Review Article

TOBACCO ABUSE AND ITS HEALTH EFFECT

[1]Jacob Amos Dunga, [2]Yakubu Adamu, [3]Ibrahim Musa Kida, [4]Datonya Alasiya, [1]Yusuf Jibrin, [1]Umar Sabo, [5]Christian Ukoli, [5]CH Chuhwak, [1]Jafiada Jacob Musa

[1]Abubakar Tafawa Balewa University Teaching Hospital, Bauchi, Nigeria
[2]US Department of Defense, HIV/AIDS Research Abuja, Nigeria
[3]University of Maiduguri Teaching Hospital, Maiduguri, Nigeria.
[4]University of Port Harcourt Teaching Hospital, Rivers, Nigeria
[5]Jos University Teaching Hospital, Plateau, Nigeria.

ABSTRACT

Tobacco smoking is still one of the most important risk factor for Respiratory and cardiovascular diseases and an estimated 90% of causes of lung cancer are attributable to Tobacco smocking and equally 90% of peripheral vascular disease in non-diabetic population is attributable to Tobacco smoking, despite the health effect there is disturbing figures of people who take up smoking habit daily and increase level of failed quit smoking attempts. Environment and genetics still plays major role, and various forms of tobacco is used worldwide and its health consequence has been highlighted. Monitoring tobacco use and prevention policies through effective tax laws is paramount to reduction of the tobacco health effects in our environments.

KEY WORD: Tobacco abuse, cigarrete smoking, health effect.

NigerJMed2015: 354-362

INTRODUCTION

Tobacco is an important cash crop in American farming, native to North and South American continent[1]. The use of tobacco leaf to create and satisfy nicotine addiction was introduced to Columbus by Native Americans in 14th century and spread rapidly to Europe.[1]

The use of tobacco as cigarette, however, is predominantly a 20th century phenomenon just as the epidemic of disease caused by this form of tobacco use[1]. Nicotine is the principal constituent of tobacco responsible for its additive characteristics. Addictive smokers regulate their nicotine intake and blood levels by adjusting the frequency and intensity of their tobacco use to obtain the desired psychoactive effect and avoid withdrawal[1]. Tobacco use is a powerful, independent predictor of deaths related to cardiovascular disease and is an important contributor to deaths from major chronic diseases. Unfortunately, increasing rates of tobacco use throughout the world is contributing significantly to the burden of death and disability from these diseases, especially in developing countries. If current trends continue, the annual numbers of people killed by tobacco use will more than triple, to 10 million by 2030.[2]

Corresponding Author: Jacob Amos Dunga
Abubakar Tafawa Balewa University Teaching Hospital, Bauchi, Nigeria

REASONS FOR TOBACCO ABUSE

Approximately 90% of individuals who will become smokers initiate the behavior during adolescence.[3] Factors that promote adolescent initiation are parental or older generation cigarette smoking, tobacco advertisement and promotional activities, the availability of cigarette, culture, and the social acceptability of smoking. Others include the need for an enhanced self image and to initiate adult behavior. It is greater among those adolescents who have the least external validation of their self worth. This may explain in part the enormous difference in adolescent smoking prevalence by socioeconomic and school performance strata[3].

Genetic considerations:

Several genes have been associated with nicotine addiction.[4] Some reduce the clearance of nicotine and others have been associated with increased likelihood of becoming dependant on tobacco and other drugs as well as a higher incidence of depression.[4] Genetic alteration that involves the neurotransmitter dopamine, serotogenic and cholinergic neuro-regulatory pathways are being explored but their contribution to development and maintenance of nicotine addiction remains to be determined. It is unlikely that genetic factors are the principal determinants of addiction.[5] The rate of smoking initiation among males and corresponding rates of nicotine addiction have dropped by almost 50% since

the 1950s, suggesting that factors other than genetics are the principal determinants of addiction.[5] It is more likely that genetic polymorphism represents a range of biologic susceptibility conditioning, the intensity of cigarette use and the probability that experimentation with tobacco as an adolescent leads to addiction as an adult [3,4]

In Africans in addition to the abuse factors, smoking is more common in the low socioeconomic and less educated populace particularly in the field laborers who believe nicotine makes them alert and ease fatigue in order to do more work to enhance more pay.[6] Some indulge in smoking as a way of letting off mental stress, due to poverty as a result of failure to achieve a goal.[6]

Gender:

Recently in the west despite male dominance there has been an increase in smoking habit among female adults in high schools and college institutions which is basically due to pair smoking, exposure to western movies, role of parent smoking, self concept coupled with the freedom the female child gets when she is away from home where parental supervision is less attainable. Many have the mind of quitting after graduation, but become addicted eventually, however overall there is still male predominance in smoking in most societies with some up to 95% male dominance recorded.[7,8]

Environmental exposure

The health hazard due to exposure to environmental tobacco smoke (ETS) are increasingly established, ETS contains thousands of chemicals including 43 known carcinogens[9].

