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Smoking Prevention and Cessation

Edited by Mirjana Rajer

SMOKING PREVENTION AND CESSATION

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Meet the editor

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Preface

This edited volume is a collection of reviewed and relevant research chapters concerning developments within the smoking prevention and cessation field of study.

The book includes scholarly contributions by various authors, edited by expert pertinent to field of pulmonology. Each contribution comes as a separate chapter but is directly related to the book's topics and objectives.

The book is divided into two sections: Effects of Smoking and Smoking Prevention and Cessation Counselling.

The first section includes chapters dealing with pulmonary effects of passive smoking among adults, effects of smoking on oxidative stress and vascular function and smoking habit and nicotine effects, while chapters on smoking cessation counselling and evaluation of effectiveness of peer education on smoking behaviour are included in the second section of this book.

The target audience comprises scholars and specialists in the field.

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Section 1

Effects of Smoking

Chapter 1

Pulmonary Effects of Passive Smoking Among Adults

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Ruxandra-Mioara Rajnoveanu and
Ruxandra Ulmeanu

Additional information is available at the end of the chapter

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Abstract

Passive smoking, also known as environmental tobacco smoke (ETS) or second-hand smoke (SHS) represents the involuntary inhaling of tobacco released by others in the ambient air. Passive smoking exposure occurs in homes, workplaces, and in other public places such as bars, restaurants, and recreation venues. It consists of a complex mixture of mainstream smoke exhaled by smokers and the smoke given off by the combustion of tobacco products. Non-smokers, being exposed to the same toxic substances as identified in mainstream tobacco smoke are, therefore, at an increased risk for serious adverse health effects. Although attention has centered mainly on the harmful effects of SHS exposure in the pediatric population, epidemiologic data from the last 20 years showed increased risks on various respiratory pathologies of the adult. Inhaling SHS causes injury to the respiratory tract, resulting in a high prevalence of respiratory symptoms, asthma, impairment of lung function and increased bronchial responsiveness. In adults, passive smoking is also associated with an increased risk of lung cancer, especially in those with high exposure. On the basis of recent publications, we propose a review of history, biologic basis and effects on different respiratory pathologies of the exposure to SHS in adults.

Keywords: passive smoking, secondhand smoke exposure, tobacco, respiratory diseases, lung cancer

1. Introduction

Tobacco smoke is one of the major health hazards in the general population. Although the large body of research findings has indicated the serious health effects of tobacco in smokers, there is substantial evidence accumulated showing that non-smokers, exposed to tobacco smoke, are also at an increased risk for several chronic diseases, including lung cancer [1, 2].

Passive smoking, also called second-hand smoking (SHS), or environmental tobacco smoke exposure, represents the inhalation of smoke that has been exhaled, or breathed out, by the other person smoking. Most frequent exposure to passive smoking occurs in homes and workplaces [1]. People can be also exposed in public places, such as in bars, restaurants, and recreation venues, as well as in cars and other vehicles [3].

SHS is a complex mixture containing many toxicants, part of them emitted from the smoldering tobacco between puffs, sidestream smoke (SS), and the other part exhaled from the smoker, mainstream smoke (MS) [4]. These components are the same as those that have been identified in MS, over 70 of them being known or suspected human carcinogens [3, 4]. Non-smokers, being exposed at the same toxic substances as identified in mainstream tobacco smoke are, therefore, at an increased risk for serious adverse health effects. Since 1964, more than 2,500,000 deaths among nonsmokers have been caused by exposure to passive smoking [3]. Passive smoking is responsible for significant effects on the health of children, increasing the number and severity of asthma attacks, of respiratory and middle ear infections, and it increases the risk of sudden infant death syndrome [2, 3]. In adults who have never smoked, SHS can cause lung cancer, coronary heart disease, stroke and various respiratory pathologies [1–4]. SHS exposure is responsible for approximately 3000 deaths per year from lung cancer [1]. On the basis of recent publications, this chapter will review the epidemiology, biologic basis, and effects on different respiratory pathologies of the exposure to SHS in adults.

Research reviewed in this article indicates that the level of exposure to SHS has steadily decreased in the United States and most of the European countries over time [5, 6]. This decrease is likely due to the significant declines of smoking prevalence, the growing number of countries with enforced laws to protect non-smoking people against exposure to tobacco smoke (not allowing smoking in indoor areas of workplaces and public places), and the decreased level of the social acceptance of smoking around non-smokers [2, 6].

Despite the large implementation of smoke-free policies, many people are still exposed to tobacco smoke, the level of exposure depending on race and ethnicity, income and occupation, with large differences between different goups [2–4]. In this chapter, the most important actions regarding protection from exposure to second-hand smoke will be discussed in detail.

2. Passive smoking in adults

2.1. Environmental tobacco exposure prevalence

While the literature comprise a large number of statistics on the prevalence of smoking around the world, the magnitude of exposure to SHS among adults, and which socio-demographic groups are at greatest risk, is more difficult to estimate. According to the WHO [7], a majority of the world population (93%) still live in countries not totally covered by smoke-free public health regulations, and exposure to SHS in homes or public places is still common. Globally, among adult non-smoking population, 33% of males, and 35% of females, are regularly exposed to the harmful effects of SHS [8]. The level of exposure varies widely, ranging from 13% in Africa to more than 50% in Eastern Europe or in Western Pacific [7, 8].

Centers for Disease Control and Prevention from the United States reported approximately 58 million (25.3%) non-smokers exposed to SHS in 1 year (2011–2012), down from 52.5% during 1999–2000 [9]. Important differences have been reported between racial and ethnic subgroups, the highest second-hand exposure being encountered among non-Hispanic black (46.8%) compared with non-Hispanic white non-smokers (21.8%) [2]. Data from the European Commission estimated that 28% of European people were exposed to tobacco smoke in bars in 2012, down from 46% in 2009 [6]. In Canada, 27% of nonsmokers self-reported recent SHS exposure [10].

Public places and the workplace were the most frequently self-reported sites of tobacco smoke exposure, but passive smoking was commonly reported in private locations such as personal homes, homes of friends or relatives, and vehicles [2–6].

Tobacco smoke exposure is higher among people with low education and income level. Nearly half (43.2%) of non-smoking adults who lived below the poverty level were exposed to SHS [9]. Disparities in environmental tobacco smoke exposure related to occupation decreased over the past 20 years, but important differences are still recorded. High levels of SHS exposure exist for some groups, including construction an extraction workers, installation maintenance, and transportation workers [9, 11].

2.2. Burden of disease caused by second-hand smoke in adults

Evidence of the harmful effects of passive smoking have been recorded since 1928, when Schonherr suspected that exposure of non-smoking wives to husbands' tobacco smoke, could cause lung cancer [12]. Since then a large body of research findings about adverse health effects of SHS has appeared. The direct link between passive smoking exposure and disability, specific diseases and death has been documented in publication by many international health organizations [1–3, 7]. Furthermore, it has been recognized that there is no risk-free level of exposure to tobacco smoke; even short exposures can be harmful to health [2, 13]. The health effects attributable to SHS in adults have been quantified as deaths and disability-adjusted life-years (DALYs) lost [7, 8].

Passive smoking was estimated to have caused 603.000 deaths in 2004, corresponding to 1.0% of worldwide mortality [7]. Of these, 35.800 deaths were attributed to asthma and 21.400 to lung cancer that occurred in non-smoking adults. As a result of deaths caused by tobacco exposure, 6.6 billion dollars were lost in productivity [14]. More deaths attributable to SHS exposure, with 281.000 (47%) per year, occurred in women, compared with 156.000 (26%) in men [7, 8]. This situation could be explained by the fact that the absolute number of non-smoking women is approximately 60% higher than that of non-smoking men [13]. Second, in many parts of the world, such as Africa, the Eastern Mediterranean, Southeast Asia, and some parts of the Americas, women are at least 50% more likely to be exposed to passive smoking than are men [8].

The evaluation of the burden of disease, quantified as DALYs lost because of exposure to SHS, revealed a number of 10.9 million DALYs lost that occurred worldwide, in 2004, corresponding to 0.7% of the total worldwide burden of diseases in DALYs [7, 8]. A significant part (1.246.000) was due to asthma in adults.

Considering all these adverse health effects, passive smoking is considered to be the third most frequent risk factor for avoidable deaths, after active smoking and inadequate dietary intake [15, 16].

3. Respiratory effects of passive smoking in adults

Passive smoking causes many of the same diseases such as direct smoking, including lung cancer, respiratory and cardiovascular diseases [1–5]. This chapter focuses on the respiratory health outcomes of involuntary smoking in adults, such as respiratory symptoms, pulmonary function, and respiratory diseases (**Table 1**), detailing chronic obstructive diseases and lung cancer.

3.1. Pathophysiology

Second-hand smoke is formed mainly (85%) of sidestream smoke (SS), which is the product of incomplete combustion, released from the burning tip of a cigarette, and only a small part (15%) of mainstream smoke (MS), which is exhaled by smokers [17]. The diameter of SS components is 10 times smaller compared to the particle diameter of MS and, as such, SS components have the potential to reach the most distal alveoli from where they cannot be expelled easily [17, 18]. The composition of environmental tobacco smoke consists of a complex mixture of more than 4000 chemical substances, with proinflammatory and cytotoxic effects [19].

Several experimental studies conducted on animals [20–22] and humans [23, 24] showed relevant evidence and information on the underlying mechanisms for the effects of passive smoking on the respiratory tract. Pulmonary emphysema, including the loss of elasticity in the lung tissue, was induced in rats through a 3 months exposure to tobacco smoke (mean CO at 35 ppm) for 90 min per day [20]. Alterations of airway defense mechanisms and enhanced allergic inflammatory responses has also been observed as a response to SS exposure [21, 22]. High levels of total serum immunoglobulin E (IgE) were observed in adult population exposed to cigarette smoke [25], although not all studies found this association [26]. IgE enhanced values might be associated with the later development of allergies [27]. The increase in susceptibility to allergic diseases may also be explained by the depletion of Thl cytokine-secreting cells in the human airway caused by cigarette smoke exposure [22].

The cigarette smoke induces increases of neuroendocrine cells in the lung, which synthesize and release bronchoconstriction mediators, responsible for bronchial hyperreactivity and asthma [26]. The stimulation of C-fibers by components of SHS including nicotine, acrolein,

Respiratory symptoms	Respiratory illnesses	Pulmonary function
Cough	Asthma	Declines in FEV1 levels
Phlegm production	COPD	Increased bronchial responsiveness
Wheeze	Exacerbation of cystic fibrosis	
Chest tightness	Allergies	
Dyspnoea on exertion	Bronchitis	
Shortness of breath at rest	Pneumonia	
Nocturnal breathlessness	Bronchiolitis	
	Lung cancer	

Table 1. Environmental tobacco smoke-related respiratory effects in adult population.

and oxidants can trigger intense respiratory responses through local and central nervous system reflexes, inducing mucous secretion, bronchoconstriction, and increased microvascular leakage [28]. In vivo and in vitro studies have demonstrated the inhibitory effect of cigarette smoke on exhaled nitric oxide (NO) [24, 29]. The inhibition of NO synthesis increases bronchial response in persons exposed to SHS [24]. Bronchial hyperreactivity may also result from smoke-induced inflammation [24, 29]. The outcome of several studies showed an increase in circulating PMNs, significantly greater PMN chemotactic activity, and the augmented release of oxidants upon stimulation, in nonsmokers exposed to SHS [23, 30].

Exposure to passive smoking increases susceptibility to respiratory infections and/or worsens infections through the inhibition of antibody responses, impairing macrophage responsiveness and mucociliary clearance, increasing bacterial adherence, and disruption of the respiratory epithelium [31, 32].

3.2. Non-malignant respiratory health effects

In the last years, there has been published more information regarding the causal relationship of passive smoking with acute and chronic respiratory health effects in adults.

3.2.1. Respiratory symptoms

In several large cross-sectional population studies, SHS exposure was found to be an independent risk factor for different respiratory symptoms, including wheezing, cough, dyspnea, nocturnal breathlessness and shortness of breath at rest [26, 33–36]. The outcome of all studies strengthened the evidence of a causal effect by a significant dose effect relationship between the amount of smoke exposure and the possibility of having symptoms. Among different irritants, such as car exhaust fumes, cold air and strong smells, passive smoking was the most often identified by the subjects as causing lower airway symptoms [34]. Data from a study involving 2335 never-smoking Italian women, of which, 73% reported lifetime SHS exposure, showed that the combined exposure both to the husband and at work was a significant risk factor for each health outcome. Current dyspnea was the most frequently (26.9%) reported health outcome [36]. A recent study has provided the evidence of significantly increasing the risk of persistent respiratory symptoms into young adult life if there was exposure to parental smoking during childhood [35].

3.2.2. Lung function and bronchial responsiveness

Studies of the relation between passive smoking and lung function have generally found a harmful effect of such exposure [26, 33, 37–39]. The extent of this impairment is, however, relatively low, with a decrease of forced expiratory volume in 1 s (FEV1) in the range of 50–100 mL in subjects with the highest level of exposure compared to unexposed subjects [37]. Some studies have shown that passive smoking is associated with decreased lung function suggestive of airflow limitation (FEV1 and FEV1/FVC) in female but not male non-smokers [33, 38]. Women with middle level exposure had on average 88 mL reduction of FEV1 and 2% for FEV1/FVC compared to those with low exposure [33]. This gender-specific susceptibility of lung function impairment in women can result from the fact that most studies usually

involve a small number of men non-smokers. The results of the European Community Respiratory Health Survey (ECRHS) involving a large adult population (18.992 subjects) from 17 European countries, have demonstrated a positive association between exposure to parental smoking in childhood and impaired lung function in adult lifetime [39]. Regarding the short-term passive smoking effect on lung function in asthmatic adult population, the report of the Surgeon General concluded that the evidences are suggestive but not sufficient to infer a causal relationship [40]. The impairment of pulmonary function among asthmatic adults, particularly in asthmatic women, was demonstrated in The Swiss Study on Air Pollution and Lung Diseases in Adults (SAPALDIA), where the duration of environmental smoke exposure at work was associated with a decrease in lung function (FEV1–6% per hour of smoke exposure at work ($p = 0.01$); FEF25–75%: -3.4%/h ($p < 0.05$)) [41].

Association between passive smoking in adulthood and increased bronchial responsiveness has been shown in both epidemiological [26] and experimental studies [42, 43]. Adults with asthma have even greater bronchial hyperresponsiveness after SHS exposure [39, 44].

3.2.3. Asthma

A large number of published studies have examined the links between involuntary smoking and new-onset asthma and the exacerbation of pre-existing asthma among adults [10, 19, 33, 36, 38, 41, 44–54]. The causal relationship between SHS exposure and self-reported or physician diagnosis of asthma has been observed in a large variety of study designs, including case–controlled, cross-sectional, and cohort studies, involving different populations from different countries around the world [45].

Passive smoking exposure during childhood has been shown to lead to an increased prevalence of asthma diagnosis in never-smoking adults, especially those without any family history of asthma [19]. In a large population-based Swedish study involving over 8000 never-smoker adults, the prevalence of physician-diagnosed asthma was more frequently reported among subjects who reported environmental tobacco smoke exposure during childhood (7.6%), compared with unexposed persons (5.8%) ($p = 0.035$) [46]. Another study also found a similar pattern of results, with a 1.39-fold increase of asthma diagnosis in the never-smoking adults with childhood exposure to passive smoking [47].

Numerous studies assessed the causal relationship between the whole lifetime exposure to passive smoking and new-onset asthma in adults. Increased risk of asthma was observed among non-smokers adults exposed to passive smoking home only [48–50], at work only [51, 52], and to both types of exposures, home and workplace [10, 33, 36, 41]. Home exposure to parental smoking was related to an increased risk of self-reported asthma in young adulthood (OR 1.8; 95% CI 1.1–3.0 for maternal smoking, OR 1.6; 95% CI 1.1–2.4 for paternal smoking, respectively) [48]. The results of another population-based case–controlled study have also demonstrated the strong association between home exposure to passive smoking and adult-onset asthma but with a higher risk among males (OR 4.8; 95% CI 2–11.6) than females (OR 1.5; 95% CI 0.8–3.1) [49]. An Indian population-based cross-sectional study involving 62,109 never-smoking adults showed a higher risk of having asthma among subjects who reported both childhood and home exposure (OR 1.69; 95% CI 1.38–2.07) [50].

Regarding workplace exposure, it has been shown that daily exposure to passive smoking (more than 5 h) increases the risk of physician-diagnosed adult asthma (OR 1.79) [34]. The duration of working with a smoker is directly related with an increased risk of developing asthma (OR 1.5 per 10-year increment; 95% CI 1.2–1.8) [51, 52], but a population-based case-controlled study from Finland has shown a strong association between new-onset adult asthma and passive smoking exposure during only the preceding 12 months of asthma diagnosis (OR 1.97; 95% CI 1.19–3.25), with evidence of an exposure response relationship (OR 1.33 per 10 cigarettes per day; 95% CI 1.02–1.75) [53].

Current evidence suggest that passive smoking in asthmatic subjects is related to a negative effect on prognosis, with an increased risk of exacerbations and hospitalization, pulmonary function impairment, and a decrease in the quality of life [37, 45]. Extended data from literature, including studies [10, 36, 38, 41, 44, 54] and reviews [37, 40, 45] strongly link passive smoking with poorer asthma status in adults diagnosed with this condition.

3.2.4. Chronic obstructive pulmonary disease

Compared to asthma, the causal association between passive smoking exposure and chronic obstructive pulmonary disease (COPD) has been less widely the object of epidemiological studies [16, 55–59]. The relative risk (RR) for the association between SHS exposure and COPD, calculated in a recent published meta-analysis [60], based on cohort and case-control studies (shown in **Table 2**), was 1.66, with a comparatively small confidence interval (95% CI: 1.38–2.00). A higher RR of 2.17 (95% CI: 1.48–3.18) was obtained for women, than for men, of which RR of 1.50 (95% CI: 0.96–2.28) was inherited from one single study [55].

In a large population-based study conducted in Germany [16], whose of aim was to quantify the adverse health effects attributable to passive smoking, the results indicated that 231,973 persons were affected by COPD caused by SHS exposure in 2014, representing 0.35% of the male and 0.22% of the female German population. The higher prevalence of COPD cases attributable to passive smoking occurred in the age group 60 years and older (1.15% of men and 0.55% of women).

Similar to the impact of passive smoking on asthma evolution, several studies have shown the worsening effect of SHS exposure on COPD patients [56–58]. In longitudinal analysis, the highest level of tobacco exposure, measured by cotinine urine level, was associated with worse COPD severity [56]. Another publication observed an increased risk of dying from COPD in never-smokers exposed to SHS [57]. Passive smoking in COPD patients was also associated with increased hospital readmission rates [58].

3.3. Lung cancer

A major shift in policy approaches to controlling tobacco occurred in the late 1980s, with the rise of new evidence that SHS causes death and disease in non-smokers [59], especially after 1981, when Hirayama's study demonstrated that non-smoking wives of smokers were almost twice as likely to die of lung cancer compared with non-smoking wives of non-smokers [68]. In 1986, the US Surgeon General, the International Agency for Research on Cancer, and the US

Authors	Type	Participants	Exposure	Relative risk (95%CI)
Kalandidi et al. [61]	Case–control	103 female patients, 179 female controls, never-smokers	home: spousal smoking (1–20 cigarettes/day)	women: 1.79 (1.17–2.57)
McGhee et al. [55]	Case–control	4838 cases (2680 men, 2158 women) 763 controls (418 men, 345 women), never-smokers	home	1.81 (1.24–2.65) men: 1.50 (0.96–2.28) women: 2.59 (1.30–5.27)
Chan-Yeung et al. [62]	Case–control	289 patients (243 men, 46 women) 289 controls (243 men, 46 women)	home and workplace	1.64 (0.97–2.03)
Yin et al. [63]	Cohort	15,379 (1777 men, 13,602 women) never-smokers	home: ≥ 5 years of 40 h/week	1.60 (1.23–2.10)
Schwartz et al. [64]	Case–control	562 female cases, 564 female controls	home	women: 1.68 (1.12–2.61)
Wu et al. [65]	Case–control	205 female cases 205 female controls	home and workplace	women: 3.12 (1.56–6.50)
Johannessen et al. [66]	Case–control	433 patients (258 men, 175 women) 325 controls (176 men, 149 women)	home	men: 0.98 (0.81–1.17) women: 1.14 (0.93–1.37)
He et al. [67]	Cohort	910 (439 men, 471 women) never-smokers	home and workplace	2.30 (1.06–5.00) men: 2.15 (0.86–5.39) women: 3.31 (0.69–15.82)

[1]Adapted from Fischer F et al. [60].

Table 2. Studies assessing the relative risk for the association between passive smoking exposure and COPD[1].

National Academy of Sciences concluded that SHS causes lung cancer in non-smokers [40]. Environmental tobacco smoke has been classified as a known (Group A) human lung carcinogen by the United States Environmental Protection Agency (EPA), since 1993 [2]. After this year, this risk factor was intensively studied in relation to lung cancer. At least 17 carcinogenic chemicals contained in tobacco smoke are emitted at higher levels in sidestream smoke than in mainstream smoke [40], with one like benzo(a)pyrene diol epoxide, which shows a direct etiological association with lung cancer, founded also in both mainstream and side stream smoke [69].

