaired capability of B-cells to differentiate and produce all types of immunoglobulins but IgE has been described in cigarette smokers (13, 14). Although serum level of IgE was not correlated with enhanced skin-test reactivity and increased incidence of bronchial asthma in smokers, smoking was described as a predisposing factor for autoimmune diseases However, an increase in the secretion of pro-in- flammatory cytokines in smokers has also been reported (6, 16). As cigarette smoke contains at least 3800 components in addition to nicotine, it was suggestedthat some of them, such as tobacco-related globulin (TRG), might exert immunostimulatory effect (16). Most investigators have used the alveolar-derivedcells (macrophages and NK) in order to test immune functions in smokers. Although macrophages can migrate freely through blood and body tissues, it remains unclear whether blood. (A. ZEIDEL et al., <https://assignbuster.com/the-major-risk-factors-of-cardiovascular-diseases-health-and-social-care-essay/>)

2002). A. ZEIDEL et al., 2002 demonstrated that cigarette smoking affects the immune system. Impairment of alveolar mononuclear cell function, described previously, may contribute to the higher rate of postoperative respiratory infections. However, increased susceptibility of smokers to infections of other origin (e. g. wound-related) implies that tobacco effect is not restricted to the respiratory immune competent cells. A. ZEIDEL et al., 2002 investigated the systemic effect of tobacco smoking as it exerted on blood-derived immune cell and measured systemic cytotoxic activity of natural killer cells, production of pro- and anti-inflammatory cytokines by blood mononuclear cells and their proliferation in response to mitogens. Mononuclear cells from smokers showed increased production of the pro-inflammatory cytokines IL-1b, IL-6 and TNFa and enhanced proliferative response to mitogens as compared to non-smoking population. The secretion of IL-2 and the anti-inflammatory cytokines IL-1ra and IL-10 was similar in both groups. NK cell cytotoxic activity was suppressed in the smokers. A. ZEIDEL et al., 2002 concluded that Cigarette smokers without chronic obstructive pulmonary disease (COPD) exhibit impaired NK cytotoxic activity in peripheral blood and unbalanced systemic production of pro and anti-inflammatory cytokines. These changes may serve as predisposing factors for respiratory and systemic infections in the postoperative period and should alert an anesthetist during perioperative management. Cigarette smoking is a strong and established risk factor for vascular disease (e. g., coronary heart disease, cerebrovascular disease), respiratory disease, and some cancers (Joint Committee on Smoking and Health, 1995; U. S.

Department of Health and Human Services, 2004) and the leading cause of human health problems in developed countries (World Health Organization <https://assignbuster.com/the-major-risk-factors-of-cardiovascular-diseases-health-and-social-care-essay/>

[WHO], 2002). Exercise helps prevent obesity, hypertension, and dyslipidemia, and increased physical activity is recommended for health promotion and prevention of lifestyle-related diseases such as diabetes and vascular disease (American College of Sports Medicine, 2005; American Heart Association Scientific Council, 1992). In the National Nutrition Survey in Japan in 2003, 46.8% and 69.7% of male adults were current smokers and had a sedentary lifestyle of less than 30 min/day or 2 days/week of exercise, respectively (Ministry of Health, Labour and Welfare, 2005). Because of these known factors, smoking avoidance or cessation and increased physical activity are both important public health tasks for Japanese males. It has been reported that smoking and sedentary lifestyle appear to coexist (Blair, Jacobs, & Powell, 1985; Conway & Cronan, 1992; Emmons, Marcus, Linnan, Rossi, & Abrams, 1994; Klesges, Eck, Isbell, Fullerton, & Hanson, 1990; Kvaavik, Meyer, & Tverdal, 2004), that habitual exercise may reduce the progression of smoking from occasional to heavier smoking (Audrain-McGovern, Rodriguez, & Moss, 2003), and that interventions to increase habitual exercise may help current smokers cease smoking (Ussher, West, Taylor, & McEwen). Teruo Nagaya et al., 2007, 7-year longitudinal follow-up on the relationship of smoking behaviors to habitual exercise in healthy men. Two main results are that (a) persistent smokers consistently exercised less than never-smokers through the follow-ups, and that (b) habitual exercise gradually increased after smoking cessation to the levels of never-smokers and decreased after smoking relapse toward the levels of persistent smokers within 3–4 years. Smoking is a leading factor in adverse health outcomes in developed areas (WHO, 2002). Its adverse impacts on health are derived not only from the direct