Other determinants are family problems, lack of knowledge, siblings and friend's use.[7,8]

TYPES OF TOBACCO AND MODE OF ABUSE

1. Cigarette: This is the most widely used form of tobacco, partly due to its wide availability and affordability thereby making it more accessible to all socio-economic class. It is a fine cylinder of finely cut tobacco leaves rolled in thin paper for smoking. Most modern manufactured cigarette also called ready rolls, is filtered and includes reconstituted tobacco and other additives.[10] Its absorption through the oral mucosa is almost impossible and so smokers have to inhale into the lungs in order to achieve the desired effect, where it causes more harm to the airways [11].
2. Moist snuff: This is another form of tobacco which is widely use in sub-Saharan Africa. It is finely grounded or shredded, moistened smokeless tobacco leaf, it is also referred commonly and idiomatically as dip and sometimes as rub, other terminology used to describe moist snuff include: chew, chaw, daps, bacca and spit tobacco.[12] The shredded tobacco is easily introduced into the oral cavity. It could also be deposited in the cheek and gum resulting in gum disease and oral cancers.[13]
3. Snuff: It is also a smokeless tobacco made from ground or pulverized tobacco leaves, it is insufflated or snuffed into the nasal cavity, into each nostril, delivering a swift hit of nicotine. Traditionally it is sniffed or inhaled lightly after a pinch of snuff is placed onto the back surface of the hand. This has the effect of causing inflammation of the nasal mucosa, loss of intra nasal hair and loss of secretion of sebum which lubricates the skin layer within the upper airway. All this culminate in the loss of external defense mechanism of the upper airway thereby rendering it more susceptible to upper respiratory tract infections, intense inflammation and mutation of the cellular lining.[14]
4. Beedi: This form of tobacco is a thin cigarette filled with tobacco flakes wrapped in a paper or “temburni” leaf, it is a traditional method of tobacco use throughout south Asia and part of the middle east.[15] Today beedies are popular and inexpensive and its use tend to be associated with lower social standing.[16] Its use is associated with high nicotine, carbon monoxide and tar content and carry greater risk of chronic gum disease and can result in oral cancer.[17]
5. Chatta: This is a coarsely prepared medium size particle which is wrapped in a dried tobacco or jack fruit leaves. It is smoked with the burning end inside the mouth. Its use is common in parts of India and Latin America e.g. Peru.[3]
6. Chillum and sulpha: Both are clay pipes in which dried tobacco leaves is pounded and is smoked. Chillum smoking is practiced in parts of India and Sulpha smoking in parts of rural Nepal. The culture of owning and smoking a chillium has spread from India to the rest of the world.[3]
7. Sisha, Hooka, Goza and Hubble: Tobacco mixed with molasses is burnt and the smoke is passed through water before inhalation. The hubble-bubble apparatus is usually passed round from person to person in a form of group smoking making transmission of respiratory tract infections a risk. Hookah smoking is prevalent in Bangladesh, India, Nepal and Pakistan; Goza smoking is

prevalent in Egypt and the countries of South-West Asia.[3]

8. Smokeless Tobacco and e-cigarette: These are new products and have become increasingly popular in the western world over the last few years; they produce vapour from nicotine dissolved in propylene glycol or glycerine. Low levels of contaminating chemicals have been found in them but e-cigarettes are almost certainly far safer than smoking cigarettes. E-cigarettes may have the potential to help smokers escape their deadly addiction to tobacco, however more evidence is needed to prove this – there have only been a few small trials so far. And these new products are not regulated yet, so they can vary significantly in quality and any possible long-term risks are not known. At the moment there is great debate about the wider impact of e-cigarettes. They could potentially save millions of smokers by providing the option for them to swap to a less harmful product. Or they could be used along with cigarettes and sustain addiction or renormalize smoking.

All forms of burnt tobacco generate toxic and carcinogenic smoke similar to that of cigarette smoke. The difference in disease consequence on tobacco use relates to frequency of use and depth of inhalation.[18] The risk of upper airway cancer is similar among cigarette and cigar smokers while those who have smoked only once have a much lower risk of lung cancer, heart disease and chronic obstructive pulmonary disease.[19] It suffices to know that cigarette smokers who switch to pipes or cigars tend to inhale the smoke thereby increasing their risk of exposure and it is likely that comparable inhalation and frequency of exposure to tobacco smoke from any of these forms of tobacco use is associated with a resurgence of cigar and bidi use among adolescents of both genders, raising concern that these older forms of tobacco are once again causing a public health concern.[17]