SHS exposure is associated with an excess relative risk of lung cancer of around 20% [70]. The results of the meta-analysis published in 2007 have shown a 27% excess in the risk of

lung cancer among never-smoking women exposed to spousal passive smoking, compared with not exposed never-smoking women [71]. Exposure at home seemed to have a stronger effect than exposure at the workplace, probably because exposure at home, especially from the spouse, is more likely to be of greater duration and intensity than exposure at work. Other results suggest that people with combined exposure (home and work) are more likely to develop lung cancer than those exposed at one location only [70, 72].

Specific and nonspecific pathways through which smoking causes cancer are common for passive and active smoking. There are DNA binding and mutations, but also epigenetic mechanisms. Tobacco-specific carcinogens form adducts and lead to mutations in oncogenes and tumor-suppressor genes [73].

A prospective study in 10 European countries, estimated the proportion of lung cancers in never- and ex-smokers attributable to passive smoking at 16–24%, mainly due to the contribution of work-related exposures [74].

On the overall population, exposure to SHS smoke increases the risk of lung cancer by approximately 30 and 60% for non-small cell lung cancer (NSCLC), and small cell lung cancer (SCLC), respectively [75]. Among never smokers, passive smoking increased the risk by approximately 30 and 200% for NSCLC and SCLC, respectively [74, 75].

Lung cancer in never smokers has been recognized as a distinct clinical entity apart from lung cancer in former/current smokers due to the initial observation of a significantly higher prevalence of adenocarcinoma (ADK), female gender, and advanced stage at presentation, but a better overall survival compared with lung cancer in ever smokers [76]. There is weak evidence that exposure to SHS increases the risk of lung adenocarcinoma in situ/minimally invasive adenocarcinoma [77].

The adjusted ORs for the association between passive smoking and lung cancer among never smokers were 1.26 (95% CI 1.10–1.44) for ADK, 1.41 (95% CI 0.99–1.99) for squamous cell carcinoma, 1.48 (95% CI 0.89–2.45) for large cell carcinoma, and 3.09 (95% CI 1.62–5.89) for SCLC [75].

There is no clear association between passive smoke exposure and somatic profile in lifelong, never-smoker lung cancer, even if EGFR and HER2 mutations and ALK rearrangement are known to be related to lung cancer in never-smokers, while KRAS, BRAF and PIK3CA mutations are typically observed among smokers [78].

4. Protection from exposure to second-hand smoke and impact on respiratory diseases

After 2005, when the Framework Convention on Tobacco Control (FCTC) has been acceded to, ratified, accepted, or approved by 40 States, many governments had a multitude of possibilities of interventions at their disposal to reduce tobacco use and SHS exposure, such as raising cigarette taxes, banning advertising and promotion of tobacco products, creating smoke-free areas in public places and worksites, mandatory introduction of health warnings

on cigarette packs, and promoting educational mass media campaigns regarding the harmful effect of tobacco use. From that moment, despite the efforts, the differences in implementation of FCTC measurements are still persisting from one country to another [79, 80]. Nevertheless, smokers in EU countries with high tobacco control scale (TCS) scores are more concerned about the effect of their smoke on others; this can be regarded as a marker of tobacco use denormalization, and as a support for tobacco control measures. These findings support the idea that the issue of passive smoking is essential for tobacco control in Europe [80]. It also happens like this for other continents. Within 20 years of the first Surgeon Generals' Reports, research showed that even the far lower doses of SHS have harmful effects on respiratory health [81].

4.1. Smoke-free legislation and his consequences

Self-regulation and smoking restrictions imposed by individual employers and venue operators have failed to protect staff and patrons in many enclosed environments, such as restaurants, pubs and casinos [82].

Smoke-free legislation is the best way to protect the passive smokers from SHS. Those who benefit most from this kind of policy are, undoubtedly, non-smokers; tobacco smoke is eliminated from the workplace, and they are able to breathe better-quality air. In addition, the working environment is improved thanks to the existence of a consensus among all the workplace's employees, which may lead to the resolution of possible conflicts between smokers and non-smokers [83]. Lack of knowledge of the health consequences of passive smoking exposure is an information failure and an important motivator of smoke-free policies.

Extended data from literature have described the tobacco industry's efforts around the world to undermine and discredit the evidence-based link between passive smoking and disease [84, 85]. Comprehensive smoke-free measures are the only effective means of fully protecting the public from the risks associated with SHS exposure. Partial measures that restrict, rather than eliminate, indoor smoking (e.g., designated smoking sections, separate ventilation) can reduce the exposure but do not offer adequate protection from his harmful health effects [86]. The offers of tobacco industry and tobacco lobbyists, such as "separating smokers from non-smokers, cleaning the air, and ventilating buildings, cannot eliminate exposure of non-smokers to second-hand smoke," as the US Surgeon General has concluded [40].

At the end of 2014, 49 countries had implemented the national comprehensive smoke-free legislation. This means that 18% of the world's population (1.3 billion people) is protected from the dangers of SHS by a comprehensive smoke-free law [87]. WHO reports that strong smoke-free legislation is the most widely adopted tobacco control measure, but low-income countries are less likely to have it adopted, than high- and middle-income countries [88]. In a growing number of countries, smoke-free policies have been extended beyond workplaces and indoor public spaces to include previously unregulated areas, such as outdoor spaces (public beaches and in public parks, as in Vancouver, or in pedestrian areas, as in New York) [87].

Comprehensive smoke-free legislation is a major policy intervention that works at several levels. It improves air quality and reduces non-smokers' SHS exposure, encouraging smokers

to reduce their tobacco consumption, limiting the times and places where they can smoke and motivating smokers to attempt to quit [89].

Smoke-free policies improve health outcomes. We have now significant and consistent evidences from around the world, showing that comprehensive smoke-free laws are associated with improved respiratory health and reduced cardiovascular disease [90–96]. There are multiple examples: 1 month after the implementation of a smoke-free law in Scotland, asthmatic bar workers demonstrated improvements in airway inflammation and self-reported quality of life [90], a smaller decrease in lung function was observed from the beginning to the end of a work shift, after smoke-free legislation was implemented in Norway [91], less likely to report adverse symptoms such as wheezing, coughing, and shortness of breath in the months after that state's smoke-free law took effect in hospitality workers in New York State [92], fewer current asthma symptoms and fewer physician visits because of asthma symptoms [93], and reduced asthma incidence, symptoms, and hospitalizations and/or emergency room visits in adults and/or children [94].

The lowest levels of SHS exposure are found in workplaces that completely ban smoking. Completely smoke-free workplaces achieve a number of public health benefits, such as reducing workers' exposure to the toxins contained by cigarette smoke, by 80–95% [95].

Regarding home SHS exposure, it has been observed that people of lower individual socioeconomic status are less likely to have smoke-free home rules and less likely to be protected by smoke free workplace policies [96], and that is why future SHS interventions need to pay more attention to people with this condition, in order to gain a better control of SHS exposure.

4.2. Other tobacco control measures

A 50% increase in cigarette prices will correspond to a 30–40% decrease of tobacco use for the poor, a much larger relative decline than among the rich. Another major consequence of this measure is that poor people benefit the most, economically and in health, from smoking cessation following a tax rate hike [88]. Evidence from Thailand showed that the poor paid only 6% of increased tobacco taxes, but got 58% of the health benefits [97]. These benefits consist of the consequences on respiratory and cardiovascular diseases, not only for smokers but also for nonsmokers. Taxes are very important; an increase of 10% in the price of cigarettes would decrease cigarette consumption by 3–5%, and, if implemented comprehensively, it could prevent 5–16 million smoking-related deaths worldwide and SHS exposure, also [98]. A number of studies have demonstrated that youth are relatively more sensitive to price than adults, implying that raising cigarette taxes would be a useful tobacco prevention intervention [88, 97, 98].

The major information source regarding awareness on the health effects of SHS exposure was radio media advert, especially in low income countries. Multi-level tobacco-control strategies should therefore include messages about harmful effects of SHS exposure to protect innocent never-smoked individuals, while warnings on cigarette packets should also include information on the deleterious effects of SHS on the health of adult, children, and pregnant women [99].

Educational activities are also important. It is a lack of appreciation among the general population of risk posed by SHS exposure to a vulnerable subset of population, namely,

children. Protection of children from SHS needs global attention and special educational campaigns in schools but also in adult population [100]. Social diffusion posits that restricting smoking in public spaces leads to increases in voluntary home smoking bans or restrictions [101]. Smoke-free legislation may stimulate smokers to establish total smoking bans in their homes.

An introduction of pictorial warning labels was shown to have a statistically significant effect on smoking prevalence, exposure of non-smokers, and quit attempts and significantly decreased the odds of being a smoker. Plain packaging of tobacco introduced in 2012 in Australia, brought a 78% increase in the number of calls to the national cessation quit line [102]. It is another action with a proof impact.

For electronic nicotine delivery systems (ENDS) and Heat-Not-Burn (HNB) tobacco products, governments should ban indoor use, because the aerosol released from both tobacco products contains many of the harmful constituents found in cigarette smoke [103]. There are more studies in this moment for e-cigarettes. There are likely health risks from being exposed to second-hand aerosol of HNB tobacco products [103, 104], but long term studies for analyzing the second hand effects at a distance are needed.

Policy and advocacy with effect on SHS concerning respiratory diseases must be complex and should use multiple action tools. In line with the FCTC, in 2008, WHO introduced the MPOWER measures, consisting of a set of six cost-effective and high-impact measures that help countries reducing the demand for tobacco and also for SHS exposure. These measures consist of: "monitoring tobacco use and prevention policies, protecting people from tobacco smoke, offering help to quit tobacco use, warning about the dangers of tobacco, enforcing bans on tobacco advertising, promotion and sponsorship, and raising taxes on tobacco." At the moment, more than half of all countries, representing approximately 40% of the world's population, have implemented at least one MPOWER measure to the highest level of achievement [87].

5. Conclusions

Passive smoking is a major wide-spread contaminant of indoor air and represents an important risk factor for pulmonary diseases among adult population. Extent epidemiologic evidence showed the strong association between passive smoking exposure and persistent respiratory symptoms, impaired lung function, and bronchial hyperresponsiveness. New-onset asthma and COPD, along with worsened prognosis of both diseases, are also attributable to passive smoking. Among never smokers, passive smoking increases the risk by approximately 30 and 200% for NSCLC and SCLC, respectively. Policies prohibiting smoking in the community have multiple positive effects on respiratory health. Comprehensive smoke-free legislation is a major policy intervention that should be widely implemented to protect non-smokers from the adverse health effects caused by passive smoking.

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Chapter 2

Effects of Smoking on Oxidative Stress and Vascular Function

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Additional information is available at the end of the chapter

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Abstract

Tobacco smoking is the single most preventable risk factor related to the development of cardiovascular disease. It was demonstrated that tobacco smoke contains a thousand compounds potentially harmful to human health. As tobacco use declined over time, electronic cigarettes were introduced as an alternative. E-cigarettes are a modern and technological surrogate of traditional cigarettes and use heat to convert a nicotine solution or a flavored nicotine-free solution into vapor. Even though all the ingredients contained in the liquid of E-cigarettes are approved as food additives, the harmlessness of these electronic devices is still not fully proven in humans. The general mechanisms by which smoking results in cardiovascular events include the development of atherosclerotic changes with a hypercoagulable state and an increased risk of thrombosis. Endothelial dysfunction has been recognized as a hallmark of preclinical systemic atherosclerosis and as a useful marker to stratify the risk of cardiovascular disease. Based on these considerations, in this chapter, we (1) discussed the role of endothelial dysfunction and its contributing factors, such as oxidative stress and inflammation, in the development of cardiovascular diseases and (2) reported the studies which investigated the effect of tobacco and electronic smoking on the biomarkers of endothelial dysfunction, oxidative stress, and inflammation.

Keywords: smoking, tobacco, electronic devices, endothelial dysfunction, inflammation, oxidative stress

1. Introduction

Cardiovascular diseases (CVDs) are the main cause of death in industrialized countries. The term CVD includes all the pathologies of the heart and of the systemic circulation including stroke, ischemic and not ischemic, and peripheral arterial disease, mainly of atherosclerotic type.

For the development of CVD, there are risk factors that can be distinguished in non-modifiable and modifiable. Non-modifiable risk factors are age (risk increase as you get older), gender (before the age of 60, men are at a greater risk than women), and family history (risk increase if relatives experienced early heart disease) [1]. Anyway, there are other factors, named modifiable, which can be changed, pharmacologically or changing lifestyle: hypercholesterolemia, hypertension, diabetes, and smoking [2]. These are atherosclerotic risk factors and are associated with endothelial dysfunction. In the diseased endothelium, all the oxidizing, inflammatory, and thrombotic molecules are not in equilibrium and therefore a pathological condition is observed. This condition is pro-inflammatory, prooxidant, and prothrombotic [3].

The mechanism of endothelial dysfunction is related to the increased vascular production of reactive oxygen species (ROS) and inflammation condition. Therefore, oxidative stress, inflammation, and endothelial dysfunction are related and together represent the risk factor for the development of atherosclerosis and subsequent clinical events such as myocardial infarction or stroke [4]. Many studies in the literature since decades demonstrated that cigarette smoking influences endothelial function, by acting on oxidative stress, inflammation, and platelet activation, and it is well known that tobacco consumption is a leading cause of death worldwide. In the last years, many alternative products, such as electronic cigarette (e-cig) and i-Quit-Ordinary-Smoking (iQOS), have entered the market. iQOS is a heat-not-burn (HNB) tobacco product that heats the tobacco just enough to release a flavorful nicotine-containing vapor but without burning the tobacco and then do not release combustion products. These devices became the sought-after product because people believe that they are safer than traditional cigarettes [5].

For these reasons, in the following paragraphs, we evaluated the impact of the traditional cigarette, e-cig, and iQOS on vascular function and CVD development.

2. Oxidative stress and vascular function

Human cells produce energy through aerobic respiration, a metabolic process that requires oxygen and leads to the production of ROS, small and simple molecules.

ROS play an important role in cellular function because they are implied in signal transmission and regulation, and under homeostatic conditions, cells can balance their presence through the action of antioxidant species and enzymatic defensive systems [6].

However, cellular defensive mechanisms cannot always prevent ROS accumulation. Increased levels of these species have dangerous effects, leading to serious cellular alterations. This condition is known as "oxidative stress," and it is considered to be associated with several diseases [7].

ROS are naturally produced during metabolism reactions. ROS include both free radicals, such as $O_2^{\bullet-}$ (superoxide), $ONOO^{\bullet-}$ (peroxynitrite), and OH (hydroxyl) and non-radicals such as H_2O_2 (hydrogen peroxide) which can be generated by enzymes such as xanthine oxidase, cyclooxygenase, lipooxygenases, myeloperoxidases, cytochrome P450 monooxygenase, uncoupled nitric oxide synthase (NOS), peroxidase, and NADPH oxidase [8]. The activity of these

pro-oxidant enzymes is in opposition to the defensive role of antioxidant endogenous systems such as superoxide dismutase (SOD), glutathione peroxidase (GPx), and catalase [9]. Moreover, nonenzymatic systems can contrast the oxidant action of ROS: these systems include glutathione, α-tocopherol, ascorbic acid, and many other antioxidant compounds taken with the diet.

Cellular ROS formation is a physiologic process in the vasculature and occurs in endothelial and smooth muscle cells (SMCs). Despite their high and random reactivity, they perform a fundamental role in the organism since they appear to be subtle physiologic modulators of biochemical processes involved in intracellular signaling [10].

ROS also participate in normal processes of cell growth and death and are involved in inflammatory responses, in regulation of vascular tone and in the production of erythropoietin in relation to the oxygen tension. They also play an important role in natural immunity, enhancing the effects of oxidizing agents produced by macrophages and granulocytes.

When the slight balance between ROS and cellular antioxidant defensive mechanisms fails, their physiological role quickly turns into a pathological activity. ROS demonstrate their toxic effects in various ways: for example, they can induce the oxidation of sulfhydryl groups into disulfide bonds of cysteine residues, highly present in numerous enzymes. This mechanism leads to the modification of proteins conformation that may cause alterations in the activity of enzymes or can determine DNA binding [11].

Furthermore, they can negatively interact with ion channels and transcription factors and can cause important cellular alterations such as lipid damage, an increase of cell permeability, cell apoptosis or death, alteration of growth factors, and endothelial dysfunction [7].

The vessel wall is the main molecular ROS source involved in the development of oxidative stress and associated impaired vascular function. Moreover, the activity of immune cells like polymorphonuclear lymphocytes and macrophages can enhance the severity of this stress condition [12].

Another leading factor of vessel damage is inflammation. Oxidative stress and inflammation are phenomena closely related to each other. Indeed, both result to be correlated with endothelial dysfunction and vascular damage, which have a critical role in the pathophysiology of several cardiovascular diseases [8, 13, 14].

The endothelium plays a crucial role in the maintenance of the vascular homeostasis since it can be considered an autocrine and paracrine organ which produces chemical mediators, growth factors, and vasoactive molecules with vasodilation, antiproliferative, and antithrombotic functions such as nitric oxide (NO), thromboxane (Tx) A2, prostaglandin (PG) A2, and cytokines [15].

The correct working of endothelium is fundamental for the regulation of vasal permeability, vasal tone and structure, and for the control of hemostasis, and inflammation.

The compromising of the endocrine endothelial activity, caused by typical cardiac risk factors such as hypertension, atherosclerosis, dyslipidemia, diabetes mellitus, cardiovascular disease, and smoke, can promote a pathologic condition called "endothelial dysfunction." This condition is characterized by weak vasodilatation, vascular remodeling, pro-coagulant,

and pro-inflammatory activity that strongly expose the organism to vascular damages and cardiovascular disorders such as atherosclerosis, plaque instability, and thrombosis [16].

One of the first and most important consequences of the stimulation of the endothelium activity is the impairment of NO levels, an important vasoactive molecule that regulates vascular tone since it promotes vascular smooth cell dilatation, inhibition of platelet activity, leucocyte adhesion, and vascular smooth muscle cell proliferation [17] (**Figure 1**).

Even if NO has a protective role for the endothelium, high concentrations of this molecule can lead to the generation of a toxic compound named peroxynitrite by the reaction with superoxide anion.

Peroxynitrite and ROS as reactive intermediates have proatherogenic effects in general and are able to induce molecular changes that lead to low-density lipoprotein (LDL) oxidation [18]. This type of modification induces macrophage infiltration in the endothelium with the aim of internalizing, through scavenger receptors, and rapidly degrading modified LDL. The result is the macrophage's inability to metabolize cholesterol and the consequent formation of foam cells, a characteristic of fatty streaks [19].

Another possible source of ROS that may act as a potential stimulus for ox-LDL generation is represented by platelets [20]. It was demonstrated that these circulating cells express four

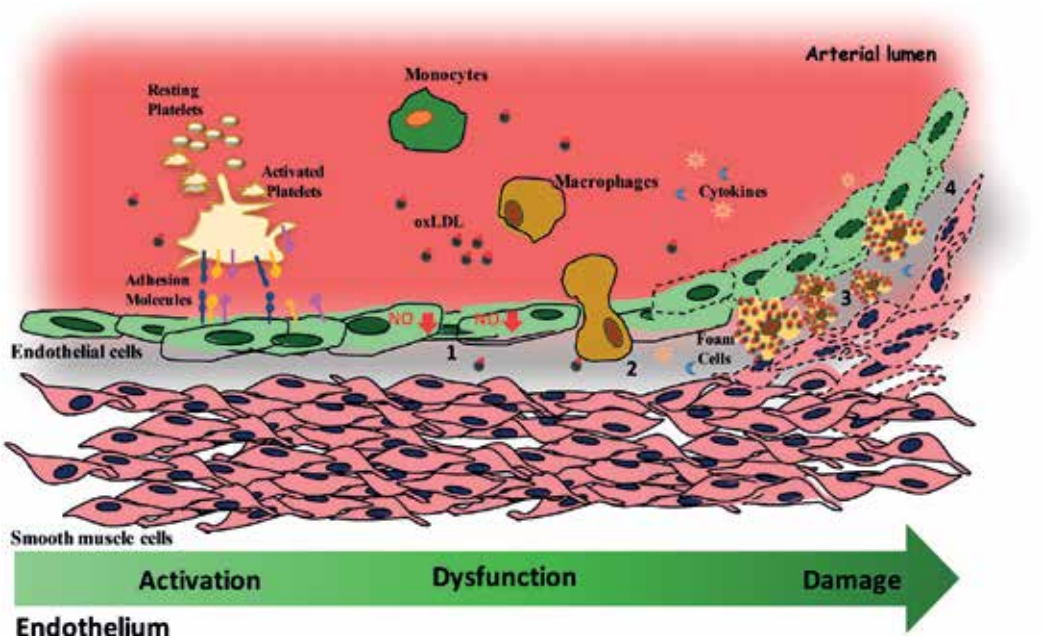

Figure 1. Schematic representation of signaling pathways involved in endothelial dysfunction. Oxidative stress activates the endothelium by inducing the expression of adhesion molecules and the activation of macrophages and platelets. Endothelial dysfunction is characterized by: (1) the reduction of NO levels; (2) platelet adhesion and macrophages transendothelial migration; (3) oxidative modification of LDL and their scavenger receptor-mediated uptake by macrophages inducing the formation of the so-called foam cells; and (4) the loss of function of smooth muscle cells (SMCs) in the vessel media and of endothelial cells.

subunits of NADPH oxidase: the most important enzymatic source of $O_2^{\bullet-}$ [21]. Under particular conditions, platelets may produce superoxide, which in turn is able to stimulate their functions and to help their recruitment [22, 23].