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effects of toxic components of tobacco and tobacco smoke but also from its possible impact on behaviors, including sedentary lifestyle, unhealthy diet, and heavy alcohol consumption. The behavioral impacts of smoking seem sufficiently significant that smoking cessation would improve human behaviors and lifestyles and promote improved health. Conversely, smoking initiation or relapse may aggravate healthaverse behaviors and lifestyles. Public health initiatives should make this information widely available to smokers, nonsmokers, and health workers as a means of preventing smoking resumption or initiation and helping smokers cease smoking. Exercise play an important role in the acute management of tobacco withdrawal symptoms (e. g. depression, irritability, restlessness, poor concentration) and cravings that predict smoking relapse. Pomerleau and colleagues [25] and Everson and colleagues [26] reported no significant difference on cravings between cycling at 80% versus 30% of VO_{2max} (for 30 minutes), and between 55% versus 44% of age-predicted maximal heart rate (for 10 minutes), respectively. Pomerleau study was underpowered with just 10 subjects; a trend towards lower cravings in response to higher intensity exercise was reported. Also, the participants had only 30 minutes of abstinence prior to treatment, suggesting that baseline cravings may not have been particularly high (they were not reported) and a flooring effect may have masked any effects of exercise. The absence of a significant difference in cravings between the two intensities of exercise in the Everson study seems due, probably, to the relatively similar exercise intensities. West and colleagues [30] showed a reduction in ratings of strength of desire to smoke of up to 1.0 point on a sevenpoint scale for glucose versus placebo; whereas, using the same scale, exercise shows a mean reduction in ratings of desire to smoke, <https://assignbuster.com/the-major-risk-factors-of-cardiovascular-diseases-health-and-social-care-essay/>

compared with control, ranging from 0.7 points for 5 minutes of isometric exercise [22], to 4.6 points for a 1-mile brisk walk [18]. Also, exercise tends to show a more consistent and rapid effect on cravings and withdrawal symptoms when compared with studies of oral NRT [15, 31]. For example, in one of the most rigorous studies, effects were not evident until 10 minutes after taking the nicotine gum, relative to a placebo (Taylor AH, Ussher MH, Faulkner G 2007.)

DISCUSSION This study aimed at finding the reasons why adolescent start smoking. Six articles (Can et al. 2009; Weiss et al. 2008; Damianaki et al. 2008; DiNapoli 2009; Hayes & Plowfield 2007; Kulbok et al. 2008) were systematically selected to answer the research question. The results showed that friends are an important influence in the smoking behaviour of adolescents. From the study it was found that adolescents whose friends smoked were more at risk than those whose friends did not smoke. This is consistent with studies by Kobus (2003, 45) and Harakeh (2007, 280) who found that adolescents who had best friends who smoked cigarettes were more susceptible to smoking. According to Pärna (2003, 356), having a friend who smoked was one of the major and strongest factors associated with smoking. Also smoking rates were higher among popular students in schools with high smoking prevalence in a study by Alexander et al (2001, 27). This is supported by a study by Valente et al. (2005, 323) which also found that popular middle school students were more likely to become smokers compared to their less popular peers. Hayes & Plowfield (2007, 115) found peer pressure to be the greatest contributing factor for starting to smoke. According to Brook et al (2005, 214), adolescents with tobacco-prone personalities tend to associate with tobacco using and deviant peers and also having friends who smoke was linked with