LOWER TAR AND NICOTINE CIGARETTE: The toxicity of cigarette smoke is contained in the tar while nicotine is the principal additive agent in cigarette, it has been suggested that cigarette that delivers low tar and nicotine to the smoker might not be safer.[20, 21] There is 10-20% reduction in risk of developing lung cancer when tar yield is reduced by 75% and this holds if there is no compensatory increase in intensity of smoking and is not likely to reduce cancer risk for sites other than lungs, larynx, esophagus and mouth.[22, 23]

BIOCHEMICAL CONTENT OF TOBACCO: Un burnt tobacco contains nicotine, carcinogen and other toxins capable of causing gum disease, lungs and oral cancer, when tobacco is burnt, the resultant smoke contains, in addition to nicotine, carbon monoxide and 74,000 other compounds that result from volatization, pyrolysis and pyrosynthesis of tobacco products.[1] The smoke is composed of a fine aerosol, with a particle size distribution predominantly in the airways and alveoli surfaces of the lungs, and a vapor phase. The bulk of the toxicity and carcinogenicity of the smoke resides in the aerosolized and particulate phase, which contains a large number of toxic constituents that has over 40 carcinogenic compounds.[1]

The aggregate of particulate matter after subtracting nicotine and moisture, is referred to as tar. The vapor phase contains carbon monoxide, respiratory irritants, and ciliotoxins as well as many of the volatile compounds responsible for the distinctive smell of cigarette smoke.[1] The alkaline Ph of smoke from blends of tobacco used for pipes and cigars tend to prevent the smoke inhalation into the lung, confining the toxic and carcinogenic exposure largely to the upper airways for most users of these products.[1] The acidic Ph of smoke generated by the tobacco used in cigarettes dramatically reduces absorption of nicotine in the mouth necessitating inhalation of the smoke into the larger surface of the lungs, in order to absorb enough quantities of nicotine sufficient to satisfy the smoker's addiction. The shift to using tobacco as cigarette, with resultant increased deposition of smoke in the lung has increased the epidemics of heart disease, lung diseases and lung cancer that dominate the current disease manifestations of tobacco use.[1, 19]

EFFECT OF TOBACCO ABUSE ON HEALTH

Over 400,000 individuals die prematurely each year in the United States from cigarette use. Approximately 40% of cigarette smokers will die prematurely due to cigarette smoking unless they quit.[1, 24] In Africa the effect of tobacco appears much earlier and its more devastating probably because of low socio-economic status, poor health facilities, lack of good health insurance policies among others.[25] All of these leads to the adverse effect of tobacco in the developing countries.[25] The incidence of smoking related disease is proportionately greater in younger than in older smokers, particularly for coronary artery disease and stroke.[8, 26,] At older age the rate of disease in non smokers increase, diminishing the fractional contribution of smoking and the relative risk. However, absolute excess rates of disease compared to non smokers increase with increasing age. The organ damage caused by smoking and the number of smokers who die from smoking are both greater among elderly as one would expect from the process of cumulative injury.[1, 27]

CARDIOVASCULAR DISEASE: Cigarette smokers are more likely to develop large vessel atherosclerosis

as well as small vessel disease. Approximately 90% of peripheral vascular disease in non diabetic population can be attributed to cigarette smoking.[1,28] And approximately 10% of exclusive cerebrovascular disease are caused by cigarette smoking.[1] Cigarette smoking acutely exert an hypertensive effect, mainly through stimulation of sympathetic nervous system, hypertensive smokers are more likely to develop severe form of hypertension, including malignant and renovascular hypertension .[29] There is a multiplier effect between cigarette smoking and other cardiac risk factors, such that the increment in risk produced by smoking among individuals with hypertension or elevated serum lipids is greater than the increase in risk produced by smoking for individuals without this risk factors.[1,29]

Cigarette smoking also increases the likelihood of myocardial infarction (MI) and sudden cardiac death by promoting platelet aggregation and vascular occlusion, the cessation of smoking has been shown to be accompanied by a reduction in a new coronary event in those that have survived the first MI.[1] This could also explain the high rates of graft occlusion among those who continue to smoke following vascular by pass surgery for cardiac or peripheral vascular disease, as well as the high rate of failure of angioplasty procedure among continuing smokers.[1, 2, 4] Cessation of cigarette smoking reduces the risk of second coronary event within 6-12 month after quitting, and rates of first myocardial infarction or death from coronary heart disease within the first few years after 15 years of cessation[1, 2]. The risk of a new myocardial infarction or death from coronary heart disease in those who have smoked in the past is similar to that in those who have never smoked.[1, 2, 4] The importance of smoking has been confirmed as the cause of acute myocardial infarction, about 50% of non fatal infarct can be attributable to cigarette smoking.[30]