Furthermore, endothelial dysfunction contributes to the expression of the adhesive molecules and to the production of chemotactic signals that may induce macrophages and T lymphocytes adhesion and migration starting inflammatory state [24].

Increased ROS intracellular levels lead to the activation of the nuclear factor NF-kB, whose activity is normally inhibited by NO. NF-kB is a nuclear factor able to enhance the transcription of pro-inflammatory genes and the expression of the adhesive molecule (E-selectin, VCAM1, and ICAM1). The inflammatory response induced by this mechanism is therefore implicated in the immune cells recruitment to the vessel wall, in the release of constrictor agents such as angiotensin-II and in the loss of endothelium antithrombotic function through a reduced production of prostacyclin and fibrinolytic factors [25].

Oxidative stress and endothelial dysfunction represent the most important pathophysiological mechanism of serious growing conditions and diseases.

3. Traditional smoking and oxidative stress-mediated vascular dysfunction

3.1. Functional, cellular, and molecular implication of the cigarette tobacco smoke on cardiovascular disease

Cigarette smoking is a risk factor for the development and progression of CVD. This is demonstrated by many epidemiological studies, which showed a significant correlation between morbidity, mortality, and cardiovascular disease. Indeed, tobacco smoke is associated with pathological conditions such as endothelial dysfunction, inflammation, insulin resistance, dyslipidemia, hemodynamic alterations, and hypercoagulability that act together to furthering the progression of atheromatous plaque in tobacco users [26].

Cigarette smoke is represented by a mixture of different toxic chemicals. Among the constituents of the cigarette, which are considered the major participants in the development of CVD, there are nicotine, carbon monoxide, and oxidizing gases [27].

Nicotine is a potent stimulant of the ganglial nervous system and the central nervous system. Nicotine increases heart rate, blood pressure, and cardiac output, all of which translates into an increased myocardial oxygen demand. It is not yet clear whether nicotine has a direct role in the development of CVD. The effects reported are, mainly, on NO, contributing to endothelial dysfunction in cigarette users [28].

Carbon monoxide (CO), another constituent of cigarette smoke, does not appear to be involved in the processes of atherosclerosis or thrombosis associated with cigarette smoking. In unhealthy people with similar concentrations to tobacco users, CO does not affect blood pressure, plasma catecholamines, and platelet aggregation or serum reactive protein [29].

Oxidizing gases could contribute to the occurrence of a physiopathological state called oxidative stress. Oxidative stress is mediated by ROS overproduction and plays an important role in the development of CVD. In the context of cigarette smoking, free radicals could derive from the following:

1. vapor or particulate phases of cigarette smoke;
2. macrophages or neutrophils circulating or activated in situ;
3. platelets activation;
4. ROS production deriving from endogenous systems such as uncoupled endothelial nitric oxide synthase, xanthine oxidase, and the mitochondrial electron transport chain.

Oxidizing chemicals, including NO and many free radicals, are present at high concentrations in cigarette smoke and over time mediate endothelial dysfunction [30].

Cigarette smoke also contains a large number of metals, including aluminum, cadmium, copper, lead, mercury, nickel, and zinc, which are involved in the oxidation of cellular proteins causing structural damage and endothelial dysfunction [31].

As previously mentioned, tobacco influences various pathophysiological pathways that lead to the development and progression of atherothrombosis. Endothelial dysfunction, oxidative stress, inflammation, and prothrombotic state seem to be the main mechanisms involved.

Cigarette smoke could cause damage to the vascular wall, with a consequent reduction of prostacyclin production and an increased activation of platelets that interact with the injured vessel. The effect on endothelial function is mainly associated with the action of oxidative chemicals, which oxidize low-density lipoproteins (LDLs) and reduce the generation of NO [32].

It has been demonstrated that cigarette smoke-induced oxidative stress is responsible for endothelium activation through the expression of adhesion molecules and the activation of macrophages and platelets. Endothelial activation is characterized by a reduction of NO levels in endothelial cells (ECs) and consequently a loss of cellular function, in particular of the smooth muscle cells (SMC) of the vessel. After exposure to smoking, endothelial cells release inflammatory and proatherogenic cytokines. These events contribute to endothelial dysfunction. The direct effect of smoke compounds is the ROS overproduction that induces endothelial cell loss through apoptosis or necrosis processes. In addition to endothelial cells, also macrophages are activated. The expression of receptors promotes the recognition of adhesion molecules on the endothelium. After adhesion and migration through the endothelium, macrophages capture the oxidized lipids. These oxidative changes are promoted by the increase of smoking-induced ROS production. The receptor-mediated uptake induces the formation of the foaming cells inside the vessel wall and thus leads to the generation of lipid-induced plaques. Furthermore, it has been suggested that smoke induces an increase of SMC's proliferation and migration; this process leads to a thickening of the intima and plaque formation. Another consequence of exposure to cigarette smoke is the death of SMC by necrosis resulting in the activation of inflammatory signals, the release of proteolytic enzymes, and damage to the extracellular matrix [33].

Cigarette smoke induces an alteration of the fibrinolytic system by inhibiting the tissue plasminogen activator release from the vascular endothelium. Furthermore, it is responsible for activation of different pathways, involved in platelets activation and thrombus formation. In fact, cigarette smoke leads to a greater expression of the adhesion molecules on the platelet surface and therefore to a greater activation of the platelets [34].

Cigarette smoking is a risk factor for the development of type 2 diabetes, and the risk decreases after cessation [35]. It is documented that smokers show an increase of insulin resistance, central obesity, dyslipidemia, and a greater risk of metabolic syndrome onset. The link between smoking and insulin resistance could be attributed to the role of nicotine on the activation of the sympathetic nervous system and to the release of corticosteroids [36].

However, the mechanism is unclear. Nevertheless, it has been demonstrated that insulin resistance negatively affects the lipid profile, induces endothelial dysfunction and oxidative stress, driving to the formation of atheromatous plaque, and the development of cardiovascular diseases [37]. Moreover, a recent study compared plasma glucagon concentrations between 11 smokers and 12 nonsmokers diabetic patients before and after meal intake. The authors demonstrated that diabetic smokers had higher levels of glucagon compared to nonsmokers diabetic patients. In particular, they showed that nicotine smoke-derived and subsequent activation of nicotinic cholinergic receptors in the gastrointestinal tract and in the autonomic nervous system has an injurious effect on postprandial glucose metabolism. For this reason, a link between cigarette smoking and the development of type 2 diabetes could be hypothesized [38].

3.2. Cigarette smoke and biomarkers of oxidative stress

Free radicals are a product of cigarette smoke and are considered to have negative effects producing oxidative stress.

The imbalance between ROS and antioxidant systems inside the cell is called oxidative stress. The cell, under physiological conditions, produces ROS by oxygen metabolism, which plays an important role in cellular signaling and safe. The presence of oxidative stress, a pathophysiological condition, causes an excessive production of ROS that causes lipid peroxidation, DNA strand breaks, and other damages that are injurious to structure and functionality cell [39].

ROS that accumulate inside cells can be exogenous and endogenous. Exogenous ROS derive mainly from inhaled toxic gases (e.g., environmental pollutants, car exhaust fumes, and cigarette smoke). Endogenous ROS come from the processes of mitochondrial respiration, from peroxisomes, from the NADPH oxidase system, and from inflammatory cells [40]. However, the process of oxidative phosphorylation in mitochondria is one of the main endogenous sources of ROS [41].

Tobacco smoke contains about 5000 compounds of harmful chemicals, which include polycyclic aromatic hydrocarbons, free radicals, and oxidative gases [42].

Therefore, together with the induction of ROS intracellular production, the components of cigarette smoke reduce intracellular antioxidant mechanisms, leading to oxidation stress [43]. Many studies showed the involvement of cigarette smoke in the oxidative stress. Recently, Karademirci et al. in a case–control analytical study, conducted on 78 smoking and

82 nonsmoking men, demonstrated that the total antioxidant status (TAS), vitamin C, and vitamin E parameters were significantly higher in the nonsmoker group than in the smoker group. The total oxidant status (TOS) and oxidative stress index (OSI) levels were higher in the smoker group [44].

In another study on 32 healthy volunteers, 16 active smokers and 16 nonsmokers, the markers of oxidative stress in the blood were analyzed before and after exposure to cigarette smoke. The results obtained showed that acute exposure to cigarette smoking affects hematological indexes and oxidative stress biomarkers negatively. The same results are found in both groups [45]. Carnevale and collaborators demonstrated the acute impact of tobacco cigarette on oxidative stress by measuring important biomarkers. Their data showed that cigarette smoke leads to a significant increase in the levels of soluble NOX2-derived peptide (sNOX2-dp) and 8-iso-prostaglandin F2α (8-isoPGF2α), while vitamin E levels were reduced [46].

Other studies, on animal models, showed that a reduced and defective antioxidant defense after cigarette smoke exposure (a decreased glutathione peroxidase and superoxide dismutase activity, an increased lipid peroxidation, and mitochondrial dysfunction) [47] leads to the increase of H_2O_2 generation [48]. This unbalance results in an increased cardiac damage caused by oxidative stress induced by cigarette smoking.

3.3. Cigarette smoke and biomarkers inflammation

Inflammation is strongly implicated in the pathogenesis of atherosclerosis, and there are numerous lines of evidence that associate cigarette smoking with conditions of chronic inflammation [26].

Oxidative stress and inflammation are two closely related conditions. Indeed, the overproduction of ROS triggers NF-κB and histone acetyltransferase activation, promoting the expression of inflammatory genes and consequently the production of inflammatory cytokines [49].

Different studies demonstrated that the inflammatory process is reflected by numerous markers expression, which play an important role in the atherosclerosis process [50]. Furthermore, it is well known and accepted that inflammatory processes are strongly induced by exposure to cigarette smoke [51]. Between inflammatory markers, it is possible to analyze leukocyte count that increases in a dose-related manner with the number of cigarettes smoked daily. Indeed, many crossover and cross-sectional studies have observed an increase in the number of leukocytes following exposure to cigarette smoke [52].

Another marker analyzed in association with smoking is the plasma level of high-sensitivity C-reactive protein (hs-CRP). This marker of inflammation is significantly increased in long-term smoking and is correlated to the number of pack-years of cigarettes. Inflammation markers were found significantly increased in another study where levels of circulating total white blood cells, lymphocytes, monocytes, neutrophils, basophils, and C-reactive protein (CRP) were higher in 31 current smokers than in 33 never-users of tobacco products [53].

Important inflammatory markers are inflammatory and anti-inflammatory cytokines. IL-6 is a pro-inflammatory cytokine that in the acute phase of inflammation is able to induce other cytokines and growth factors as well as activating the platelets and the coagulation cascade [54].

Many studies conducted on large populations of smokers showed an increase in the expression of IL-6 compared to nonsmokers. A recent case–control study showed that smoking increases inflammatory marker such as IL-6 and VEGF levels, while IL-10, an anti-inflammatory marker, was lower in smokers group [55].

These data are important because high levels of IL-6 are related with an increased risk of cardiovascular disease, in particular, with myocardial infarction and severe heart failure [56].

3.4. Cigarette smoke and biomarkers of endothelial dysfunction

Endothelial cells are the main protagonists in vascular function control. The lack of regulatory mechanisms activated by these cells leads to inflammation, vascular remodeling, and development of endothelial dysfunction. Endothelial dysfunction is an early event in atherosclerosis and is characterized by an imbalance between vasodilatation and vasoconstriction, a pro-inflammatory endothelial cell status, an increased monocyte adhesion, and a reduced bioavailability of NO [57]. There is considerable evidence that cigarettes smoke induces functional, biochemical, and morphological changes of the endothelium [58].

Wiest et al. demonstrated that flow-mediated dilation (FMD) as a clinical evaluation of endothelial dysfunction is compromised in smokers. FMD can be considered a predictor marker for future cardiovascular events and that smoking cessation can improve this parameter [59].

Furthermore, in a crossover study, performed on 40 healthy subjects, FMD and NO bioavailability levels were measured. The authors documented a significant decrease of these parameters, proving the negative effect of smoking on endothelial function [46].

Moreover, the serum nitrate and nitrite concentration, the final metabolic products of NO, are significantly decreased in smokers than in nonsmokers. Furthermore, in smoker subjects, low-density lipoprotein (LDL) is more susceptible to oxidation by excessive ROS and NOS presence. Oxidized LDL (ox-LDL) causes less bioactivity of NO deriving from the endothelium; this loss of bioavailability is associated with an increase of inflammatory cells that cross the vascular wall. Finally, the uptake of ox-LDL by macrophages via recognition by receptors results in foam cells formation [60].

Another mechanism involved in endothelial dysfunction is the increased ability of endothelial cells to adhere to effector immunity cells (monocytes, macrophages, T lymphocytes, platelets). In fact, the level of adhesion molecules is higher in plasma of smokers. Many studies reported significantly higher levels of soluble intracellular adhesion (ICAM-1), P-selectin, and E-selectin in smokers than in nonsmokers [56, 61]. Generally, endothelial dysfunction caused by cigarette smoking can lead to an increased possibility of atheromatous plaque formation and progression [62].

3.5. Cigarette smoke and biomarkers of platelet activation

Platelet activation and enhanced coagulation are two events related to cardiovascular disease and atheromatous plaque formation. Platelet activation and aggregation are involved in both physiological hemostasis and pathological thrombus formation [63]. Smoking has been reported to enhance platelet aggregability [64]; in fact, it has long been demonstrated that

platelets isolated from tobacco cigarette users showed an increase of aggregation [65]. In an in vitro study, traditional smoking extracts from cigarettes significantly increased oxidative stress-induced platelet activation [66].

Platelet activation can be evaluated by various markers. An important platelet activation pathway is the synthesis of thromboxane A_2 via COX-1. The evaluation of urinary levels 11-dehydrotromboxane B_{2}, a stable metabolite of thromboxane A_{2}, reflects this type of platelet activation. 11-dehydrothromboxane B_2 levels in the urines of 13 healthy smokers were significantly elevated as compared to 10 healthy nonsmokers [67]. Numerous studies arrived at the same conclusions. In fact, data that correlate the influence of cigarette smoke and platelet activation, considering various markers, are present in the study since the early 1980s [68–70].

4. New electronic devices and their effect on vascular function

4.1. The rise of electronic cigarette: the impact on public health

The first reference to the electronic cigarette (e-cig) has been documented since 1927 but it was in 2003 that a Chinese pharmacist, Hon Lik, invented the modern version of the e-cig. Afterwards, this device was patented internationally in 2007 and was subsequently introduced into the global market. Today, the e-cig represents an alternative to traditional cigarettes and has gained popularity particularly among young adults. Structurally, these products are designed in order to closely resemble traditional tobacco cigarettes [71]. There are four types of e-cig: disposable; first-generation e-cigs that are tobacco cigarette-shaped; the second generation that looks like pens, larger than a cigarette, with a refillable cartridge; third generation considerably larger than the first- or second-generation e-cig [72]. Functionally, e-cigs are battery-operated devices with a heating element that heats the e-liquid, a solution of nicotine, and other additives including propylene glycol, vegetable glycerine, and flavoring agents, to a temperature of about 200–300°C to form an aerosol which is inhaled into the lungs. As tobacco cigarette, nicotine is the primary addictive substance in e-cig with levels, efficacy, and consistency that vary considerably among different brands and models. Schroeder et al. [73] revised the study related to the clinical pharmacology of nicotine contained in the e-cig. They reported studies that analyzed nicotine yield in the aerosol. For example, according to the analysis conducted by Goniewicz and colleagues [74] on the levels of nicotine vaporization of 16 cig brands, 20 series of 15 puffs generate a level of nicotine in the vapor that vary from 0.5 to 15.4 mg. By an HPLC method, Trehy and colleagues [75] evaluated nicotine content, ranging from 11 to 24 mg/cartridge. In 100-mL puffs from different e-cig brands, nicotine yield was highly variable, ranging from 0 to 43.2 μg nicotine. Unlike the traditional cigarette, the e-liquid of e-cig contains different flavoring agents including buttery minty, cherry or almond cinnamon, and chocolate flavors. Several studies suggest that some flavorings will promote e-cig use among youth [76, 77] so that we have observed the exponential spread of e-cig use in high school student in the last few years [78]. However, up to 250 different compounds have been identified in the inhaled E-liquid vapor [79, 80], suggesting that e-cig discharges a range of compounds capable of physiological damage to users.

Indeed, there is increasing evidence showing the short-term negative effect of e-cig. The results of these studies documented the effect on the respiratory tract, which is the primary system exposed

to vapors from e-cig. In healthy smokers, using an e-cig for 5 min has an immediate adverse effect on pulmonary function evaluated as exhaled nitric oxide measurements, lung volumes, and total respiratory resistance [81]. However, Flouris et al. [82] evaluated the lung function in 15 cigarette smokers after a brief session of active e-cigarette smoking and after a 1-h passive e-cigarette smoking and they found that e-cig generates smaller changes in lung function.

Other studies demonstrated the deleterious effects of e-cig use on multiple biological systems, such as central nervous system, immune system, and cardiovascular system. Regarding this latter, as previously discussed, there is definitive evidence that cardiovascular disease is the major cause of death among smokers. On the contrary, few and contrasting data regarding the effect of e-cig on the cardiovascular system have been obtained. There are several studies that evaluated the detrimental effect on cardiovascular function such as the increase in heart rate [83] or an increase in both diastolic blood pressure and heart rate in e-cig smokers, but to a lesser extent when compared with tobacco smokers [84]. These data were also supported by a recent study demonstrating that habitual e-cigarette use was associated with the imbalance of cardiac autonomic tone toward sympathetic predominance [85]. On the other hand, some studies have shown that short-term exposure to e-cig has no effect on cardiovascular system. The use of electronic cigarettes causes no changes in arterial stiffness [86], in myocardial function [87], and in smokers heart rate [88].

Here, in this paragraph, we report the study analyzing the effect of e-cig on cardiovascular risk factor, in particular, endothelial dysfunction, oxidative stress, and inflammation that are interrelated factors in the etiology of cardiovascular disease.

4.1.1. E-cig smoking and biomarkers of endothelial dysfunction

The assessment of endothelial function consists of endothelial cells responsiveness to different stimuli. The methods include (1) in vivo analysis performed by the flow-mediated dilation (FMD) [46], which is a noninvasive technique, in use in clinical practice; (2) the determination of endothelial cell functions by in vitro analysis, such as cultures of endothelial cells [89–91]; (3) the evaluation of circulating biomarkers, such as endothelial progenitor cells (EPCs) and microvesicles (MVs) [92].

Specifically, in 40 healthy subjects, 20 smokers and 20 nonsmokers, FMD was significantly affected by cigarette smoking, both tobacco and electronic, coincidentally with a significant decrease in NO bioavailability, although e-cig seemed to have a lesser impact [46]. Because of the limited availability of human vascular endothelial tissue, for in vitro studies, human umbilical vein endothelial cells (HUVEC) represent a good and useful model to understand endothelial physiology. Indeed, this cellular line is used to study the interaction of endothelial cells (ECs) with blood cells and different mediators [93].

Exposition of cells to extracts produced from e-cig vapor alters endothelial cell functions as suggested by a significant decrease in metabolic activity for HUVEC exposed, a significant increase in complement deposition, and in the expression of the receptors for C1q [89]. Moreover, the exposition of HUVEC to e-cig vapor extracts causes high cytotoxicity, inhibition of cell proliferation, and significant morphological alterations in endothelial cells disrupting the functional endothelial monolayer [90]. Anderson et al. supported e-cig-induced

cytotoxicity by demonstrating that cigarette aerosol extract (EAE) triggers both apoptosis and programmed necrosis pathways in HUVEC model [91]. Finally, Antoniewicz and colleagues evaluated in healthy young volunteers two circulating biomarkers, that is, endothelial progenitor cells (EPCs) and microvesicles (MVs). Acute endothelial dysfunction and inflammation may generate both circulating EPCs and MVs. Short-term exposure to e-cig vapor significantly increased the number of circulating EPCs without affecting MVs release [92].