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adolescent having tobacco-prone personality traits. The role played by the family in adolescent smoking initiation was also discussed. Most of the articles touched on the influence of sibling smoking. The articles simply talked about sibling influence without clarifying whether it was the younger or older sibling that influenced the adolescent smoking. Study by Harakeh et al (2007, 281) showed that only the older siblings influenced the younger siblings' smoking. Harakeh (2007, 272) explained sibling influence using the Social Learning Theory. According to Harakeh (2007, 272), the older sibling may be seen as a role model by the younger sibling. The younger sibling may think of the older sibling as more competent and knowledgeable and as such may look up to him or her for support in many areas. Therefore the younger sibling may copy the behavior of older siblings especially when they spend a lot of time together. Also adolescents with parents who smoke were found to be more at risk than adolescents with nonsmoking parents. Availability of tobacco products to adolescents may be a contributing factor to smoking initiation (Robin & Sugarman 2001, 144). According to Alexander et al (1999, 254), becoming exposed to parents or other family members who use tobacco may arouse curiosity. Psychological factors were also found to contribute to adolescent smoking initiation. Weiss et al. (2008, 149) found an association between anxiety, hostility and increased risk of smoking. Adolescents who were more worried and nervous, having troubles in regulating anger, feeling sad and lonely, were more likely to have tried smoking (Weiss 2008, 154). According to MacDonald (2004, 27), poverty may result in stress, anxiety and depression and poor people are more likely to smoke. This is also consistent with a study by Weiss et al. (2011, 596). Weiss et al. (2011, 596) found a longitudinal relationship between smoking initiation

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and psychological characteristics. In the study by Weiss et al. (2011, 596), the risk of smoking initiation was higher among students who scored higher on hostility and depressive symptoms, and were bully-victims. Adolescents who are less interested in their health, who have low self image and who have low confidence are also bound to start smoking. This is consistent with study by Hayes & Plowfield (2007, 114). A study by Song et al (2009, 490), found that the perception of risks and benefits has an effect on smoking initiation. According DiNapoli (2009, 131), poor academic abilities and low feelings of emotional wellbeing are associated with low ego development. According to DiNapoli (2009, 131), sociostructural variables within the family and community may also affect ego development (DiNapoli 2009, 131.) In the study by DiNapoli (2009, 130), early initiation of tobacco use was more likely in persons with low self-esteem, who reported being victimized. Some risk groups were identified in a number of articles. High smoking prevalence among adolescents in their last grades of high school was recorded in the study by Damianaki et al (2008, 310). This indicates that adolescents in their last grades are a risk group. Also adolescents with poor academic performance were found to initiate smoking at higher rates than those with good grades (Can et al 2009, 95; DiNipoli 2009, 130). Adolescents with body piercings and tattoos can also be considered a risk group because they have the tendency to engage in risky behaviours as discussed in the literature. Being popular in a school with high smoking prevalence can also be considered a risk for smoking. A study by Alexander et al. (2001, 27) found that popular students in schools with high smoking rates are more likely to be cigarette smokers compared to their less popular counterparts. The role played by the media in smoking initiation was not discussed in the selected

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articles. However a number of studies have found that television and movies play a crucial role in the smoking behavior of adolescents. According to Gidwani et al (2002, 505), adolescents who watched a lot of television a day were more likely to initiate smoking than those who spent lesser time on television. Weight concerns among adolescent girls may also be contributing factor to adolescent smoking (Austin & Gortmaker 2001, 446; French et al. 1994, 1820). This is also supported by Cawley et al. (2004, 309). Cawley et al.(2004, 309) found that females who have a high body mass index, who report that they are trying to lose weight, and who describe themselves as overweight are more likely to initiate smoking than other females.

CONCLUSIONThe review showed that the family, peers, adolescents sense of wellbeing and self-esteem are strong influences in the smoking habit of adolescents. The study also found that adolescents with psychological problems have a high chance of initiating smoking. Some risk groups were identified in the study. These included adolescents with low self esteem, popular students in schools with high smoking prevalence, those with weight concerns, adolescents in their last grades in school, adolescents who spend considerable amount of time on television, adolescents who performed poorly in school and adolescents with tattoos. Since prevention is said to be better than cure, it is important to examine the root cause of the problem.

Smoking prevention programs should aim at identifying risk groups and finding measures to protect these vulnerable group from initiation.

Adolescents should be counselled on the effects of keeping bad company and advised to choose their friends wisely. Programs should aim at helping adolescents gain emotional control so that they don't give in to pressures

from peers. Also since adolescents learn by imitation, older siblings and <https://assignbuster.com/the-major-risk-factors-of-cardiovascular-diseases-health-and-social-care-essay/>

family members should be educated on the dangers of smoking in the presence of adolescents and also about leaving cigarettes at the disposal of adolescents. Adolescents should be supported on ways of dealing with stress so that they don't turn to cigarettes. An important preventive strategy is promoting ego development. Ego development is vital in building self-determined health behaviour.