RESPIRATORY DISEASES: Respiratory diseases such as COPD, Lung cancer, Pneumonia, Tuberculosis and Asthma has well been associated with cigarette smoking.[31] Cigarette smoking is responsible for >90% of chronic obstructive pulmonary disease (COPD) within 1-2years of beginning to smoke regularly, many young smokers will develop inflammatory changes which do not predict development of chronic air flow obstruction.[1,31,32] After over >20years of smoking pathophysiologic changes in the lungs develops and progresses proportionately to smoking intensity and duration. Chronic mucosal hyperplasia of the larger airways results in a chronic productive cough in as many as 50% of smokers over 66years.[1, 31] Chronic inflammation and narrowing of the small airways and / or enzymatic digestion of alveolar walls resulting in pulmonary emphysema can result in reduced expiratory airflow with reduction in FEV_1, FVC, AND FEV_1 / FVC sufficient to produce clinical symptoms of respiratory limitation in approximately 15% of smokers. Changes in the small airways of young smokers will reverse after 1-2 years of cessation, there may be a small increase in measures of expiratory airflow following cessation among individuals who have developed chronic airflow obstruction, The major change following cessation is a slowing of the rate of decline in lung function with advancing age rather than a return of lung function toward / normal.[1,32]

Although COPD can be the result of exposure to occupational hazards and air pollution across the world, cigarette smoking active or passive is the most commonly cited risk factor and is associated with a high prevalence of respiratory symptoms and lung function abnormalities, the two main types of COPD are Chronic bronchitis and Emphysema.[33] Cigarette smoking can cause cancer anywhere in the body.[31,34] And is one of the most important causes of Cancer of the lungs, larynx, oral cavity, esophagus, pancreas, kidney and urinary bladder.[1,3,33] There is also a growing evidence suggesting that cigarette may play a role in increasing the risk of cervical and stomach cancer.[34,35] However, there is conflicting evidence in the relationship of cigarette smoking and cancer of the breast, but overall there does appear to be slight causal link, more research is needed before a solid conclusion can be made about a potential link.[36] There is increasing number of uterine cancer among post menopausal women who smoke.[34] The risk of cancer increases with the increasing number of cigarette smoked per day and the duration of smoking (usually quantified as pack years).[1,32] There appears to be a synergistic interaction between cigarette smoking and alcohol use for cancer of the oral cavity, esophagus and possibly lung cancers.[37] Several occupational exposures also synergistically increase lung cancer risk among cigarette smokers, most notably occupational asbestos and radon exposure[31]. Cessation of smoking reduces the risk of developing cancer relative to continuing smoking, but even after 20 years of cessation, there is a modest persistent increase of developing lung cancers.[1,31,32]

PREGNANCY: Cigarette smoking is associated with several complication of pregnancy: premature rupture of membranes, abruptio placentae and placenta previa; there is also a small increase in the risk of spontaneous abortions among smokers. Infants of smoking mothers are more likely to experience preterm delivery, have a higher prenatal mortality, and most times are small for their gestational age, they are more likely to die of sudden infant death syndrome, bronchiolities and appear to have a development lag for at least the first several years of life.[1,38]

OTHER CONSIDERATIONS: Smoking delays healing of peptic ulcers; increase the risk of osteoporosis, senile cataract and macular degeneration, it also results in premature menopause, wrinkling of the skin, gallstones and cholecystitis in women and impotence in males.[1,39]

ENVIROMENTAL TOBACCO SMOKE: Prolonged exposure to a second hand tobacco smoke, increase the risk of lung cancer and coronary artery disease among non smokers. It also increases the incidence of respiratory infections, chronic otitis media, and can lead to asthma exacerbation and a probable cause in children.[1,7]

PHARMACOLOGICAL INTERRACTIONS: Cigarette smoking may interact with a variety of other drugs in a way that may have clinically significant implications it can affect drug therapy by both pharmacokinetic and pharmacodynamics mechanism, enzyme induced by tobacco smoking may also increase the risk of cancer by enhancing the metabolic activation of carcinogens.[1,43] Polycyclic aromatic hydrocarbon cigarette induce the cytochrome P450 (CYP) system which may affect the metabolic clearance of variety of drugs such as theophylline, propranalol, haloperidol, imipramine, pentazocin, flecanide , estradiol, heparine, caffeine, tocrine.[40] This effect may result in more drug toxicity among non smokers on a fixed drug dosage schedules and inadequate serum levels in smokers as outpatients, when the dosage is established in the hospital under non smoking conditions. Although the inhibitory effect of the smoke constituents carbon monoxide and cadmium on CYP enzyme has been observed in invitro and animal model studies, the relevance of this inhibition in humans has not yet been established. The mechanism involved in most interactions between cigarette smoking and drugs involves the induction of metabolism.[43] Correspondingly serum levels may rise when smokers may also have higher first pass clearance for drugs such as lidocane, and the stimulant effects of nicotine may reduce the effect of benzodiazepines or beta blockers.[1,40]