4.1.2. E-cig smoking and biomarkers of oxidative stress

As previously reported, the ability by tobacco smoke to generate ROS and to induce oxidative stress is well documented so that it is considered as a driving factor in smoking-related diseases [94, 95]. Regarding e-cig, in a recent study, Zhao et al. [96] performed ROS characterization of e-cig emissions using acellular and cellular approaches. Findings of this study confirm total ROS and H_2O_2 generation in e-cig emissions that can contain a comparable level of ROS compared to tobacco cigarette. The role of e-cig smoking in oxidative stress generation is also supported by other studies. As oxidative stress can be defined as an alteration of the balance between pro-oxidants and antioxidants, oxidative status is studied by (1) the evaluation of intracellular oxidant species generation by fluorescent dye, that is, 2′,7′-dichlorofluorescin diacetate (DCFDA) [97, 98] and (2) changes in antioxidant defense, that is, nonenzymatic α-tocopherol [91], enzymatic glutathione ratio [98], or total antioxidant defense [97].

Specifically, the treatment of HUVEC with e-cig aerosol extract (EAE) induces an unbalance between pro- and antioxidant molecules. Indeed, there is an increase of ROS production in a concentration-dependent manner and a decrease of antioxidant molecules such as α-tocopherol and n-acetyl-l-cysteine. This alteration induces cell death in vascular endothelial cells providing evidence about the role of ROS in e-cigarette-induced cytotoxicity [91]. The role of e-cig aerosol extract in oxidative was also supported by Ganapathy et al. [97]. By using human oral and lung epithelial cells, they found that e-cig aerosol extracts exposure significantly increased ROS production with a concomitant reduction in antioxidant defense, evaluated by total antioxidant capacity (TAC).

Taylor and colleagues [98], using an *in vitro* model of the airway epithelium (human bronchial epithelial cells), evaluated the effect of aqueous aerosol extracts (AqE) on oxidative stress. They evaluated the cellular ratios of reduced glutathione (GSH) to GSSG, intracellular generation of ROS, and the activation of the transcription factor nuclear factor erythroid-related factor 2 (Nrf2) that activates systems implicated in the neutralization of ROS and repair oxidative damage. After exposure to AqE, they did not find significant responses in the in vitro assays of oxidative stress or cytotoxicity [98].

In vivo, the effect of e-cig on systemic oxidative stress is evaluated in two studies. In healthy subjects, short-term e-cig smoking is associated with an increased oxidative stress, as demonstrated by an increased activation of NADPH oxidase (Nox) 2, which is an essential enzyme producing ROS by phagocytes [99] and platelets [100]. Moreover, there is an increase in 8-iso-prostaglandin F2α production coincidentally with a decreased antioxidant vitamin E [46]. In healthy individuals, the habitual e-cig use is associated with an increase in low-density

lipoprotein (LDL) oxidizability, indicative of the susceptibility of apolipoprotein B-containing lipoproteins to oxidation, compared to nonusers. Conversely, no difference was observed for high-density antioxidant/anti-inflammatory capacity and paraoxonase-1 activity [85], which exerts its physiological function in the degradation of specific oxidized cholesteryl esters and oxidized phospholipids in lipoproteins and cell membranes [101].

4.1.3. E-cig smoking and biomarkers of inflammation

As documented in the previous paragraph, tobacco products can lead to cardiovascular disease development by activating pro-inflammatory and/or prothrombotic function within the vasculature. The absorbed or dissolved fine-particulate matter, toxic chemicals, and nicotine act to produce oxidative stress factors that could induce the release of pro-inflammatory cytokines. Therefore, oxidative stress and inflammation are closely interrelated. Regarding the role of e-cig in inflammation, its effect was evaluated by using different model of in vitro cells and by measuring in particular the complement activation [102, 103] and interleukins release [102, 104].

Specifically, Rubenstein and colleagues used, as in vitro model, the Kupffer cells that are macrophages resident in liver sinusoids [105]. These cells interact with platelets and leukocytes to mediate inflammatory response [106]. In this study, the treatment of Kupffer cells with e-cig vapor extracts induces (1) the deposition of complement products C1q, C3b, C4d, and C5b-9, (2) the expression of gC1qR and cC1qR complement receptors, and (3) the release of pro-inflammatory interleukins 2, 4, 6, and 13 [102]. The deposition of complement was also analyzed onto the platelet surface [103]. After exposure to e-vapor extracts, the deposition of C1q and C5b-9 was not altered after exposure, whereas the deposition of C3b and C4d was significantly enhanced. Moreover, using differentiated THP-1 macrophages, the effect of e-cigarette components such as e-liquid flavors, nicotine, vegetable glycerine, and propylene glycol has been evaluated. Results demonstrated that IL-8 secretion increased with flavor and nicotine, while TNFa, IL-1b, IL-6, MIP-1a, MIP-1b, and MCP-1 decreased after exposure to most flavors and nicotine [104].

4.2. The new heat-not-burn smoking device: the little we know

The heat-not-burn (HnB) tobacco device iQOS was introduced in 2014 in Japan and Italy and currently is marketed in 30 countries. Unlike the e-cig, which heats the e-liquid containing different substances but not tobacco, the iQOS device allows heating a cigarette composed of tobacco without activating a combustion process. Farsalinos and colleagues [107] evaluated the content and nicotine delivery to the aerosol of iQOS compared to e-cig and tobacco cigarette. First, the authors concluded that HnB devices contain nicotine in concentration similar to that found in tobacco cigarettes and that deliver nicotine less than tobacco but higher than e-cig. Another study [108] confirmed the presence of nicotine levels in tobacco fillers and in the mainstream smoke, same as those of conventional cigarettes. Regarding the effect of iQOS on health, there are no scientific data. For this reason, it is necessary to invest in the research aimed at defining the impact of these new smoking devices on health.

5. Conclusions

The evidence of the deleterious effects of smoking standard cigarette on cardiovascular system dates to the first half of 1900, and since then, the risk of smoking on health has been confirmed by numerous studies. Years of research into the effects of smoking have led to the conclusion that smoking represents an important risk factor for many diseases affecting numerous biological systems. Here, we described the effect of smoking on cardiovascular diseases, with attention to the pathways implicated in endothelial dysfunction. These data support the effect of cigarette smoking on oxidative stress and inflammation with a generally detrimental effect on cardiovascular system (**Figure 2**).

According to the World Health Organization, by 2030, tobacco will be responsible for more than 10 million deaths per year. From these data, the need to quit smoking considering the general and almost immediate benefits in the cardiovascular health is evident [109].

In the program of smoking cessation, e-cig represents an alternative to the traditional cigarette. Despite the increase in use especially among younger, the e-cig is not safer than traditional. As reported in the dedicated paragraph, e-cig contains potentially harmful substances that can affect health in general and the cardiovascular system in particular (**Figure 2**). Several studies report the deleterious effects of e-cigs even if to a lesser extent than the traditional cigarette. At present, the data in the literature concerning this topic are derived from short-term

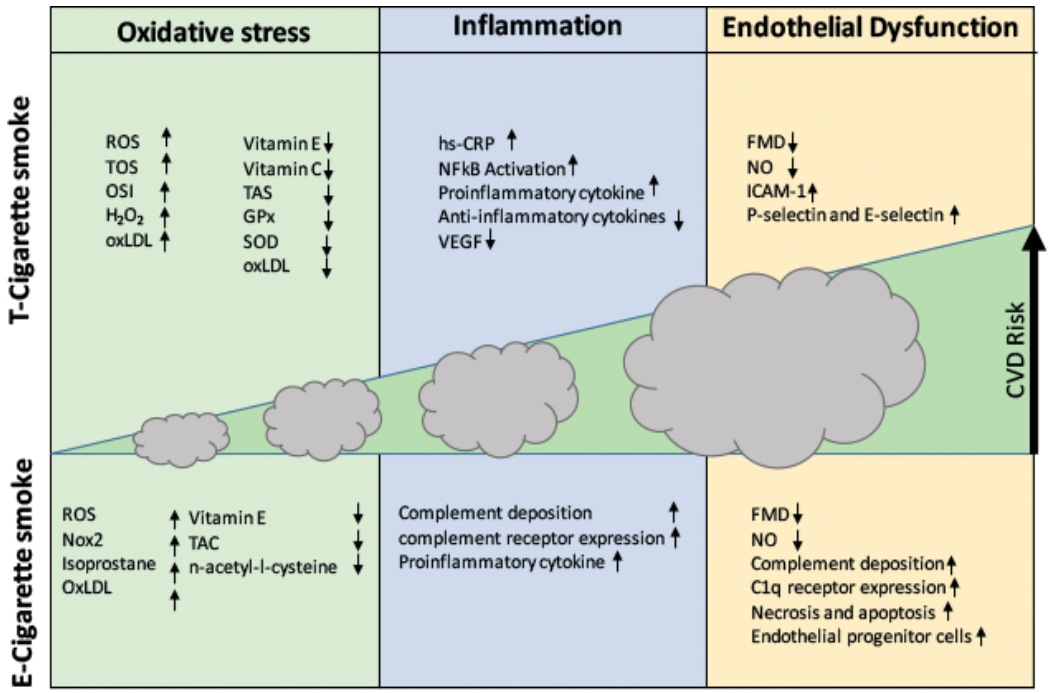

Figure 2. The impact of tobacco cigarette and E-cigarette exposure on CVD risk. The increased exposure to the smoke of tobacco cigarette and e-cig has an important impact on health. Smoking affects biomarkers of oxidative stress, inflammation, and endothelial dysfunction, thereby increasing the risk of cardiovascular disease. A schematic summary of the effect of smoking on the biomarkers considered in the chapter is reported.

exposure studies. To have a complete view of the impact of e-cig on health, it is important to evaluate the long-term effects. These researches would be important not only because it is crucial to clarify the effects of e-cig on health but also for its social impact. Indeed, while e-cig could help tobacco smokers to quit smoking, they could encourage nonsmokers, especially teenagers, initially to use e-cigs and later traditional ones [110].

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Chapter 3

Smoking Habit and Nicotine Effects

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Additional information is available at the end of the chapter

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Abstract

Smoking is a gained habit with which one starts experimenting at the age of 10, and it usually becomes part of the habit at the age of 20. It is the combination of narcotic addiction and deep-seated smoking habits. Nicotine is the main cause of smoking addiction, and the custom of preparation for smoking itself and smell of cigarettes create addictive behavior among smokers. Today, nicotine is socially most widely accepted legal drug in the world, and its uptake into the organism through the respiratory tract is 10 times stronger than heroin. Addiction and necessity for cigarettes are constant and intense, and the treatment for smoking addicts is long and difficult. Smoking is a worldwide epidemic, which is closely connected to other addictions such as coffee, alcohol, drugs, and gambling.

Keywords: smoking, nicotine, addiction

1. Introduction

Smoking is a chronic non-contagious disease that causes a variety of diseases leading to premature death and a significant reduction in the quality of life. The way to consume tobacco is by inhaling smoke from a stunned tobacco leaf in cigarettes, cigars, or tobacco pipe. The occurrence of addiction is massive and it is the most widespread social disease. Tobacco smoke is one of the biggest risk factors responsible for 63% of the total mortality. By 2030, it is estimated that 10 million people per year will die of the consequences of smoking [1]. According to World Health Organization data, tobacco smoking is among the first causes of death that can be prevented. Epidemiological studies around the world have shown that there is a strong link between smoking and incidence of cigarettes, nonsmoking and chronic diseases such as

cardiovascular disease, lung disease, stroke [2, 3], and cancer. The global epidemic of smoking worldwide causes about 6 million deaths every year, including about 6,000,000 people who were nonsmokers but were exposed to inhalation of tobacco smoke. Every 8 s, a person dies from tobacco addiction. [4]. The International Cancer Research Organization has included more than 60 substances [5], which tobacco cigarettes contain, in the carcinogen category. Studies show that smoking prevalence comes from low- or middle-income countries.

Tobacco is the most accessible drug, and its consumption has its history. There are data that tobacco smoking had been practiced somewhere between 5000 and 3000 B.C. In the archeological finds in areas where the Indians from the Mayan tribe lived, the priests were shown smoking a tobacco pipe during a ceremony [6]. A common pipe was smoked during various ceremonies, as a welcome sign, for the end of warfare and peacemaking, as the famous *peace flag*. Tribal healers used tobacco, dry leaves, or powdered leaves as a healing remedy for wounds, pain relief, fatigue, thirst, or hunger [7]. There was an overwhelming belief that tobacco was a sacred plant. Tobacco is considered to be abundant in North America [6]. The history of tobacco dates back to 1492 when Christopher Columbus wrote in his journal: *here, in the new country there are men and women with a half-burnt weed in their hands, being the herbs they are accustomed to smoke…* [8]. Colombian sailors transferred tobacco to Spain and Portugal, and from there it was spread across European countries and other continents. In 1560, French Ambassador of Portugal Jean Nicot brought tobacco seeds to the royal court and gave it to Queen Katarina Medici as a miraculous and healing plant. Tobacco got its Latin name after his surname *Nicotiana*. There are 70 different species of *Nicotiana*, and the best known is the one from the *Solanaceae* family—Nicotiana *t*abacum [9]. It is a 1-year plant that can grow up to 2 meters, and tobacco is produced in the root and deposited in the leaves [9]. The name is derived from the Arabic word *tobacco*, meaning smoke. From 1600 to 1700, smoking spread across Europe in all social classes [10]. During the seventeenth century, tobacco planting was extended to other European countries: Germany, Sweden, Denmark, Austria, and then the Far East and China [9]. The first laws prohibiting smoking were adopted in the seventeenth century. Sultan Murat brought a law that punished anyone who smoked with a death sentence because it was suspected that smokers caused a major fire in Constantinople. In the same period with the support of the church, anti-smoking laws were being adopted in Europe [11]. British King James I condemned the first tobacco importer in England with a death penalty. In the mid-nineteenth century, industrial cigarette production began, and smoking became fashionable. Tobacco expansion and the number of smokers increased especially after the end of World War I where a large number of smokers recorded were among women.

Since the appearance of tobacco and smoking in Europe, we have witnessed the first conflicts between two sides, those who advocate smoking and tobacco as a healing plant, and others who consider it harmful and dangerous to health. None of the two parties could prove that they were right or wrong until the beginning of the twentieth century.

During the 1920s, the dangers of tobacco smoking leading to mortality and the many consequences of smoking on the economic and social level were recognized. From the early 1930s to the mid-1950s of the twenty-first century, the first experimental evidence confirms the association between tobacco smoke and lungs, bronchus diseases, and the carcinogenicity of cigarette smoke substances. Epidemiological research has shown that there is a causal

link between excessive cigarette smoking and lung cancer. In 1960, a statutory obligation was issued to highlight the health hazard warning on each cigarette box. From 1962 to 1964, Luther L. Terry had published more than 7000 articles on smoking and health and ended with a famous report titled *Terry's Bomb*. Thereafter, the scientific approach to the smoking problem intensified, and in 1967, the First World Conference on Smoking and Health was held by the World Health Organization (WHO) as a systematic action plan. In 1971, WHO began a systematic anti-smoking campaign and adopted the first anti-smoking resolution and first action plans. In 1975, smoking was first listed in the International Classification of Diseases, and in 1995, it was classified under mental disorders and tobacco-related disorders under code F17.

2. Nicotine dependence

Smoking is initially a habit leading to physical and psychological dependence. An important role in the smoking-related phenomenon is a number of repeated movements in the smoking act of the hand and oral muscles, cigarette outbreaks, lighting a cigarette, blowing smoke, holding, and extinguishing the cigarette butt. All these movements are repeated a few hundred times a day [6] and become a ritual that is repeated. A smoker thus makes about 500–600 kisses daily by activating their oral muscles. The smell of smoke, fire, or lighter, and the way cigarette is lit also affect dependence. Cigarettes are like magic wands that are used to relax, calm down, relieve stress, or as a substitute for enhanced food enjoyment. The act of smoking contributes toward self-destruction and pollution of the environment. Therefore, smoking is not just the problem of the person who smokes, but it creates problems for the entire community and society where the smoker lives and works. People who smoke cigarettes can be divided into four groups: permanent smoker, occasional smoker, former smoker, and non-smoker. The consequences of smoking can affect non-smokers, who we call passive smokers. The largest group of victims is children whose parent(s) smoke because cigarette smoke remains in the air for the next 8 h [12] after a person has finished the task of smoking, and is called environmental tobacco smoke or *Second-Hand Tobacco Smoke* (SHS) [13].

2.1. Harmful effects of nicotine

Nicotine is the main tobacco alkaloid, isolated in 1828. It is a colorless, oily liquid, odorless, and one of the strongest herbal poisons and neurotoxins that act on the central and peripheral nervous system. The most dangerous ingredient is tobacco smoke, which stays in the air, and is connected with oxygen with a characteristic smell of tobacco. The fast "dose" of nicotine is obtained from 7 to 10 s and is one reason why smoking becomes a hard-core addiction that speeds up heart activity, elevates blood sugar levels, increases blood pressure, triggers an apparent fatigue condition that leads to slowing down of heart causing uneasiness and the desire to recycle the cigarette so that the discomfort disappears and nicotine accumulates in the blood. This pattern cyclically repeats and promotes the sustenance of nicotine addiction, called *nicotinism*. Its effect on the central nervous system makes smoking comfortable and acts on brain cells in a way that regulates emotions, alleviates anxiety, irritability, and improves mood. The smoker is able to control how much the brain receives nicotine by blowing the smoke at his own pace [14]. From lighting the cigarette, the smokers inhale about 15–20%

smoke on average. Acute nicotine poisoning causes symptoms such as vomiting, weakness, blush, dizziness, drowsiness, headache, and sweating. Abdominal pain, cramps, anxiety disorder, low blood pressure, poorly filled pulse, salivation, epileptic cramps, collapse, loss of consciousness, cessation of breathing, and death are the result of larger quantities of poisoning. The smoker's body launches various defense mechanisms against tobacco poisoning that overwhelm the inhaled poisons, reduce or prevent their adverse effects, and reduce toxic effects on the human body, and therefore no symptoms of acute poisoning appear, as with the novice smoker. With regular smokers, symptoms of poisoning can occur if the smoker smokes double or multiple cigarettes, or more cigarettes than usual.

2.2. Tobacco smoke

Tobacco smoke contains more than 4000 different compounds such as alkaloids; alcohols; phenols; ethers; ketones; quinones; esters; nitrites; hydrocarbons; sulfur organic compounds; various inorganic compounds of lead, iron, copper, manganese, nickel, hydrocarbon molybdenum; and other metabolites. The composition of tobacco smoke depends on the type of tobacco, the temperature and the rate of combustion, various supplements, the length of cigarettes, the technological processing, and the production of cigarettes. The most potent tobacco smoke ingredients introduced into the human body are nicotine, cyanide compounds, and carbon monoxide. Nicotine, inhaled with other tobacco smoke ingredients or absorbed in the mucous membrane of the mouth, penetrates the blood and enters all parts of the body. In 7 s, it reaches the brain cells and increases dopamine gain, which causes euphoria and relaxation. Smoke pollutes all parts of the nasal cavity with various poisons and makes the gas exchange process difficult. The nasal mucus destroys and prevents viruses and bacteria growing, and smoking tobacco reduces this function. When excessive smoking occurs, the mucous membrane is formed in the throat, speech becomes difficult, and the voice lurks. With frequent smoking, smokers cough more and more, thereby releasing *smoking mucus* from lung bronchi into the mouth cavity with harmful ingredients. Chronic persistent cough is very common with smokers, and it is considered that about 75% of smokers have chronic coughs. Sinus cavities are constantly coated with tobacco smoke particles, causing the mucous membranes to be chronically inflated and thickened, and the openings are reduced. Many smokers therefore have occasional headaches, especially during colds and other diseases of the upper respiratory organs. The outer part of a smoker's lungs is dark gray to black. Plush tissue looks like a black sponge dipped in diluted tar. Due to the accumulation of tobacco particles on the bronchi, bronchial walls, and stronger bronchial mucus, their diameter narrows and reduces the flowability of the gases. Tobacco smoking causes increased bronchial hyperreactivity [15]. Oxidants and free radicals cause seizure and accumulation of neutrophils in pulmonary microcirculation, as well as accumulation of macrophages in respiratory bronchioles, whereby macrophages are a new potential oxidant reservoir that damages the tissue found [16]. Disordered harmony between ventilation and perfusion of alveolar spaces causes obstructive pulmonary diseases such as bronchitis, emphysema, asthma, bronchitis and bronchiectation. Smoking is a predisposing factor for respiratory infections [17–19] and exacerbation of asthma [20], as well as exposure to tobacco smoke from the environment that increases asthma and its exacerbation [21]. Lung cancer in men occurs between 35 and 75 year

of age. According to world surveys, 95% of throat cancer patients were smokers, and laryngeal cancer grows due to a rise in the number of smokers among women [22, 23]. Smoking increases the risk of obesity and stomach cancer, as well as the development of osteoporosis that changes the physical appearance of a person. Smoking is a risk factor for the development of cardiovascular diseases and adverse effects on endothelial blood vessels [24, 25]. In families with hereditary characteristics of tumor development associated with smoking, there is a high probability of autosomal dominant inheritance of the predisposition for cancer [26–28]. The effect of smoking on reproductive system in men leads to erectile dysfunction [29] and sperm quality [30]. Sexual glands are very sensitive to nicotine and if consumed with alcohol leads to reduced sexual activity. Tobacco poisons damage the molecules inside the cell nucleus, especially the DNA that contains the chromosomes. Tobacco smoke poisons also slow the transmission of nerve impulses and reflex mechanisms that may be delayed or slower. Weight loss proves that tobacco poisons interfere with metabolism because smoking requires more energy and reduces appetite. It affects calcium and insulin activity (insulin resistance), and plasma cortisol is associated with the amount of cigarette smoked in 1 day. People exposed to passive smoking have higher IgE production overall and specific for allergenic patients [31]. Smokers have a higher level of total E immunoglobulin than non-smokers [32]. There is a cumulative effect of early and late exposure to smoking, including tobacco smoke from the environment and the appearance of atopic dermatitis [33].

Because of the impassibility of the smallest blood vessels, a person's appearance is changed, and the skin is pale—yellowish tone, aging faster, does not get the necessary food and oxygen, face and nails become yellow, the lips become blue, body has an unpleasant smell, wrinkles on the face, yellow teeth, and a rough voice.

Nicotine and other components of the cigarette smoke cause different endocrine imbalances and have a negative effect on the pituitary, thyroid, and adrenal glands; testes, egg cells; and their function. It significantly affects the probability of successful pregnancy in healthy women, not just in assisted reproduction cases. Women have adverse effects on reproductive ability, pregnancy-related difficulties, the use of oral contraceptives, menstrual cramps, and urinary bladder. Smoking destroys the human embryo and fetus throughout the entire perinatal development [34, 35], after delivery, during breastfeeding, and throughout the life of the baby. The consequences of its effect are complications in pregnancy, premature birth, reduced birth weight [36], and increased perinatal mortality. Children, during later stages of development after birth, may be prone to allergies, asthma, neurodermatitis, reduced sense of smell, tumor, disruption of intellectual maturation, frequency of aggression, and behavioral disorders. With pregnant women who smoke, a fetus gets a number of harmful substances and poisons through the bloodstream, and it is the only source of nutrients and oxygen. The most common are nicotine and carbon monoxide. Nicotine causes narrowing of blood vessels throughout the mother's body, as well as those in the umbilical cord and causes disturbances in the maturation of the placenta function leading to intrauterine oxygen deficiency. Carbon monoxide produced by smoking is bound to pigments in red blood cells suppressing oxygen, which in turn limits the supply of oxygen to the fetus. After a newborn's birth, the lungs are the most endangered organ, as they are underdeveloped and are unable to supply the whole body with oxygen due to which the newborn experiences breathing problems. Passive

smoking with pregnant women in a smoking family or exposure to tobacco smoke is equally as dangerous as smoking. It has been demonstrated that children whose mothers were smoking during pregnancy have an increased risk of developing respiratory disease later in childhood [37]. It is estimated that 165,000 children die from the consequence of passive smoking per year [38], and it has been shown that passive exposure to tobacco smoke is associated with increased risk of sudden infant death syndrome, malignant diseases, particularly leukemia and lymphoma, cardiovascular disease, slowed psycho-motor development, difficulties in learning, problem behaviors, and obesity [39]. The damaging effects of the mother's active and passive smoking during pregnancy on growth and development of the child are directly related to the number of cigarettes smoked [40, 41].

People begin smoking generally during puberty or early adult age [42], which act as predictors of smoking in adulthood [43, 44]. Companies marketing cigarette brands are aware of their role in the creation of child smokers because there is a high likelihood that they will become lifelong buyers. Children and young people begin to smoke for fun, curiosity, imitation of friends or parents who are smoking, or because of the aspiration to be accepted in society. Their increased use of cigarettes on weekends may be triggered by factors such as socialization with peers, alcohol, and drug consumption at the same time [45, 46]. Studies have shown that adolescents smoke cigarettes even though they know and understand all the adverse health effects [14, 47].

3. Measures in the implementation of tobacco smoking reduction

Health-care systems and surveillance systems can contribute to and monitor efforts to change behaviors. A useful framework for policymakers, advocacy groups, researchers, clinicians, community, and other stakeholders is to understand and implement the most effective lifestyle changes to improve health. The effectiveness of different population strategies should help to inform policy priorities in different countries. Evidence-based interventions could be carried out in combinations, either at the same time or in stages, providing access to improved food, increased physical activity, and reduced tobacco use. If more interventions are made simultaneously, they are much more effective in smoking cessation. At the population level, changes in risk behavior and risk factors can significantly alter health outcomes and the risk of disease. Lifestyle behavior affects a multitude of individual, social, economic, regular mass media, and other environmental factors. Population-based interventions can affect many of these factors. Due to limited funding and other resources for preventive efforts, knowledge and assessment of most evidence-based strategies are essential for promoting priorities.

Academic research centers should focus on projects that would implement knowledge to set policy rules that promise political support and encourage further development.

Smoking behavior is a complex phenomenon, and without help in behavioral interventions, very few people are capable of getting rid of this habit. Behavioral change is divided into the different phases through which people pass to change. Non-effectiveness in taking away a habit is due to the lack of knowledge about behavior, interest, negative experience of

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termination, and lack of motivation. Social support of persons plays a key role in adopting health behavior and defines the enjoyment of love, support, and care by family members and others [48]. Emotional support includes love, compassion, acceptance, and respect for the individual. Instrumental means material, real and objective assistance from others [49]. Research has shown that the most common types of social support are information, emotional, and instrumental support.

Schools and educational systems play an important role in the prevention and education of the harmful consequences of tobacco smoking. Investigations have found that the earliest people begin to smoke is at the age of 11–16. The level of knowledge of this population about the harmful consequences of smoking is relatively low. It is important to educate and raise awareness of the need for smoking cessation for educators, as they should demonstrate with their example and point out to children and young people the smoking hazards.

Public and media are included in the national anti-smoking program under the leadership and coordination of governmental institutions as well as other independent professional associations and individuals. Their role, particularly television, radio, Internet, daily press, and film industry, is extremely important in terms of systematic information gathering and educating citizens about the adverse consequences of smoking tobacco, promoting health education, and anti-smoking messages.

4. Tobacco laws and policies

Legislation should precede an organized public education strategy, implementation plan and infrastructure, and appropriate mechanisms of control and evaluation of implementation. A legislative policy positively affects smoking reduction and tobacco smoke exposure in public places if strictly implemented [50].

Smoking prohibitions or "smoke-free" laws imply public policies, including crime laws and regulations on occupational health, which prohibit the use of tobacco at work and other public places. The smoke-free environment appears to be effective as a control mechanism in reducing the number of current and future smokers [51]. The smoke-free environment protects the non-smoker's health and has a favorable outcome in reducing smoking. Smoking restriction in public places reduces average tobacco consumption by 4–10% [52]. The aim of the tobacco control strategy is not only to stimulate smoking cessation but also to protect non-smokers from passive exposure to tobacco smoke in public places, as well as to protect the rights of non-smokers in a non-tobacco smoke environment [53]. Interventions toward young people and the realization of difficulty in availing tobacco, have no effect on the prevalence of smoking, as opposed to a completely smoke-free environment that is an effective measure of reducing smoking in young people [54]. Young people living in a home without tobacco smoke are more likely (74%) to be non-smokers when compared to peers living with parents who smoke [55]. According to World Medical Association, governments are tasked with helping smokers stop smoking and choosing a healthier way of life, not to be scared by the tobacco industry [56].

5. Conclusion

Smoking is rightly considered one of the biggest public health problems of today. Noxiousness of smoking and tobacco smoke is associated with loss of treatment costs, loss of productivity due to illness, increased disability, premature death, and environmental damage. Public cooperation in the implementation of the smoking ban laws positively affects smoking reduction and tobacco smoke exposure and conveys the message that smoking is not socially acceptable. By using effective tobacco smoking cessation interventions, health professionals and public health professionals should promote smoking cessation by joint efforts. Contemporary smoking tobacco laws are a prerequisite for a systematic anti-smoking campaign. Their application shows good results if they are systematically enforced along with other key factors such as health, education, media, religious communities, and others. They are aimed at banning the promotion of tobacco and tobacco products, promoting health and quality of life, and systematically and permanently informing citizens of the consequences of smoking as well as providing systematic deterrence assistance.

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Section 2

Smoking Prevention and Cessation Counselling

Chapter 4

Smoking Cessation Counselling: What Makes Her or Him a Good Counsellor? Can Counselling Technique Be Deduced to Other Important Lifestyle Counselling Competencies?

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Abstract

Smoking is a major health concern in both developed and developing countries. Smoking cessation counselling is of major importance for health care providers such as physicians, psychologists, nurses and many further therapeutic workers. We recently have demonstrated feasibility of a 4-hour "student-to-student course" (1 hour of scientific background and 3 hours of role plays and intervision) that provided knowledge, skills and attitude to smoking cessation counselling. A key question remains whether such knowledge, skills and attitude can be further deduced to key public health or lifestyle counselling areas like body weight management in overweight persons, management of addictions like alcohol and substance or situation (e.g., Internet and shopping) abuse, management of physical activity/exercise or lifestyle modification like workaholic lifestyle. The authors try to develop such a base for enabling patients to adapt healthier behaviour and give objectives for such counselling situations including the elaboration of clear therapeutic aims for counsellors.

Keywords: smoking cessation counselling, medical education, curriculum, communication skills, motivational interviewing, knowledge, skills, attitude, tobacco cessation education, exercise, physical activity, overweight, compulsive buying, compulsive gambling, workaholism, sleep

1. Introduction

The health sector is increasingly changing from a disease model to a health model. Salutogenesis is a term coined by Aaron Antonovsky, a professor of medical sociology. The term describes an approach focusing on factors that support human health and well-being, rather than on factors that cause disease (pathogenesis) [1]. It has become just as important to consider levels of vitality and healthfulness as degrees of impairment and disability. The quality of our health is strongly influenced by lifestyle habits [2]. The probability of illness and death is directly related to our lifestyle and health behaviour, including tobacco use, nutrition, physical activity or inactivity, alcohol consumption, drug use, sexual behaviour and so forth [3]. Hypertensive and coronary heart disease are in important part related to dietary patterns, smoking, sedentary lifestyle at work and in leisure time [4].

Because lifestyle habits play a central role in influencing the state of health of each individual, the effectiveness of future treatments may greatly depend on the extent to which patients and clients are involved in improving their state of health by actively changing their health habits. Although it can be interpreted discouraging to realize that our behaviour contributes directly to today's health problems, it can also be empowering to know that we can significantly improve the health of our patients and society at large by encouraging them to participate in healthy lifestyle habits [5]. Due to our profession, health professionals such as physicians, psychologists, nurses and physiotherapists come across a large number of people with negative and unhealthy lifestyle behaviour within a therapeutic framework. We can give individual, repeated advice and support to our patients [6]. Performing early intervention to reduce chronicity, increasing intervention rate and improving the quality of counselling could help many more people: probably there is no field of medical education and preventive medicine that contributes to a higher level of health, saves more lives and reduces medical costs than lifestyle changes [7].

Although attention is focused on the risks of unhealthy habits in professional literature and public media, information alone is often not enough to change a patient's behaviour. To get rid of or change deeply rooted negative lifestyle habits, it often requires professional help. Physicians and health professionals frequently pay limited attention to changing the unhealthy habits of patients. They provide information and point out the negative consequences and appeal to the rationality and good intentions of the patients. Knowledge of the risks of unhealthy habits and good intentions to change are only a prerequisite but not enough to change unhealthy habits for most patients. The traditional “fix-it“ role of the general practitioners may be sufficient for the minority of patients who are ready to change, but it is only the first of many steps needed for the majority of patients who are reluctant or ambivalent

about the change [8]. Unfortunately, the dominant but unspoken Cartesian reductionism of modern medicine that views the body as a machine and medical professionals as technicians whose job is to repair that machine is not effective in this field [9]. The existential philosophy of Martin Buber is a milestone in developing the inherent asymmetric clinician-patient interaction towards healing relationships. For Buber, relationship and dialogue are not issues for medicine; rather, medicine is a matter of relationship and dialogue. Healing relationships start from a more symmetrical attitude valuing patient's contribution and power. An abiding commitment to appreciate and foster patient's own competencies and self-confidence leads to a relation based on trust and hope. Hence, building up a healing relationship is mandatory for the patient to become a co-producer of his or her health [9].

However, a problem with smoking cessation is physicians' inadequate preparation for the treatment of cigarette-dependent patients. Special training in smoking counselling to improve the efficiency of medical interventions can increase the frequency and quality of smoking cessation [10] and is often meaningful as a mandatory, targeted and practical training course [11]. WHO guidelines recommend that all health professionals, including students in medical training programmes, should receive education in tobacco use and addiction treatment. Many students in the medical field have nevertheless received inadequate training in the treatment of tobacco consumption and addiction [12]. We therefore developed and evaluated an efficient 4-hour smoking cessation counselling workshop for medical students that will be in the first part be presented in this chapter. It is an easy-to-implement course. As discussed later, much content may also be adapted to other unhealthy lifestyle behaviour counselling situations.

Every switch from a pathogenesis-oriented "patient management" to a "resource integrating salutogenetic mode of patient counselling" requires a fundamental change in the attitude and the role behaviour of health professional towards the patient during the consultation. Digging only for failures and pathology is frequently not helpful. For building up relational trust and needed energy for difficult change processes, it becomes mandatory to build up resources from patient's experience of earlier attempts and by this appreciating and integrating them. This shift of focus might be even more difficult as all pathogenetic distractors may seduce students and health professionals alike to switch back in a traditional top-down or directive role, which may be less helpful in enabling patients to intrinsically change to a healthier lifestyle.

Our chapter is aimed to give students, physicians and other health care professionals an introductory overview on the role of lifestyle behaviour that may affect the health of their patient or clients: be it smoking, be it another subject, to be best prepared to encourage, influence and motivate lifestyle changes.

2. An educational basis: a 4-hour smoking cessation counselling workshop for medical students was performed and evaluated

Behavioural and pharmacological interventions in combination with professional counselling seem necessary to improve smoking cessation rates. In order to ensure benefit, effective courses on preventive medicine content are needed in the curricula of medical students.

Only a part of medical students and later also few physicians and other therapists receive formal training in smoking cessation [13, 14]. Fear and the feeling of being ill-prepared for practice are common for medical but less for psychology students [15]. In order to satisfy the importance of smoking in practically all areas of medicine, a 4-hour comprehensive smoking cessation course was offered for the first time in 2006 at the Medical Faculty of the Saarland University in Homburg/Saar, Germany. The course was thoroughly evaluated, and its results were published [16].

2.1. Description of the course

The course is a compact, comprehensive and interactive 4-hour smoking cessation course for medical students with the aim of teaching students how to offer smoking cessation counselling tailored to the individual willingness and motivation of the smoker including pharmacological therapy. Conducted by a doctoral student, it is thus a course "by medical students for medical students", with full-time supervision by a medical expert with smoking-specific medical education and many years of experience in the field of smoking counselling. The course consists of an introductory theoretical part (1/4) and a practical part (3/4) and is based on the stages-of-change model of Prochaska et al. [17]. This is an evidence-based model of behavioural change that has been developed and tested over the past decade relating to smoking cessation [18]. The theoretical part included a presentation and relevant literature on communication, changes in health behaviour and smoking cessation. In the practical part, the theoretical basics were trained by role plays. Each student received a case report corresponding to a phase of the six stages of change of Prochaska and DiClemente. The counselling interview was played and learned in the form of a role playing according to the "stages-of-change model" by Prochaska and DiClemente, once being the patient and once being the therapist at any stage situation. Depending on the stage, a certain approach is efficient and may therefore be optimal for a successful counselling. Each case was discussed after the role playing, and one received the direct feedback from the students, the doctoral student and the supervisor. The course was voluntary and could be attended by all medical students.

2.2. Evaluation of the course

For the evaluation of the course, pre-course and follow-up assessments (4 weeks after the course) were carried out in five courses with a total of 87 students. Questionnaires and video recordings were blindly evaluated to examine the outcome regarding the competence of the students who attended the course. For the purpose of an efficient training methodology and an appropriate evaluation of the topic of smoking cessation, competence was differentiated into the components "knowledge", "skills" and "attitude" [19] as discussed in more detail in Chapter 3. Another part of the evaluation was the anonymous course valuation directly after the course, which all students completed to optimize future courses.

2.3. Effectiveness of the course

The study confirms that a compact comprehensive 4-hour interactive smoking cessation workshop for medical students is effective in terms of a profound short-term effect on the participants'

counselling abilities. Significant and relevant increases in the competence dimensions of medical students (knowledge, skills and attitude) were measured for successful patient-centred smoking cessation counselling. Knowledge of smoking counselling and smoking cessation has been significantly ($p < 0.001$) and clinically relevantly (Cohen's $d = 2.7$) improved by 81% (see **Figure 1**, bottom left), as also reflected in the subjective anonymous feedback. The sum of the skills was also significantly ($p > 0.001$) and relevantly (Cohen's $d = 1.3$) increased by the course.

In detail, significant gains were observed in the investigation of the motivation to quit smoking, in recommendations for stopping smoking, and suggestions for practical strategies to facilitate smoking cessation and prevent relapse. The evaluation of the duration and amount

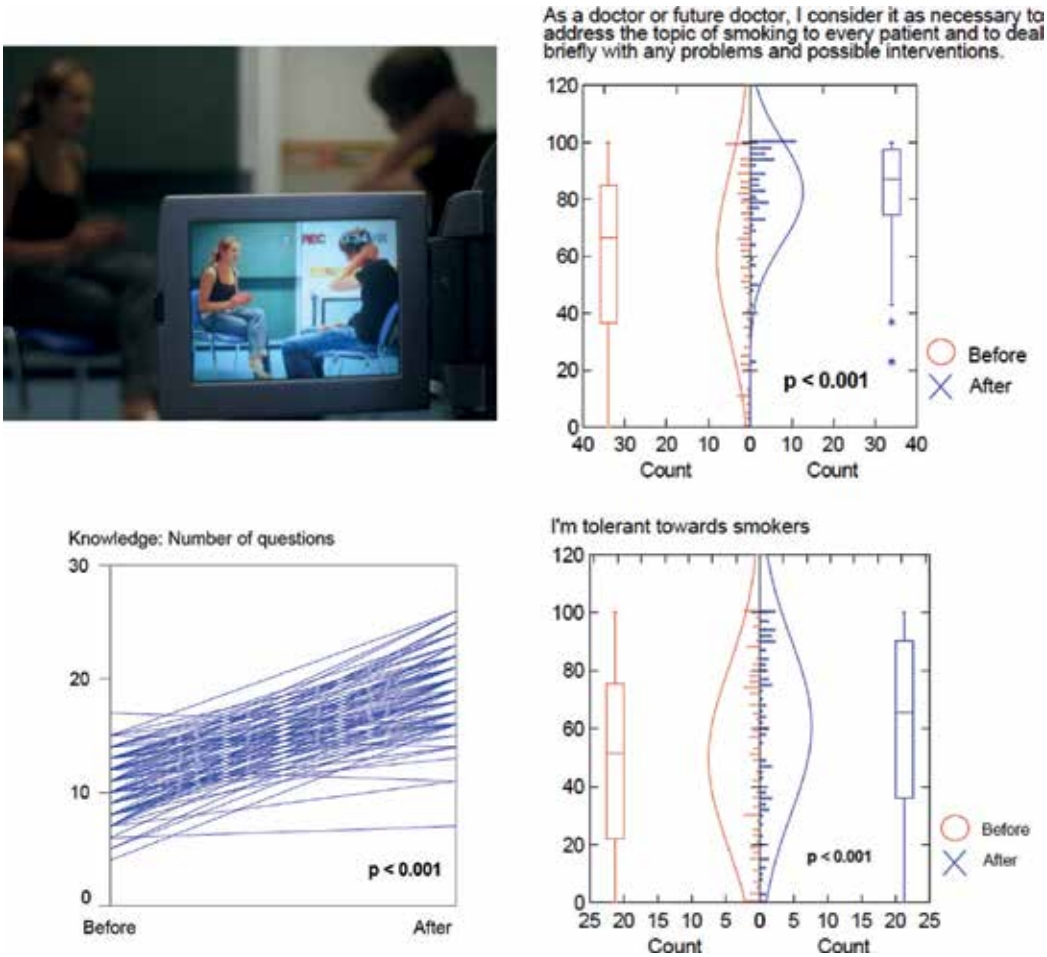

Figure 1. Upper left: assessment of student with a figured patient. The film material was analysed in a blinded fashion to the situation before or 4 weeks after the student course. Lower left: improvement of knowledge according to question scores before and 4 weeks after the course. Upper right: improvement of attitude to address smoking in any consultation before and 4 weeks after the course: a huge shift towards addressing the problem of smoking is observed. Lower right: the course changed another attitude, it led to more tolerance towards smokers.

of tobacco consumption was determined more adequately, and the encouragement of the patient to reflect on present smoking behaviour was driven by the elaboration of arguments for and against smoking. Increased information was provided on smoking cessation and withdrawal process, and nicotine replacement therapy and support were offered for smoking cessation by arranging further appointments.

The attitude of the students changed significantly ($p > 0.001$) and relevantly (Cohen's $d = 0.5–1.3$) in a positive direction. Tolerance, emotional understanding of dependency, respect, sense of responsibility, and interpersonal relationships have been enhanced by the course (see **Figure 1**, upper and bottom right).