CESSATION OF SMOKING: The process of stopping smoking is often a cyclical one, with the smoker making multiple attempts to quit and failing before finally being successful, tobacco contains nicotine which is additive. Approximately 70 to 80% of smokers would like to quit smoking, about 13% of current smokers attempt to quit smoking each year and >90% of those unassisted quit attempts fail.[1,41] Smokers have been categorized into those who are not thinking about quitting (contemplation) and those who are in the action phase of quitting.[1] Smokers generally go through five successive stages in the process of quitting, each involving different issues and challenges, these are pre-contemplation- not thinking about quitting. Contemplation stage-thinking about quitting but not ready to quit. Preparation stage-getting ready to quit. Action-quitting and Maintenance-remaining a non-smoker.[1,41] A successful conceptualization of the cessation process is one where smokers cycle through the stages of cessation, each time smokers go round the cycle, a few more smokers become successful in their cessation effort.[1,41,42] One goal of clinician based smoking intervention lies between moving smokers from one stage of the cessation cycle to another, and effort can be focused in moving the smokers to the next stage rather than focusing exclusively on cessation.[6] The move from thinking about quitting to making a quit attempt is often triggered by a variety of environmental stimuli independent of physician control. The cost of cigarette can be a powerful trigger for cessation attempts.[1] Media campaigns, particularly when coupled with cessation events are also able to trigger cessation attempts in a large numbers of smokers. Changes in workplace rules, such as restricting smoking in the workplace have been associated with quit attempts in a substantial number of workers. Physician's can use the five A's frame work (ask, advise, assess, assist and arrange).[1,43] Physician's advice to quit particularly around an acute illness is also a powerful trigger for cessation attempts, with about half of patients who are advised to quit, making a cessation attempt.[1] Telephone counseling and nicotine replacement therapy are all useful enhancer of long term cessation. Successful clinic based cessation for those who can be recruited to participate, and a physician's recommendation can double the fraction of smokers who are willing to participate in these programme.[1] Other modalities are: Cut down to quit-gradual reduction which slowly reduces ones daily intake of nicotine. Community intervention-multiple channels to provide reinforcement, support, and norms for not smoking has an effect on smoking cessation outcomes among adults.[44] Psychosocial approaches-group or individual psychological support can help people who want to quit, this form of counselling can be effective alone or in combination with medication. These counselling styles include, motivational interviewing, cognitive behavioral therapy, acceptance and commitment therapy.[45, 46] Self Help- Cochrane review found that self-help material such news group, nicotine anonymous, mobile phone, self-help books, spirituality, interactive web based and stand-alone computer programme may produce only a small increase in quit rates.[47] Biochemical feedback-This include various method which allow smokers to see the impact of their tobacco use, carbon dioxide (CO) concentration in the blood has direct correlation with CO in the blood known as percent carboxyhemoglobin, the demonstration of CO concentration to a smoker through noninvasive breath sample, this links the

smoking habit and physiological harm associated with smoking, this might aid in quitting.[47] Cotinine a metabolite of nicotine is present in smokers like CO, a cotinine test can serve as a reliable biomarker to determine smoking status, this could be tested through urine, blood, saliva or hair samples, with its only concern being it invasiveness.[48]These two methods can be used either alone or in combination, for instance in situation where abstinence verification needs additional verifications.[49] Other methods used include completion and incentives through quit and win promos, Health care system based intervention through advice by healthcare providers, substitute for cigarrete like e-cigarrete have all been found to have some degree of success in quitting smoking.[50]

PHYSICIANS INTERVENTION: There is a health care policy and research clinical guidelines for smoking cessation.[51] All patients should be asked whether they smoke, their past experience with quitting and whether they are currently interested in quitting.[1,54] Those who are not interested in quitting should be encouraged and motivated to quit, they should be provided with a clear, strong and personalized physician message that smoking is an important health concern, and offered the assistance. A quit date should be negotiated, usually not the visiting days but within few weeks and a follow up contact by office staff around the times of the quit date should be provided.[1,51] The American cancer society estimate that between 25 and 33% of smokers who use medicine can stay smoke-free for over 6 months.[42] There are a variety of nicotine replacement products including over the counter nicotine patch and gum, as well as nicotine nasal and oral inhalers available by prescription.[1,44] Drugs like clonidine and recently Bupropion which is an anti depressant drug have also been shown to be effective in smoking cessation.[1,44,42] Some evidence supports the combination of nicotine replacement therapy and anti depressants. Nicotine replacement therapy is provided in different dosage for use with smokers of different number of cigarette smoked per day.[42] Anti-depressant is more effective in those with a history of depressive symptoms. A more comprehensive approach with pharmacological assistance and counseling can increase cessation success of smokers by three folds.[1, 42] the following changes are essential in institutionalizing smoking intervention with medical practice.[1]

1. Asking of smoking habit and interest in cessation
2. Listing smoking as part of the problem in the medical record.
3. Automating follow up with patient on their quit date.

CONTROL OF TOBACCO SMOKING

Raising tobacco taxes is the most effective and cost-effective strategy for reducing tobacco use. The effectiveness of tobacco tax increases is enhanced when implemented as part of a comprehensive tobacco control strategy[53,56]. Increasing tobacco taxation has been classified as a "phenomenal" intervention within the framework of the Sustainable Development Goals. WHO estimates the cost of implementing and administering tobacco tax increases at US$ 0.05 per person per year in low- and middle-income countries, making it the least costly of all tobacco control policies[54, 55, 56]. Not only is tobacco taxation cost-effective and efficient to implement, it also has the potential to generate significant additional revenue for governments to fund health programmes and other essential services. Evidence from a growing number of countries shows that tax increases that lead to a 10% rise in retail tobacco product prices will cut consumption by 2% to 8%. In low-income countries, for example, a doubling of excise tax per packet of cigarettes increases cigarette tax revenues by more than 30%[56]."

The Framework Convention on Tobacco Control by The World Health Organization, (WHO) have made specific commitments to implement strong tobacco control policies as an important means of providing that protection. WHO introduced the "MPOWER" package[56] of measures to assist in the country-level implementation of effective interventions to reduce the demand for tobacco. The six components of MPOWER are: monitor tobacco use and prevention policies; protect people from tobacco smoke; offer help to quit tobacco use; warn about the dangers of tobacco; enforce bans on tobacco advertising, promotion and sponsorship and raise taxes on tobacco[56].

Today, more than half of the world's countries, with nearly 40% of the world's population (2.8 billion people), have implemented at least one MPOWER measure to the highest level of achievement and many countries have implemented multiple MPOWER measures to the highest level[56].

The Nigerian government has spent N5, 500,000 on tobacco control based on recent report[55]. The Nigerian government has implemented some aspect of the MPOWER. Below is the summary of the level of implementation of the MPOWER

1. Monitor tobacco use and prevention policies: There exist recent and representative data from either adults or youth
2. Protect people from tobacco smoke: Three to Five public places completely smoke free
3. Offer help to quit tobacco use: There exist some cessation services (at least one of which is cost-covered)
4. Warn about the dangers of tobacco: No warning services or small warning

5. Enforce bans on tobacco advertising, promotion and sponsorship: Complete absence of ban, or ban that does not cover national television, radio and print media
6. Raise taxes on tobacco: Less than 25% of retail price is tax. Nigeria tax 5% on tobacco product

The basic modalities of prevention of smoking in any country will depend on:

1. Self-Motivation: The desire of an individual not to smoke is based on his or her personal conviction of the health, social, religious and financial implications. The presence of this strong conviction in addition to other modalities has significant effect in preventing people from developing the habit of smoking.
2. Parents can set a positive example for their children by not smoking themselves, and keeping their homes smoke free and restriction on movies that portrays smoking as a social norm.
3. Government Policy-This will aim at making legislations that will discourage smoking habits and its manufacturing.

EDUCATION AND INFORMATION

Evidence about the harmful effects of tobacco use and the addictive nature of nicotine should be widely disseminated to the public.

Mass media summaries of published scientific and epidemiological research, warning labels on tobacco products, school and community programs, and counter-marketing strategies, have all been shown to be effective in varying degree.[56]

CONCLUSION

Cigarette smoking is addictive mainly because of the nicotine content, however in addition to nicotine tobacco which is the leaves used in making cigarette contains thousands of other chemical substance which can affect virtually every organ in the body with high morbidity and mortality rate.

Preventing smoking habit is the most important strategy to avoid its harmful effect, however quit smoking strategies, centered on patient motivation, physician approach, family support and government policies also plays significant role in reducing the harmful effect of cigarette smoking.

Raising tobacco taxes is the most effective and cost-effective strategy for reducing tobacco use. The effectiveness of tobacco tax increases is enhanced when implemented as part of a comprehensive tobacco control strategy. Tobacco taxation offers a 'win-win' policy option for governments, where raising tobacco taxes will both generate extra revenue to fund health programmes and other essential services and also reduce consumption.