The most important results may be this remarkable shift in attitude, probably most influencing in the long-term behaviour towards patients. The course seems highly effective in promoting future physicians' ability in smoking cessation counselling and thus in the long-term retention of medical students' preventive medical competence. The stage-of-changes model proved advantageous for promoting behavioural change in addiction. Such trained competences may foster general counselling competences in further areas as discussed later [20].

3. The competence concepts of knowledge, skills and attitude and their role in counselling

Competence in medicine is defined by various authors as "the usual and reasonable use of communication, knowledge, technical skills, clinical thinking, emotions, values and reflection" [21] or "medical knowledge, care for the patient, professionalism, communication and interpersonal skills, practice-based learning and improvement of what has been learnt" [22]. There are many definitions of the term "competence", but in the end, it amounts to the same concept. It depends on a person's ability to adapt the own abilities to requirements and tasks and to cope with a certain situation. Thus, competence is context-dependent.

3.1. Knowledge: the network of necessary facts we need for professional work

What kind of knowledge is important for smoking counselling? Based on the evaluation points of the smoking counselling interviews in Swiss courses [23, 24], the knowledge questions examined in the study by Purkabiri et al. relate to a basic knowledge of the procedure for counselling, the steps leading to a smoking stop and its effects, cigarettes, nicotine and nicotine replacement therapy [16]. Knowledge is therefore the basis for finding well-founded arguments, identifying the right time for a suitable strategy, drawing up an individual weaning plan and understanding the importance of smoking counselling.

The main topics in the courses of other studies are broadly similar to those of the smoking counselling course presented in this chapter [7, 25]. The contents refer especially to cigarette

smoking and the associated diseases, the pharmacology of nicotine and the medicinal and therapeutic treatment methods of cigarette addiction. The smoking counselling strategies based on the theories of Prochaska and DiClemente [26] and Humair et al. [6, 10, 23, 24, 27] were taught in the Homburg courses.

3.2. Skills: a basis to communication and efficient interaction for therapists

Skills are defined as the abilities gained through learning, practice and experience. By using a good strategy to solve a particular problem, the performer responds appropriately to the task. Of course, there are many strategies, but the term "skills" includes selecting and applying the most effective ones. In the case of smoking counselling, it would be the choice and application of proven strategies for smoking cessation, which the doctor masters through practice and experience.

Skills consist of three main components:

- Awareness of goals or problems and understanding of all relevant factors: Recognizing that smoking presents a high health risk, the problems associated with it and the difficulties of weaning, and how they may be addressed.
- Choosing the reaction: making a decision: How do I react to the problem of smoking and how do I solve the problem of smoking counselling.
- Implementation of the selected strategy/reaction: It usually requires coordination and "timing"—consulting based on efficient strategies at the right time and in the right way.

Thus, the skills include communication factors, emotions and reflective thinking, as presented in the competence definition of other authors [21, 22]. The skills that should be taught in the courses correspond to the ability to give practical counselling to smokers. It is checked whether the student has addressed the most important points of the smoking counselling, such as the recognition of nicotine dependence, information on health and other consequences of smoking, nicotine replacement therapy and so on. Before a visible change of behaviour is possible, an inner sensitization, awareness and motivation process must take place in which the individual explores and sorts out his or her contradictory values, expectations and feelings (ambivalence). The successful handling of this ambivalence reflectively listening in the form of giving feedback what we have perceived and what the patient or client has said and encouraging to come up with possible own solutions or alternative behaviour, and thus, the dissolution of resistance to change is a prerequisite for lasting changes in behaviour (theory of "motivational interviewing" [28]). Elements of motivational interviewing can provide useful help in this important task of counselling. Motivational interviewing [29] is a direct, patient-centred style of counselling that encourages behavioural change by helping patients to explore and resolve their ambivalence [28].

Valuable therapeutic communication is virtually exclusively possible with empathy and symmetry: it is the door to therapeutic intervention. The above-mentioned imparting of empathy,

which is regarded as pivotal for motivational interviewing, is a skill aspect of medicine that has been sometimes in part neglected. Thus, it was crucial to promote this empathy in the courses.

The skills were evaluated, among other things, by video analyses of a counselling interview (see **Figure 1**, upper left). One advantage of the filmed sequences is the precise recording of different dimensions, such as the quality of counselling. The individual film sequences can be viewed as often as you like, and thus, different criteria such as attitude, body posture and facial expressions can be evaluated. Furthermore, video recordings offer the possibility of blinded evaluation, which was done in the study presented here. The examiner did not know whether the recording was before or after the course. In principle, simulated patients are being used much more frequently to test and evaluate the skills of a medical student [21, 30].

3.3. Attitude: the probable key to gain a significant patient relationship and thus patient self-efficacy to elaborate a perspective and reach an important aim

Attitude means "spirit" or "perspective" for an assessment of topics, people and objects. This attitude consists of a cognitive and an emotional component. This mixture of opinions, beliefs and values has to do with respect for the individual and his or her socio-cultural environment, a strong sense of responsibility and care, empathy, patience, perseverance and trust in the opposite person as well as in himself or herself. The "attitude" changes in the course of life through different experiences [31]. For example, someone may have the competence to do a task but not the "attitude" to do it. In other words, being competent does not necessarily mean that you want to achieve something. Just because someone knows how to give advice to smokers [32] and has the skills to do so does not mean that they are necessarily interested in giving advice to smokers and are motivated to do so.

The question, therefore, arises whether health care professionals will use this competence in the future, that is, whether they will give advice to smokers. That depends on whether and how much sense they see in the counselling of smokers. It depends on their point of view, their attitude and their tolerance towards smokers and the recognition of the smoker as a serious person and smoking as an addictive disease. It also depends on whether they recognize the possibility and necessity of helping a patient. Attitude is subjective and based on emotions. It corresponds to what a person thinks and feels and what he or she is motivated to do. And precisely because the best competence is usually only put into practice when there is a correspondingly open, positive attitude towards patients who smoke, so much emphasis is placed on conveying attitudes in smoking counselling courses and measuring their change. In the discussions and in the role playing, the students were able to experience and live the attitude conveyed in the course, "to do everything for the smoker and do nothing against him/her", which was well understandable and was ultimately adopted to a large extent. The remarkable change in attitudes resulting from a smoking counselling course can best provide positive outcomes in the long term, as it is likely that behaviour towards the patient is most strongly influenced in this dimension. Attitude is therefore extremely important in order to be able to apply an existing

competence and thus a prerequisite for a successful result. In the case of smoking counselling, the latter does not only mean the ultimate goal of stopping smoking but also every step towards weaning.

4. General concepts regarding behavioural change in lifestyle

This chapter introduces concepts that are useful for advice. The concepts in this chapter are applied to smoking counselling, but they can also be used for counselling where other behavioural changes are in focus. At the end of the chapter, there is a handout that can be used as an overview and cheat sheet for the consultant.

4.1. The different motivational stages of smoking cessation

Smoking is an addiction, and smoking cessation follows certain stages that merge into each other [26]. The doctor can help with this process, but the patient has to make the decision to quit smoking. The doctor can contribute to this decision by informing the patient using targeted strategies and arguments and leading the smoker to the next stage. This is already considered a success, and in some cases, when the smoker reaches a high level of motivation, the doctor can take the preparations for a smoke stop and accompany him/her.

During the cessation of smoking, the smoker experiences different stages of motivation. The different stages are described in the "stage-of-change" model of Prochaska and DiClemente [26, 33]. Depending on the stage, the probability of becoming a non-smoker increases.

The smoker is initially at the stage of carelessness ("pre-contemplation"), followed by the intellectual debate ("contemplation") and the preparation ("preparation"). In order to help the smoker specifically with smoking cessation, the doctor must find out to what extent the patient is motivated to quit smoking. After that, the doctor can gradually guide him/her to the stage of action. Finally, the maintenance of abstinence after a smoke stop ("maintenance") must be achieved, and a relapse must be avoided by successfully maintaining it [23] (see **Figure 2** and **Table 1**).

In our society, approximately 70% of patients are in the stage of pre-contemplation, 20% are in the stage of contemplation and about 10% are in the stage of preparation [33].

On average, a smoker needs several cessation attempts as "learning episodes"—up to seven times is not uncommon. Since passing through the various stages of smoking cessation process is a learning process, it is not a waste of time, but rather a further success. Ex-smokers are usually considered permanently smoke-free in studies after 1 year abstinence (**Table 2**).

4.2. Five major steps to intervention (the "5 As")

The following five As have proven their worth in smoking counselling. It is an easy to follow scheme that summarizes the practical smoking advice and can serve as an aide memoire ("cheat sheet") during the counselling interview [34].

- *Ask:* Systematically ask smokers at each visit (since when they have been smoking, how much they smoke).
- *Advise:* Tell every smoker to stop smoking. Give him/her some good reasons in a short and concise way.
- *Assess:* Evaluate the patient's willingness to quit smoking.
- *Assist:* When the patient is ready to stop, help him/her. Develop a management plan together.
- *Arrange:* Arrange further appointments.

Every patient should be questioned about his or her smoking habits during a doctor's visit as discussed above. According to the guidelines, the doctor has to advise the smoker to give up smoking with clear and specific words. The following two sentences could be said in this regard: "It is important that you stop smoking immediately. I can help you." or "I am your

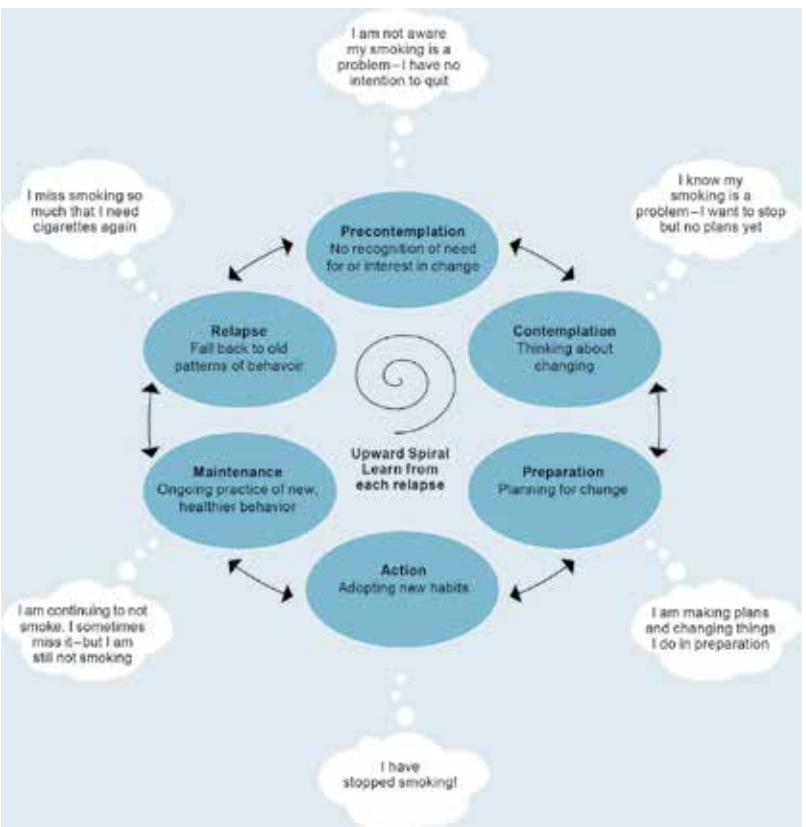

Figure 2. Stage of change model according to Prochaska and Di Clemente.

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Stage	Definition	State
1. Precontemplation	No change planned for the next 6 months	Is currently against a smoke stop Is not very receptive to messages to stop smoking
2. Contemplation	Intention to make a change in the next 6 months	Is ambivalent: sees the advantages of stopping smoking but also of tobacco consumption
3. Preparation	Intention to make a change in the current month	The advantages of stopping smoking outweigh the drawbacks, seeking help and/or advice
4. Action	Smoke stop less than 6 months ago	Risk of relapse, patient actively tries to stay non-smoking
5. Maintenance	Smoke stop more than 6 months ago	Risk of relapse decreases, patient actively tries to stay non-smoking
6. Relapse	Step backwards from any stage to one of the stages 1-3	May contain characteristics of stages 1 to 3

Table 1. Characteristics of the smoking cessation stages [26].

1. Precontemplation	Minimal medical intervention, about 5 minutes: - Raise the subject of smoking - Offer help, leave the decision to the patient - Rediscuss the topic again at the next appointment
2 .Contemplation	Short medical intervention, about 10 minutes: - Enumerate "for and against" the smoking - Discuss barriers to a smoke stop - Offer help, leave the decision to the patient - Support in the form of a new appointment
3. Preparation	Intensive medical intervention, about 15-20 minutes: - Encourage and reinforce the decision of patients - Inform patients about the withdrawal process - Set a smoking stop date with the patient - Suggest to the patient to dispose of his smoking utensils and inform his environment - For strong smokers: Nicotine substitution or alternatives - Planning further consultations - In case of relapse: Identify positive aspects of the trial, identify reasons and suggest strategies to avoid relapse.

Table 2. Objectives of the medical intervention [24].

doctor and must point out to you that stopping smoking is the most important thing you can do for your present and future health. I will support you in this." The advice must be personal and tailored to the smoking person's situation, health and social environment. For example, the danger to children can be mentioned [34].

4.3. The five Rs of motivation

Efforts to quit smoking will only be successful if the smoker has sufficient motivation and a strong will to stop smoking. In patients who are unwilling to quit smoking, the recommendation of a withdrawal program may be premature and has no effect. Details how to increase a patient's motivation to quit using tobacco products are giving in motivational interventions. Motivational interventions can be divided into five basic types, or the five Rs: relevance, risks, rewards, roadblocks and repetition [35].

The five Rs are effective in increasing a client's motivation to quit tobacco use. Using the five Rs, strategy gives clients the opportunity to express their motivation for quitting in their own words and provides the opportunity to tailor their responses to meet the specific needs of the client. The five Rs are discussed in more detail later.

4.3.1. Relevance

The patient is encouraged to look for the reasons why the attempt to quit is personally relevant to him/her. In order to motivate the patient, the inclusion of the personal situation is most effective, that is, status or risk of illness, family or social situation (e.g., children at home), health concerns, age, gender and other important factors for the person (e.g., previous cessation attempts and personal barriers on the way to a smoke stop).

4.3.2. Risks

The patient should identify potential negative effects of tobacco use. The doctor should highlight those risk factors that are the most important in this case. It should be noted that smoking cigarettes and other forms of tobacco use (smokeless tobacco, cigars and pipes) do not eliminate these risks. Examples of acute risks are shortness of breath, exacerbation of asthma, endangerment of the child to be born, impotence/infertility and increased serum carbon monoxide level. And long-term risks are, for example, myocardial infarction, stroke, lung cancer and other tumour diseases (larynx, oral cavity, throat, oesophagus, pancreas, bladder and cervix), chronic obstructive pulmonary disease (chronic bronchitis and emphysema), permanent disability and thus loss of autonomy and need for help. There are also risks for the environment: increased risk of lung cancer and heart disease for the spouse, increased probability that children will smoke, increased risk of children being born underweight or developing respiratory infections and increased risk of sudden infant death [36].

4.3.3. Rewards

The patient should be asked to mention the advantages of smoking cessation. The physician should highlight the advantages that are most important in this case. Examples of potential benefits are

better health, improved sense of taste and smell, financial savings, you feel more comfortable in general, clothes as well as house or car don't smell like cigarette smoke, better breath, no more worrying about a possible smoking cessation and exemplary function towards the children.

4.3.4. Roadblocks

The patient should be asked to name the disadvantages or barriers when stopping and to look for possible countermeasures (problem solution and pharmacotherapy). Typical obstacles are withdrawal symptoms, fear of failure, weight gain, lack of support, depression and desire to smoke.

4.3.5. Repetition

The intervention to increase motivation should be repeated each time patients express doubts about the smoking stop. Tobacco consumers who have experienced unsuccessful attempts to stop smoking should repeatedly be made aware that several serious attempts to stop smoking are the rule [24].

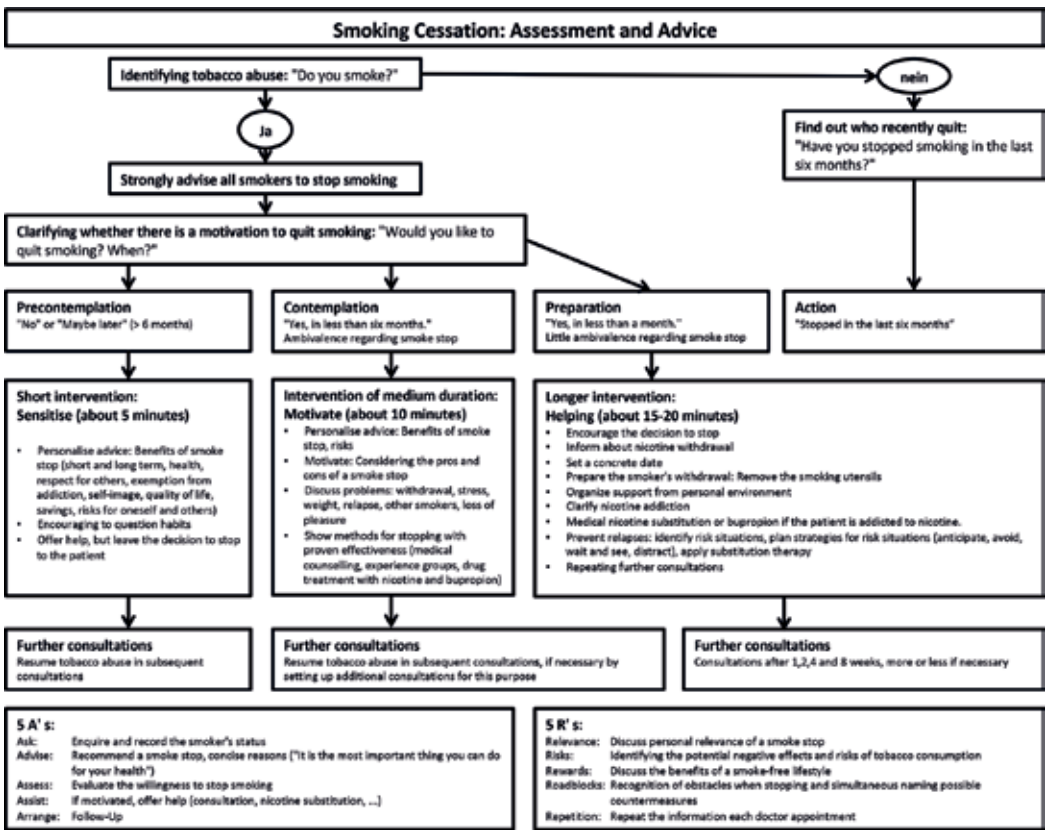

Figure 3. Smoking cessation: assessment and advice. Tobacco weaning—assessment and counselling [23]. This chart shows an aid to the procedure for practical smoking counselling.

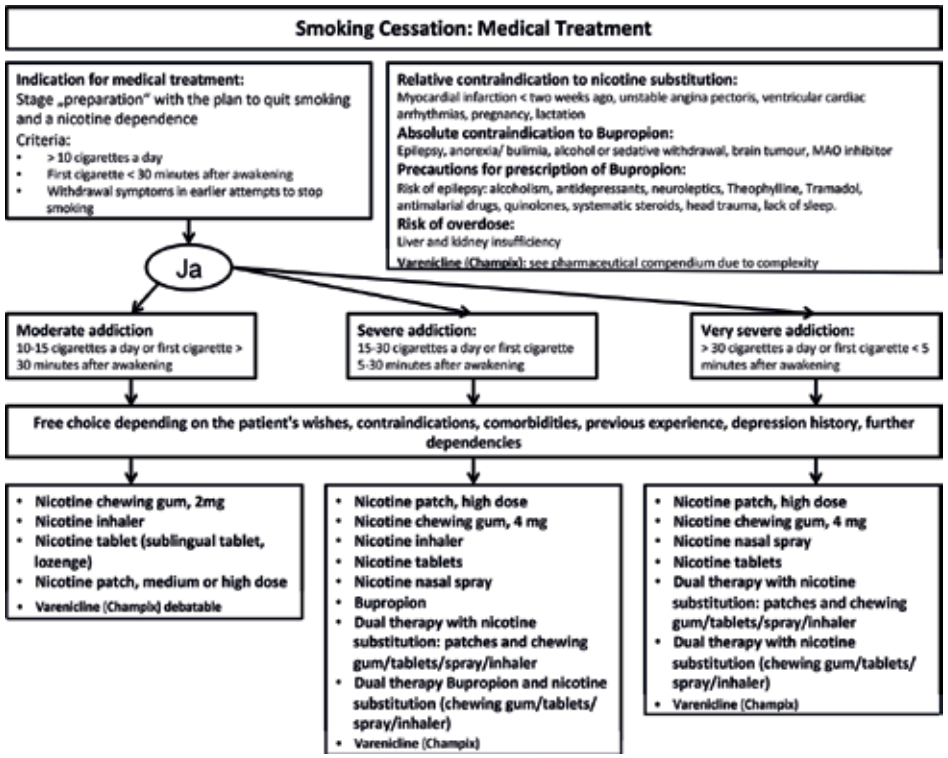

Figure 4. Smoking cessation: medical treatment.