REFERENCES

1. David M Burns. Nicotine Addiction Harrison's Principles of Internal Medicine 395; 19ed:
2. Asma S, George AM, Charles WW, Rosemarie H. Tobacco use and the cardiovascular epidemic in developing countries: Global crises and opportunity in making ethnicity and disease. 2003: (13); 81
3. Asma S, Blanton C, Erikson MP et al. Tobacco use among youth: A cross country comparison. Tab Control 2002:11;252-70.
4. Lawa JB, Pamela AF, Naomi B et al. Novel Gene Identified in high density genome wide association study for nicotine dependence. Human molecular genetics 2007 (16); 1: 24-35.
5. Lerman C, Barrettini W. Elucidating the role of genetic factor in smoking behavior and nicotine dependence. Am j med Genet B Neurpsychiatr2003(1)118;48-54
6. Taha A, Balla K. Smoking in Africa: The coming epidemic. World Smoking health 1982; 7(2): 25-30
7. Mardeline AD, James DS, Michael LB, Linder TE, Jennifer JB, et al. Effect of viewing smoking in movies on adolescent smoking initiation http: / / image.thelancet.com / extra / 03art135 web.pdf
8. Horn KA, Gao X, Dino GA, Kamal-Bahl S. Determinant of youth tobacco use in west Virginia: a comparison of smoking and smokeless tobacco use. An J Drug Alcohol Abuse 2000Feb; 26 (1):125-38. J midwifery women health 2003; (3); 48
9. Brownson RC, Erikson MP, Davis RM, Warne KE. Environmental Tobacco Smoke; Health effect and policies to reduce exposure. Ann Review public Health 1997; 18:163-85.
10. Wigard JS. Additives, Cigarette Design and Tobacco product regulation, A report to WHO, Tobacco free initiative, tobacco product regulation group, kobe, Japan June 2006.
11. Gori GB, Benowitz NL, Lynch LJ. Mouth versus deep airway absorption of nicotine in cigarette smokers. PhamacolBiochem Behar 1986; 25 (6): 1181-4
12. Gupta R, Gura H, Bathalomew JR. Smokeless Tobacco and cardiovascular risk. Arch Intern. Med164;(17):1845-9

13. LuoJ, Ye W, Zendehdel K, Adami j, Adami HO et al.Oral use of Swedish Moist Snuff (snus) and risk of cancer of the mouth, lung and pancrease in male construction workers, a retrospective cohort study. Lancet june 200716; 369(9578):2015-20.
14. Mc KimaT. Food of the Gods-The search for the original tree of knowledge- A radical history of Plant, Drugs and Evalution Bantam Book 1993:199
15. Yen, Karl L, Hachevarria, Elizabeth, Bostwick, Susan B. Bidi cigarette an emerging threat to adolescent health. Archive of pediatrics & adolescent medicine (Chicago Il:American Medical Association) 154(12):1887-89.
16. Rijo MJ, Kavita RR, Govinda RM, James M, DeshpandeRSet al. The economics of Tobacco and Tobacco Taxation in India 2010; ISBN 978-2-914365-70-3.
17. Mahbubur R, Junichi S, Tsuguya F. Beedi smoking and oral cancer: A meta analysis. International journal of cancer (Wiley-Liss, inc) 106 (4):600-4
18. Benowitz NL, Hukkanen J, Jacob P. Nicotine chemistry, metabolism, kinetics and biomarkers. Hand book of experimental pharmacology 2009; (192): 29-60
19. Paolo B, Goran P, Karl-Henz J, Francesco F et al. Cigar and pipe smoking and Lung cancer. J Natl cancer Inst. 1999; (91):697-701
20. Risk associated with smoking cigarrete with low Machine measured yield of Tar and Nicotine. Bethesda MD: National cancer Institute; 200. Smoking and Tobacco control Monogramme 13.
21. Andersan SJ, Ling PM, Glantz SA. Implication of the Federal court order banning the terms“light” and “mild”: what difference could it make? Tobacco control 2007; 16 (4): 275-79.
22. The Health consequence of smoking. The changing cigarette: A report of the Surgeon General, US Department of Human Services, Public Health Service, Office on Smoking and Health, 1981.
23. Doll R. Progress against cancer: an epidemiological assessment. The 1991 JohnC Cassel Memorial Lecture. Am J Epidemiol 1991;134: 675-88
24. Jonathan F, Cristine D, Douglas MZ, Michael BS. Health effect of Tobacco, Nicotine and exposure to tobacco smoke pollution. Handbook of the medical consequences of Alcohol and Drug Abuse 2001;13:423-59
25. Smoking and control strategies in developing countries report of world Health export committee WHO 1983 (Technical report series 695)
26. Doll R, Pet R, Boreham J, Sunderland I. Mortality in relation to smoking: 50years' observation on male British Doctors (Bir Med Jour 9clinical research ed) 2004; 328 (7455): 1519.
27. An L, Berg C, Klatt C, Perry C, Thomas J, Luo X et al. Symptoms of cough and shortness of breath among young adult smokers Nicotine and Tobacco Research 2009; 11 (2): 126-33.
28. Wolf PA, D' Agustino RB, Kannel WB, Bonita R, Belanger AJ. Cigaratte smoking as risk factor for stroke: The Farmingham study. JAMA 1988; 257 (7): 1025-9.
29. Virdis A, Giannarelli C, Neves MF, Taddei S, Ghiadoni L. Cigarette smoking and Hypertension. Curr pharm Des 2010; 16 (23): 2518-25.
30. Negri E, La Vecchia C, Nobili A, D' Avanzo B, Bechi S. Cgarette smoking and acute myocardial infarction. Euro J Epidemiol. 1994; 10 (4):36-6
31. Thun MJ, Carter BD, Faskanich D et al A 50-year trend of smoking related mortality in United state. N Engl J Med 368: 351-64.
32. Toshinori Y, Rubin MT. Pathobiology of cigarrete smoke – Induced COPD Phys Rev 2007; 87 (3): 1047-82.
33. MNWR Weekly report. Notice to readers world COPD day Nov 14 2007. 2007; 56 (44):1167.
34. Winkelstein W Jr. Smoking and cancer of the uterine cervix; hypothesis. Am J Epidemol 1977;106 (4): 257-9.
35. Tredaniel J, Boffetta P, Buietti E, Sarachi R, Hirsch A. Tobacco smoking and gastric cancer; Review and meta analysis. Int J Cancer 1997;72 (4): 565-73.
36. Gaudet MM, Gapstur SM, Sun J, Diver WR, Hannan LM, Thun MJ. Active smoking and breast cancer risk: cohort data and meta analysis J Natl cancer ins.2011;105 (8): 513-25.
37. Blot WJ, McLaughlin JK, Winn DM, et al. Smoking and Drinking in relation to oral and pharyngeal cancer. Cancer Research 1988; 48:3282-87.
38. Castles A, Adams EK, Melvin KL, Kelsch C, Boulton ML. Effect of smoking during pregnancy. Am J Med 1999; 16 (3): 208-15.
39. U.S. Department of Health and Human Services. The Health Consequences of Smoking-50 Years of Progress: A Report of the Surgeon General. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control_ and Prevention, National Center for Chronic Disease Prevention and Health_Promotion, Office on Smoking and Health, 2014 [accessed 2014 Feb 6].