Figure 5. Axes of motivation.

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4.4. The consulting algorithm

Smoking is an addiction; addiction is a disease, and it needs to be treated. Dependence is not imagination. Not only will, but also the right way is decisive (see **Figures 3** and **4**). The physician should give the patient didactic information that can help stop smoking:

- Smoking means nicotine addiction. Therefore, smoking cessation is just as serious as any other drug withdrawal. The will alone is often not enough. The patient must declare the smoke stop to be the highest priority.
- After the cessation date, total abstinence should be the goal.
- The patient should know that unpleasant nicotine withdrawal symptoms can occur. For most people, the symptoms start to appear a few days after smoking has stopped and disappeared after 1 to 2 weeks.
- The patient should be made aware of situations that can jeopardise abstinence at the very beginning of the smoke stop and should, therefore, be avoided. These can be events, states of mind, behavioural habits or activities that are associated with smoking and can, therefore, lead to recidivism (e.g., negative emotions, society of other smokers, alcohol consumption and celebrations).

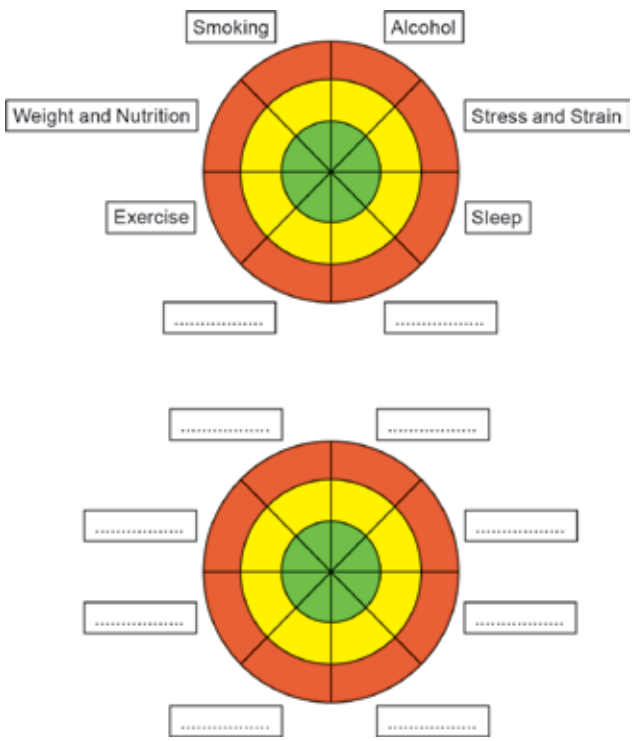

Figure 6. Spider graph assessment of different life style axes.

Figure 7. Therapeutic aspects of life style change.

- The patient should learn appropriate cognitive and behavioural techniques to prevent the desire or need for a cigarette. Examples of cognitive techniques are recalling the reasons for smoking cessation, saying that desire passes and repeating the phrase "smoking is not a solution". During craving, auto-suggestion could be performed, for example, combined with deep inspiration mimicking cigarette inhalation: "And I am so happy that I manage not to need a cigarette".
- Behaviours that can be used to resist the temptation to smoke are leaving the place, dealing with a distracting activity, deeply inhaling and seeking support in the social environment [23].

4.5. Working material

To find out which areas of a patient's or client's behaviour are problematic and which are not and to what extent he or she wants to change this behaviour, patients or clients and health care professionals can use illustrative tools as shown in **Figures 5–7** [37, 38].

5. Why should such behavioural change concepts be applied to other areas of life besides smoking cessation?

In the civilized first world, the change from nomadic life with constant daily activity of moving eight or many more hours a day and with scarce nutrition to a world of highly diverse working activity with much more non-physical, sedentary work, more spare time and with physically passive sitting transportation has become of importance and results in a number of new psychosocial problems. Time spent in sedentary posture is highly associated with waist circumference and cardiovascular risk [39]. In the past century, a dramatic shift from non-industrialized countries suffering from communicable diseases to industrialized or modernized countries burdened with chronic diseases has taken place and also affects densely populated countries such as China and Brazil [40]. This increase in chronic disease rates has created an enormous social, emotional and economic burden. However, society underestimated the problem for a long time focused too much and fostered individualised motor vehicle traffic. It thus sacrificed concepts of walkability and bikeability in most areas from little villages up to hugest towns and exposed children and their residual playing areas as well as the whole society including animals to motor traffic dangers including physical accidents, air, ambient and thus food and water pollution, noise and light.

Already nearly 2400 years ago, sedentary behaviour was considered harmful by Plato, living from 427 to 347 before Christ: "Lack of activity destroys the good condition of every human being, while movement and methodical physical exercise save it and preserve it".

It is only since 2012 that we know the real impact of physical inactivity on health. Inactivity has to be attributed to a shortening of life expectancy of about 10–11 years of individuals

exposed to such lifestyle. It, therefore, became at least as important as smoking in the Western society, leading to the health slogan "Inactivity is the new smoking".

In the light of such social development, it became increasingly important to address more lifestyle questions than only smoking in diverse health-related work. We therefore obtained to broaden the spectrum of her smoking cessation counselling student course to those normal fields and therefore aim to describe as a series of algorithms some "good" aims for a number of common problems including some substance addictions and process addictions of our patients or clients. In the forthcoming sections, he, therefore, describes very prudently, focally and not covering the whole area, and usually with only few evidence, but hopefully with the common sense of a multifaceted team of persons active in patient care, such as conceptual bases.

6. Application to other areas and problems of life: some subjects in more detail

6.1. Sedentary behaviour: "Inactivity is the new smoking"

As already mentioned in Chapter 5, one of the hugest changes in our lives is the change from daily seeking nutrition to a world that offers extremely diverse jobs, many of them very sedentary, and that offers mobility without important individual physical activity, and much spare time, again with in part huge sedentary activities such as TV and Internet. Over 10,000 years, the human body has been optimized and genetically programmed to move frequently [41]. While modern technology has made life easier, it has become an obstacle to physical activity. For example, sitting in front of a laptop all day will make a person less physically prone to move around: of seminal importance may then be residual physical activities besides laptop activity [42]. There is little evidence to suggest that reduced occupational physical activity leads to compensatory increases during leisure time or vice versa. Studies from Europe, the United States and Australia found that adults spend half of the working days sitting (4.2 hours/day) and about 2.9 hours/day of leisure time sitting [39]. Time spent in sedentary posture is associated with waist circumference and cardiovascular risk.

Physical inactivity actually accounts for up to one third of Western world persons reducing life expectancy by about 10–11 years [43] and, therefore, became as central as smoking. Physically inactive children and adolescents develop less cognitive skills than more active cohorts [42]. Sedentary living is a risk factor for global mortality that is associated with arterial hypertension and all consequences of vascular disease including coronary heart disease and stroke and with undiagnosed or diagnosed metabolic disorders especially including diabetes mellitus, obesity, liver disease and overweight. It is furthermore associated with sleep disordered breathing, with muscular and osteo-articular consequences including pain and disability, but also with increased risks of a number of cancers including breast, endometrial,

lung, prostate and colon cancer [43, 44], and probably also with low-grade inflammation [41]. Muscle mass, strength and function seem to play positive roles in recovering from illness, and muscle gain seems even to be anti-inflammatory [45]. Sarcopenia, the deterioration of muscle mass and quality, is a sign of aging but can usually be reversed by training. Muscle is of importance in self-determination in life due to independent deambulation and is necessary to circumvent home care in the elderly or to self-help: standing up without the help of one hand or of two hands differs concerning health prognosis, and many sarcopenic people cannot get up from the floor at all without help.

Physical activity is one of the three biological component modulators of health, that is, physical activity, enough and recovering sleep and adequate nutrition. However, key further elements are psychosocial aspects including relationship and sexuality; family, friends and social surrounding and professional situation. Physical activity is an effective means of curbing the prevalence of child obesity. Fundamental skills are important determinators leading to physically active or inactive behaviour in children: teaching movement skills to young children at the age of about 4 years seem therefore important [46], as motor skills in preschool age and physical activity at school age and probably later are much related [47].

6.1.1. What to recommend for physical activity to patients and clients?

Physical activity and exercise should be viewed as a medication, frequently surpassing health benefits of conventional medications, and in the absence of side effects. They change the underlying mechanisms for physiological functioning and cause increased myocardial oxygen supply, decreased myocardial oxygen demand, increased myocardium electrical stability and overall improved myocardial function [40]. There is a dose-response relationship: health benefits are gained with physical activity and exercise 150 minutes/week, but more health benefits are seen when 300 minutes of moderate physical activity is achieved [40].

Concerning physical activity recommendation, we stick to the World Health Organization recommendation.

In adults, physical activity includes leisure time physical activity, transportation (e.g., walking or cycling), occupational (i.e., work), household chores, play, games, sports or planned exercise, in the context of daily, family and community activities.

The recommendations in order to improve cardiorespiratory and muscular, fitness and bone health, reduce the risk of anxiety and depression and aim at physical activity and exercise as a part of our everyday life [40]:

1. Adults (aged 18–64) should do at least 150 minutes of moderate-intensity aerobic physical activity throughout the week or do at least 75 minutes of vigorous-intensity aerobic physical activity throughout the week or an equivalent combination of moderate- and vigorous-intensity activity.
2. Aerobic activity should be performed in bouts of at least 10-minute duration.

3. For additional health benefits, adults should increase their moderate-intensity aerobic physical activity to 300 minutes per week or engage in 150 minutes of vigorous-intensity aerobic physical activity per week or an equivalent combination of moderate- and vigorous-intensity activity.
4. Muscle-strengthening activities should be done involving major muscle groups on 2 or more days a week.

For older adults, there are further recommendations. They should increase their moderate-intensity aerobic physical activity to 300 minutes per week or engage in 150 minutes of vigorous-intensity aerobic physical activity per week or an equivalent combination of moderate- and vigorous-intensity activity. Older patients, with poor mobility, should perform physical activity to enhance balance and prevent falls on 3 or more days per week.

Some factors can contribute to physical activity. A buddy or a group event, for example, for training can help a person to keep activity due to the group or peer pressure. Appointments should be treated at the same level of priority as professional appointments; weather should in most instances not lead to cancelling exercise. It would be of profit to integrate sports as physical activity or part of it with the partner, although there might be differences in physical strength, in endurance and so on. Sometimes the differences can well be circumvented, for example, by one partner doing only part of a jogging trial, by doing biking with a normal and the less physically performing partner with an electric bike and so on. It would be of profit to plan weekends and holidays aiming at high physical activity level. For very old and highly frail and disabled or highly impaired (e.g., COPD, respiratory failure, cancer, etc.) patients, there are to our knowledge no clear data on minimal muscle mass or on data to prevent falls [48]. Note that one of the first signs of sarcopenia is motoric uncertainty. One reasonable guess of recommendation may be to achieve at least 800 steps per day in order to sustain independence in deambulation. Also, 30 times 1-minute activity per day could be an option.

6.2. Overweight and obesity

Overweight is epidemic in Western civilizations, in the USA affecting nearly 70% and obesity (body mass index $\geq$ 30 kg/m^2) one third of Americans. They result from the imbalance between caloric intake and caloric use [49]. Together with type 2 diabetes and dyslipidaemia, weight problems became one of the most serious health problems worldwide. Weight is crucial, and guidelines for type 2 diabetes may have too much focused on reducing blood sugar levels through drug treatments. One study showed that 9 of 10 people in the trial who lost 15 kg or more could lead their type 2 diabetes into remission [50]. Overweight after smoking is the second most frequent preventable factor for cancer prevention, including not only post-menopausal breast cancer, endometrial cancer, oesophageal adenocarcinoma, gastric cardia cancer, liver, pancreatic and kidney cancer, but also colorectal cancer, cancers of the thyroid, the gallbladder, meningioma and multiple myeloma [51]. Hormonal (adipokines) and growth factor production and metabolism

by fat tissue seem responsible. In the USA, estimated 10% to one third of cancers could thus be preventable by weight control. Estimations also link a body mass increase by 5 kg/m^2 to 10% additional cancer risk [52].

A part of obesity is attributed to physical exercise and activity pattern including work and spare time (sitting, television, computer, aids of car, escalator, electric dishwasher, washing machine, robotic lawn mower, robot vacuum cleaner and electric toothbrush) as above. However, body weight regulation is complex: it is dependent on appetite regulation, nutritional factors and eating behaviours, which includes biological and genetic mechanisms evidenced by heritability not only of modest effect size as in twin studies but also of epigenetic factors, and is not completely under personal control [53]. Why we eat, what and how much we eat are determined by portion size, taste, caloric density and setting. Peripheral signals from our gut and fat stereos relay information in a bidirectional pathway to our brain to tell us when we are hungry or full. While some signals translate into conscious decision-making, many do not. Therefore, many questions remain actually open on what we eat, particularly when food is always available. Known are habit, convenience, opportunity, cost and social factors [49]. Eating patterns are affected by more than the caloric and nutritional value of food. Consumptive behaviours are driven by previous experiences, timing and emotional and pleasurable aspects of eating. Modern foods and drinks are extremely highly processed, frequently with added sugar and fat and extremely appealing, and seductive to consume even more, so that we are a bit "addicted". Often we are unaware that we are eating too much. Usually, we underestimate the caloric intake quite highly even when trying to count calories, as shown by Lichtman et al. who showed the difference of 1000 calories per day on average between perceived intake and actual intake [54]. During aging, a reduction in muscle mass and physical activity leads to a lower metabolism and therefore gives tendency to weight gain that should be adapted with less caloric intake.

Overweight is very clearly visible in most patients. It is in many instances, and society-specific, perceived as an important psychosexual burden. Many patients feel ashamed and, by that, additionally lose quality of life besides that of somatic origin that is inherent with worse mobility, higher exhaustion and so on. There is an evidence of inequities of employment, stigma from health care providers, bias from educators, weight based-stereotypes in the media and rejection and exclusion in interpersonal relationships. This weight discrimination has become one of the most commonly reported types of discrimination in the Western world, and among youth, being overweight is one of the most prevalent reasons for victimization and harassment at school [55, 56].

Individuals experiencing weight stigma are vulnerable to psychological stress, including depression, anxiety, low self-esteem, poor body image, substance use disorder and suicidal thoughts and behaviours. Perhaps the consecutively increased risks of binge eating, unhealthy weight control behaviours, increased calorie intake, avoidance of physical activity, reduced motivation to diet and elevated physiological stress are less intuitive, but consistently demonstrated, all of which can reinforce obesity and weight gain [57, 58]. Such weight discrimination

increases further odds of becoming and remaining of these over time [59, 60]. Reduced quality of health care and avoidance of health care owing to experience of weight stigma have been reported [61]. Actual evidence contradicts public perceptions that fat shaming will provide the concerned individuals with incentive or motivation to lose weight. Instead, such stigma reduces the quality of life and may inadvertently worsen weight-related health outcomes [53].

Both, losing weight and, more important, maintaining weight loss, are two different entities and require distinct skills and attitudes. During weight loss, adaptive responses of metabolic, neuroendocrine and autonomic pathways try to reset weight to the previous higher weight, for example, by the systemic rise of the hunger hormone ghrelin, the decline of leptin as the key adiposities and suppressor of food intake; the resting metabolic rate—the biggest contributor to energy expenditure—declines, and skeletal muscle adapts to become more efficient, requiring fewer calories for the same work [49]. A couple of diseases not only hypothyroidism, lipoedema, M. Cushing, syndrome X and polycystic ovary syndrome hormonal changes but also sometimes depression and chronic stress are associated with more difficulty for weight loss due to a metabolic interference, which is also the case with medications such as sulfonylureas, beta-blocking agents, some antipsychotics, antidepressants, antiepileptics, hormones including anti-conceptive agents and corticosteroids.

Whereas exercise contributes rather little—but sometimes the important part—to weight loss, it plays an important role in maintaining reduced weight. It confers other benefits. It improves insulin sensitivity and blood pressure, redistributes fat and improves mood and well-being. It should, therefore, be part of any lifestyle intervention. However, some patients may be relieved to hear that they do not have to focus so much on exercise. Non-exercise activity thermogenesis is performed by standing more than sitting, doing more little walks by parking the car farther away from a store or the work site and so on. These activities may contribute to a modest increase in energy expenditure and are a way to gradually add in exercise [49].

6.2.1. What are the relevant aims for weight reduction?

Probably the most important aim is the normalisation of the physical and the psychosocial situations. In many patients' situations, this means that normalisation of weight should be the aim of weight reduction.

However, a weight reduction into a range of much better physical performance, of normalisation of actual disease such as pre-diabetes, can sometimes be a compromise. In a considerable proportion of patients, the compromise occurs itself, as it is where the patient arrives within months or years of follow-up with his weight and fitness. We still then have to keep in mind that the whole psychosexual aspect of obesity can highly impact the person's life, relational and social functioning.

There are recommendations that if people wish to have a sustained successful weight loss that then not 10,000 steps but 12,000–15,000 steps would be wise [62]. Note that this is a lot, as the 30 minutes of moderate exercise is only about 3100–4000 steps [63]. In order to build aerobic fitness, the recommendation would be to do 3000 of the daily steps fast. Note that for a 75–80 kg person, 10,000 steps are roughly 500 calories of energy. However

many variables contribute in this fact, for example, speed, denivellation and so on, 10,000 steps more per day would be 3500 calories difference and thus about the burning of 0.5 kg of fat per week (1 kg equals about 7000 calories). In 20 weeks, there would be a potential of 10 kg weight loss (that had then to be stabilized as the second challenge). Note that walking is easier for most overweight persons and is also clearly more recommended than jogging by the associations like the American College of Sports Medicine; walking is less leg joint distressing than jogging, that is, it halves maximum forces for a knee from the equivalent force of about 7 to 3.5 times the own body weight. Beginning with jogging is more difficult, as any activity will give muscle soreness, and initial "overactivity" can thus lead to pain and sufferance, frustration and potentially impede an excellent intention. Nordic walking clearly further reduces those forces; it gives further a more integral muscular training integrating virtually the whole body musculature [64]. Novel data on 40-minutes per day by an electrobicycle to go to work suggest important health profit in sedentary persons.

The surgeon general advises for an increase in physical activity, for example, to increase the steps per day by 20% each week and to get to 10,000 (or 15,000) steps [62]. Apart from Amish and from Tsimane populations, we might derive that more than 15,000 or even more than about 17,000 steps a day might be ideal in terms of health, possibly reflecting that we are not meant to sit around and that for hundred thousands of years we have been on our feet for 8 hours or more. As we all cannot necessarily hit that number, we should aim to reduce sedentary behaviour by interrupting prolonged periods of sitting with walking or standing and reflect our working, transportation and spare time procedures.

As professional life is an important integrating factor and an important axis of self-fulfilment, it may be important, especially in young persons, to best integrate or help to integrate the patient/client in professional life, to manage that the person has fulfilled adequate schools and works, that is, appropriately to her or his professional educational level in the first-job market (see the discussion earlier on inequities of employment) and to help to keep in a long-term stable and good professional life. Workload reduction may sometimes be a solution for physical activity or training in severely obese patients.

Sleep is rather frequently affected in obese and severely obese patients, either by obstructive sleep apnoea hypopnoea syndrome or by adiposity-hypoventilation. As sleep is a huge resource for any physical, mental or intellectual activity, it is of key importance to solve relevant concomitant sleep problems. Patient history and sleep screening tests should in many instances be performed, and arterial hypertension, pre-diabetes or diabetes mellitus should be diagnosed and treated.

6.3. Sleep: your body's best friend

Besides nutrition and exercise, sleep is the probably most underestimated physiological resource for a healthy life. It is of central importance for physical functioning as well as for emotional and intellectual integrity. Deep sleep and rapid eye movement sleep, for example, are the key to learning, be it memorizing facts or be it movements, and thus the key to any good functioning in a society.

Sleep is necessary to be performant. However there are untoward restrictions of sleep for many peoples, whether in school and student life, at weekends, or during the working week. Its consequences are manifold and probably more complex than long time assumed.

Sleep deprivation is one of the most prevalent problems of Western society beginning usually in adolescence. Within a 5-day week, there are a huge proportion of people who have a sleep deprivation of about one night in total. We all need individual sleep time that may bit and rarely considerably differ from about adults 7½–7¾ hours as a rule of thumb. Retired persons usually sleep 20 minutes per night longer and with better sleep quality probably due to less psychological distress [65]. Also, midlife sleep problems are probably associated with cognitive decline [66].

Sleep deprivation has many consequences. Sleep is important for any cerebral process and therefore for any functioning of our physical systems. For example, sleep influences how our bodies recovering and restoration and includes metabolic links, it influences how we learn and memorize facts or movements and it influences our psychic stability. It is also linked to weight gain. Data suggest that 30-minutes less sleeping lead to weight gain: Logically, it is practically impossible to stay committed to a healthy lifestyle if we do not have the energy for it. If we go late to bed or have a restless night, we are more likely to both skip exercise and eat less healthily.Sleep deprivation is thus linked to car and work accidents, relationship troubles, poor job performance, job-related injuries, memory problems and mood disorders. Short sleep duration, obstructive sleep apnoea and overnight shift work are underrecognized as predictors of adverse outcomes after acute coronary syndrome. Increased efforts should be made to identify, treat and educate patients about the importance of sleep for the potential prevention of cardiovascular events [67].