40. Zevin S, Benowitz NL. Drug interaction with tobacco smoking. An update Clin Phamacokinet 1999;36 (6):425-38.
41. Benowitz NL. Nicotine addiction N Engl J Med 362 (24): 2295-303.
42. Flore MC, Novotny TE, Pierce JP, Gionino GA et al. Methods used to quit smoking in the united state. JAMA 263(20):2760-5.
43. Michele ML, Dave EW. Promoting Smoking Cessation Am Fam Physician. 15; 85(6):591-598.
44. Secker-Walker RH, Gnich W, Platt S, Lancaster T. Community Intervention for reducing smoking among adults. Cochrane Database syst Rev (3): Cd001745.
45. Perkins KA, Conklin CA, Levine MD. Cognitive behavioral therapy for smoking ceasation: A practical guide book to the most effective treatment. Routledge ISBN 978-0-415-95463-1
46. Ruiz FJ. A review of acceptance and commitment therapy (ACT) empirical evidence: Correlational, experimental psychopathology, component and outcome studies. Intl Jour Psycho and psychological Therapy 10(1): 125-62.
47. Irving JM, Clark EC, Crombie IK, Smith WC. Evaluation of portable measure of expired-air carbon monoxide. Preventive Medicine, 17;109-15.
48. Florescu A, Ferrence R, Einarson T,Selbi P, et al. Methods of quantification of exposure to cigerrate smoking and environmental tobacco smoke therapeutic drug monitoring.31(1):14-30.
49. McClure JB. Are biomarkers useful treatment aid in for promoting health behavior change. Am J of Prev Med 22 (3); 200-7.
50. Hopkins DP, Briss PA, Riccard CJ, Husten CG et al. Review of evidence regarding intervention to reduce tobacco use and exposure to environmental tobacco smoke. Am J Prev Med 20 (2);16-66.
51. Martin R, Ann M, Robert W. Smoking cessation guideline for health care professionals. Thorax 1998;53:S1-S18doi:10.1136/thx.53.2008.S1.
52. Flore MC, Jaen CR, Baker TB, et al. Treating Tobacco use and dependence: clinical practice guideline US Dept of Health and Human Services 2011-02-16.
53. WHO report on the global tobacco epidemic, 2015: raising taxes on tobacco. Geneva: WHO 2015
54. The Economic and Health benefit of Tobacco Taxation. Geneva: World Health Organization. 2015
55. WHO report on the global tobacco epidemic, 2015: Nigeria country profile Geneva: WHO 2015
56. Frame work convention on Tobacco Control Technical Briefing series. Geneva: WHO 1999.