6.4. Other substance addiction than smoking

There may clearly be huge and in part vital impacts for any other substance addictions including alcohol, cannabis, oral, sniffable and inhalable or injectable drugs. There is some basis in common that makes the situation rather similar to counselling like for smoking cessation. It is of note that we know from heroin addicted persons who frequently are also smokers that heroin addiction is not more difficult to stop than smoking, but rather similar. For all those substances, there are peculiarities, for example, the limited evidence of chronic cannabis use and less employment, which is not so clearcut as cannabis is more frequently chronically used in socially less privileged persons. This also seems similar to cannabis and social functioning or the engagement in the developmentally appropriate social roles [68].

6.4.1. Compulsive buying

Shopping has become one of the most popular leisure activities. Shopping centres are increasingly replacing green areas on which people used to play, walk and breathe clean and healthy air. Complex and subtle advertising measures influence our consumer behaviour [69]. In the 1990s, it was highly controversial to realize that buying, like playing or working, can take

on the character of an addiction. Looking at the surface, the compulsive buying is actually lacking some characteristics of other addictions; it is a "clean" addiction, and the people affected are active, successful and performance-oriented and seem to have a perfect grip on their lives, while alcoholics or other addicts are regarded as unstable, weak-willed and externally controlled [70]. It is a rather discreet addiction that is difficult to recognise for outsiders. Compulsive buying rarely changes the personality at least not at an early stage [71].

Compulsive buying can be described as a persistent and recurring, and maladaptive buying of consumer products that disturbs personal, family and professional goals often even burdens them very heavily. People concerned often negate the serious psychological, social and economic consequences [72]. It is not the products purchased that constitute addiction, but rather the experience of buying itself. This alienated behaviour is characterised by increasing internal pressure, which can only be reduced by purchasing the goods. Short-term relief and the resulting feeling of happiness after the purchase are associated with long-term consequences such as financial and social problems in addition to feelings of guilt. Other features are the futile attempts to resist the impulse and the loss of control over buying behaviour [73].

Prevalence rates of 2–8%, and 80–95% of those affected are female. The disorder usually starts at the age of 20 ± 5 years. The course of the disease is generally chronic, although most patients do not receive treatment until two decades later. Systematic studies on therapy are missing [73].

Treatment of compulsive buying is much developing. Pharmacotherapeutic approaches play an important role in the USA [74], whereas the focus in Germany is on behavioural therapy [75]. "Immediate measures" for the establishment of controlled purchasing behaviour include the immediate return of credit and debit cards and the return to cash payments, the targeted analysis of the triggering situations, the avoidance of periods of high seasonal consumption (e.g., the pre-Christmas period), the inventory of one's own possessions and the permanent keeping of this inventory and the regular keeping of budget books. The therapy aims to process and modify situations that trigger compulsive buying and to strengthen the resources in order to prevent unwanted and damaging behaviour in the future [76]. Key behavioural interventions include graduated exposition with reaction prevention, self-regulation techniques and stimulus control as well as cognitive restructuring techniques.Leite et al. examined a number of cognitive-behavioural therapies [77], including cognitive-behavioural model, identifying buying problem behaviours and learning to identify the normal buying, assessment of pros and cons of compulsive buying, financial planning including putting limits or getting rid of credit cards, assessing the "pleasure of buying" behaviour that includes emotional regulation of impulsive feelings, restructuring thoughts; working with exposure; response prevention, work on self-esteem and training in social skills, stress management and problem solving. Relapse prevention and the elaboration of a relapse plan are further key elements ([77], p. 419). Affected persons can also benefit from self-help groups and self-help books.

Theoretically, unresolved addictive behaviour like compulsive behaviour can shift to other compulsive behaviour, for example, to compulsive Internet use or gambling.

6.4.2. Pathological gambling or compulsive gambling (ludomania)

Pathological gambling is a common disorder associated with social and family costs. Much is in common to substance addictions, and much less with impulse control disorders and high comorbidity exists especially with alcohol problems. According to DSM-V, an individual must have at least four of the following symptoms within 12 months [78]:

1. Needs to gamble with increased amount of money in order to achieve the desired excitement;
2. Being restless or irritable when attempting to cut down or stop gambling;
3. Having made repeated unsuccessful force to control, cut back or stop gambling;
4. Being of preoccupied with gambling (e.g., having persistent thoughts of relieving past gambling experiences, handicapping or planning the next venture and thinking of ways to get money with which to gamble);
5. Often gambling when feeling distressed (e.g., helpless, guilty, anxious and depressed);
6. After losing money gambling, of returning another day to get even ("chasing" one's losses);
7. Lies to conceal the extent of involvement with gambling;
8. Having jeopardized or lost a significant relationship, job, education or career opportunity because of gambling;
9. Relying on others to provide money or relieve desperate financial situations caused by gambling.

The lifetime suicide risk seems rather high especially in the early onset of problem gambling, and comorbid substance use and comorbid mental disorders increase its risks. Reports that are up to one in five pathological gamblers attempt suicide underline that the rate is higher than in any other addictive disorder [79]. Treatments involve counselling, step-based programs, self-help, support, medication or a combination; however, no treatment is considered to be most efficacious, and no medications have been approved for this specific addictive disorder. The SSRI paroxetin or the opioid antagonist nalmefene or in comorbid bipolar person lithium seems to have some effect [80, 81]. Similar to "Alcoholics Anonymous", "Gamblers Anonymous" exists and is in the USA, a commonly used resource. Cognitive behavioural therapy has been shown to reduce symptoms and gambling-related urges.

6.4.3. Workaholism

Cultural achievements and processes in economic and social life are based on the work of people, so that work can be understood as a central foundation of human life. Diligence, efficiency and success are considered virtues and foundations of the modern performance

society [82]. Through work, people can satisfy many basic needs, such as social contact and self-realization. As a result, the work fulfils numerous functions in addition to merely securing one's livelihood [83]. However, if a person becomes addicted to work, cannot determine the amount and duration of work, cannot be inactive and develops withdrawal symptoms, if he or she does not work [84], then work behaviour becomes pathological, problematic and is defined as workaholism. This is an uncontrollable, inner compulsion to become active not only in the world of work but also in leisure time and private life, while at the same time, other possibilities of behaviour that are subordinated to working and addictive behaviour similar to substance-related dependencies are shown [85].

The workaholic is mentally and physically addicted to work. He can no longer control his work behaviour (loss of control). He works longer than he intended or in situations in which he cannot or should not work, for example, in the event of illness or in social situations outside the workplace. It is impossible for him not to work (abstinence incapacity), so that he suffers from withdrawal symptoms when he is not working. Workaholics report that in such situations they experience feelings of pressure and tightness, deep sadness or massive states of inner restlessness. However, physical symptoms can also occur during periods of non-work, for example, nausea, headaches or sleep disorders. Due to the development of tolerance, the workaholic has to work much more to achieve the desired effects, for example, a feeling of performance, a feeling of raison d'être, the suppression of feelings of fear or displeasure, hinting to a dose increase. The working behaviour is not only extended quantitatively in the sense of more working hours, but there are also qualitative changes in the working behaviour. For example, many workaholics tend to take care of jobs for which they are not or too well qualified. Psychosocial and/or psychoreactive disorders also occur [86]. The characteristics of workaholism are summarized in **Table 3**.

Results show that workaholics complain significantly more about physical complaints than non-workaholics. In a group of workaholics, 40% report heart pain, 54% report aching limbs, 43% of workaholics suffer from stomach complaints and 58% from general exhaustion such as fatigue or feeling weak. In all aspects, the workaholics can be classified into a greater extent in the group with severe health problems [87].

Workaholics often lack the insight to be ill. Normally those affected do not take help because of excessive work, but the pressure of suffering and the desire for change grow due to the numerous negative consequences that occur [88].

The focus of therapy is on concrete instructions for future everyday life. They include meditation, relaxation, better daily schedule, flexibility and balance. Regular participation in meetings with other workaholics to talk about one's own problems, which could help to overcome conflict situations, also plays an important role. Fundamental is that the addict shows an insight into the disease and thus sees a need for action due to the pressure of suffering. The addict should analyse his or her work behaviour more closely, set new goals and refrain from all unnecessary activities. Finally, a detailed schedule is drawn up, which precisely regulates the daily schedule—in particular breaks and leisure time [89].

Being a slave to work behaviour	The entire imaginative and intellectual space is focused on the work. The thoughts and actions of the person concerned are completely geared to the work.
Loss of control	The workaholic can neither determine the extent nor the duration of the work behaviour by himself. He works out of an inner compulsion.
Inability to be abstinent	The workaholic finds it impossible not to work for a certain period of time.
Withdrawal syndromes	Withdrawal symptoms and possibly vegetative symptoms occur in the case of intentional or forced non-working.
Development of tolerance and dose increase	Since the workaholic develops a certain tolerance towards the amount of work, he must work more and more quantitatively in order to achieve the desired emotional state or the desired state of consciousness.
Psychosocial & psychoreactive disorders	Problems arise in the social and health field.

Table 3. Characteristics of workaholism [84].

Cox et al. [90] described the concept that workaholism is an endogenous addiction. In their leisure time, workaholics should be led to activities that give them pleasure. This would bring the joy itself and then the new experience of leisure activities. This could act as a stimulus for endogenous opiate secretion in a more stress free and therefore healthier way [90].

Specific individual measures are [91] a consistent time management; read and edit emails only 1–2 times a day; set up smartphone-free times, if necessary via an app; set up regular rest periods, for example, with a block as an appointment in the calendar; follow a hobby regularly, for example, minimally 2 times per week; spend time with friends and family on a regular basis; learn passive relaxation techniques, for example, autogenic training, MBSR and meditation and if required, support in individual coaching by business coach.

6.5. To summarize: how can we probably best do our counselling for our patients or clients?

Basis for any more profound professional relationship is, however, a symmetrical communication basis at the same level as the patient or client. The good relationship is essential, and its authentic emotional basis is the only way to be able to put together in question things: The patient or client accepts confrontation only when a symmetry is present and he or she feels a huge quality of relationship. Relationship that functions gives the patients or clients "moments of truth" and the authenticity of feeling and thinking that help him/her to find and believe in

his/her own resources to try a novel way with a life change. Therapy is completely oriented at the patient's or client's resources. This is contrary to physician's assessment of pathologic findings as a deficiency-oriented thought. As therapists, we have to support self-efficacy, that is, the patient's or client's self-belief in his ability to change, for example, focusing on past successes, skills and strengths and promoting self-esteem and confidence. Briefly, therapists can only help to modulate a life change when they have this sound relationship. The necessary skills and attitude for counselling can be learnt and trained, intervised or supervised, with elements from different techniques, including motivational interviewing, as shown earlier.

As soon as a therapist "lives" or communicates non-verbally or verbally a bias, for example, devalorises smoking or overweight, an emotional barrier between therapist and client or patient results that may block emotional openness to address important personal problems in an ensuing discussion. Therapeutic persons should therefore be very open-minded and supportive towards any problem that interferes with the health of a patient or client. Carl Rogers with his humanistic person-centred approach defines three essential basic elements for an optimal conversation: congruence, empathy and unconditional appreciation. Congruence means authenticity, genuineness and transparency on the part of the therapist/consultant. Empathy is the empathetic understanding, the non-valuing approach, that is, the real understanding of a person. Unconditional appreciation is the therapist/consultant's acceptance and sympathy for the feelings and statements of his patient or client. This does not mean that the therapist must necessarily agree with these feelings. But it means that he accepts his patient or client without judgement and prejudice. This attitude should be adopted by every good therapist/consultant [92]. And we should be aware of interfering central roles of relationship and sexuality, of working or occupational situation and of the patient's home situation.

One of the key questions is which aim to propose to a patient or a client. The aim should have a rational background and should be realistic to obtain in a shorter period of time as well as in long term. There might be a mixture of "tailored" aim that not only includes the assumed patient or client resources, but also some ideal should probably be included. This seems important as we sometimes overestimate patients and, especially, also sometimes underestimate their faculties: if a heroin- and nicotine-dependent persons achieve to stop smoking or to stop the heroin consumption, then it is a huge achievement that should also be reflected with the patient or client and highly valorised. In areas like overweight management or physical activity management especially, intermediate aims could also be iteratively adapted, for example, in terms of a shared decision.

In the perspective that patient and client care have changed due to a novel insight into the major role of lifestyle aspects, as discussed, that can tremendously interfere with health status and survival, we are obliged to integrate those in therapeutic situations and to improve communication on them also between different therapists. Setting together aims (e.g., stopping smoking) and defining milestones (e.g., at 3-month re-assessing smoke stop, in case of smoke stop empowerment and 3-month follow-ups, in case of continuous smoking beginning with varenicline and weekly therapist visits for smoking cessation during 2 months,

then monthly follow-ups) when to re-assess patient or client situations and defining the adequate algorithms for patient or client care would probably improve the patient or client outcome.

7. Conclusion

In a short, a 4-hour course could give 87 medical students a wide array of knowledge, skills and attitude towards smoking cessation and evidenced with a vast spectrum of measurements including blinded analyses of conversations with a figured patient. However, as many other areas are of major importance in terms of prevention, the most important question arises whether or not such gain of experience can be derived to other subjects as obesity counselling or exercise counselling in our patient and client care. While many parallels exist between the different areas of counselling on habits and lifestyles, we can only assume that counselling competencies can be deduced to such areas but cannot answer this by data. In common, for any important therapeutic effect, some bases for effective communication and a therapeutic attitude most probably exist. One common denominator is also that there is no way to make people like a lifestyle change. By means of thoughtful and empathic communication and an adequate attitude, we most probably can help for a lifestyle change, ultimately always to make the patient or client feel less threatened by performing this change.

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Conflict of interest

The authors declare no conflict of interest.

Note

This book chapter is intended to develop ideas on increasing knowledge, skills and attitude in different therapeutic settings. It is far from perfect and is aimed to lead to discussion and interaction. The authors highly welcome feedback and criticism to improve this presented work in progress.

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Chapter 5

Evaluation of Effectiveness of Peer Education on Smoking Behaviour

Nurcan Bilgiç and Türkan Günay

Additional information is available at the end of the chapter

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Abstract

Smoking is the most important cause of disability, death and preventable illness in Turkey and all over the world. Cigarette smoking affects a large part of society with increasing frequency and is propagated among young people. Therefore, the tobacco industry targets youth, adolescents and women. Decreasing the age of smoking cigarettes causes exposure of the cigarettes effects to the smokers for a longer period. Adolescents start smoking due primarily to a desire to imitate adults, peer pressure, affectation and easy access to cigarettes. A great impact on the behaviour of human beings is the adolescent peer group. This effect is valid for both risky and safe behaviours. Peer education aims to use positive peer influence on their behaviour since peers are positive models for each other. In recent years, the increase in tobacco consumption has led to increased need for per education. The purpose of this article is to explain how to use peer education in changing the behaviour of adolescent smoking.

Keywords: adolescent, transtheoretical model, peer education, use of tobacco

1. Introduction

The extended period between starting to smoke and the onset of cigarette-related diseases leads to decreased awareness in adolescents about the harmful effects of tobacco on health. Adolescents are vulnerable to tobacco use. Tobacco use starts at an early age, and since the tobacco industry is aware of this fact, they act accordingly. The tobacco industry targets both adolescents and women in an attempt to influence them by using attractive advertisements that focus on freedom, liberation and wealth and being thin, attractive and wealthy [1–4].

Adolescence, an important stage of human life, involves crucial developmental processes through which a person crosses over from childhood to adulthood. These changes may potentially pose pressure on adolescents and cause multidimensional problems necessitating a holistic approach. The majority of adolescents experience some level of emotional, behavioural and social difficulties. On the other hand, adolescents naturally tend to resist any dominant source of authority such as parents and prefer to socialise more with their peers than with their families. Research suggests that adolescents are more likely to modify their behaviours and attitudes if they receive health messages from peers who face similar concerns and pressures [5].

The importance of readily accessible tobacco-produced items, peer pressure, tobacco commercials and price policies in cigarette, cigar and narghile (shisha) on influencing adolescents starting to smoke cannot be overstated. There is a variety of different models of education and behaviour to minimise tobacco consumption among young people and change their perception of behaviours concerning smoking [6].

Approximately one-fourth of adolescents start using tobacco around age 10. Advertisements by the tobacco industry, easy access to tobacco products, low pricing and peer pressure are some of the influences that are effective in starting smoking in adolescents. An additional important factor is that adolescents try to both enhance their image and look 'cool' among their friends [6, 7].

It is essential to design programs for developing life skills to prevent adolescents from beginning to smoke.

These initiatives should include education on—how to say no, oppose insistence, cope with stress, manage anger and communicate and problem solve. These programs may include ways for improving social capabilities, taking a socially responsible approach or involving school-based interventions or peer education.

Influence of peers on youth is certainly of great importance. A peer role model is a friend whose behaviours and suggestions can convince others and that he or she is aware of both the meanings and difficulties of being a fellow peer [8, 9]. The education of peers (peer education) aims at using a positive peer influence on other fellow peers [9, 10].

Peer educators and trainers have to be chosen from among highly respected individuals who are capable of both listening and communicating. They also should have practical and scholarly leadership and role modelling skills. Peer educators and trainers should be able to provide intentional and energetic interference in both positive and negative situations when needed. Also, they should show non-judgmental or unprejudiced attitudes towards their colleagues and clients [10]. However, the process of peer education/training can be negatively affected when the above is not taken into account [11].

2. Who is a peer?

Peer is an Arabic word. The word means 'equivalent in terms of age … of the same age, contemporary'. Peers are individuals in the same social group. A social group can be based on common characteristics including age, gender, background, sexual preference, occupation,

socio-economic and/or health status and a sense of belonging. Peers play a critical role in the psychosocial development of most adolescents. Peer education can be carried out in many different places where young people hang out including schools, universities, clubs, workplaces, streets or sanctuaries [1, 5, 6, 11, 12]. Therefore, peer education is considered a health promotion strategy for adolescents [5].

3. How is peer education defined?

Education is the development of knowledge, attitudes or beliefs and behaviours after a learning process. Peer education is, in the broadest sense, that someone helps a peer. Peer education is a planned educational model that aims to change knowledge, behaviour and attitudes in groups with similar language and behaviour, which have both social interaction and equal status with each other [12].

Peer education is defined as all informal or programmed educational activities that young people are enthusiastic about being educated on and is aimed at increasing awareness of health protection by developing knowledge, attitudes, beliefs and skills.

Peer education is based on educating, volunteering and leading young people on certain subjects and sharing the acquired information with peers. According to Topping, being in a social group and not being a professional teacher is necessary for peer education and peer education is learning while educating. Formen and Cazden stated that, by definition, there should be a difference in knowledge levels of two participants for the interaction to be peer education [13].

Peer teaching is defined as the transfer of the student into the classroom, outside the classroom, under the control of the teacher of knowledge. It is based on the training both volunteer and pioneer young people have received on specific issues and then sharing the acquired knowledge with peers. Educators often use peer aid as it enhances the level of learning within the school, facilitates co-operation rather than competition among students and provides emotional benefits to participating individuals.

Peer teaching is defined as the transfer of knowledge from the student into the classroom or outside the classroom, under the control of the teacher. It is based on the education that volunteers and pioneer young people have on specific subjects and then enables them to share newly acquired knowledge with peers. Educators often use peer help as it enhances the level of learning within the school, facilitates co-operation rather than competition among students and provides emotional benefits to participating individuals [14, 15].

Peer education involves planned activities that are performed on a volunteer basis and not subject to assessment by subjects having the same experience for improving both their knowledge and skills [6, 16].

Peer education is different from classical education methods. In classical education, there is a hierarchical relationship, and knowledge transfer is in one direction, from the educator to the student. As peers do not give each other rewards or punishment, peer groups present an ideal environment for sharing information, learning new things and developing ideas through peer education [14].

Peer education is a popular concept that implies an informal approach, a communication channel, a methodology, a philosophy and a strategy. In the days of kings and queens (in England), peers were nobleman, aristocrats, lords, titled men and patricians. The English term—peer, refers to 'one that is of equal standing with another: one belonging to the same societal group especially based on age, grade or status'. In modern times, the term has come to mean fellow, equal, like, coequal or match according to the dictionary of synonyms (Oxford Thesaurus). At present, the term is used when referring to both education and training [12, 17, 18].

Peer education is considered one of many tools available to reach young people with information and skills. Activities in peer education programs vary widely in the type and frequency of activities, the number and intensity of contacts and the frequency of follow-up. Peer education is often undertaken because it is believed to be both an easy and convenient way to reach a large number of people with information, using inexpensive, volunteer staff. However, when done well, peer education requires intensive planning, coordination, supervision and resources. In fact, there are program costs associated with each element of a peer education program—training, support, supervision, supplies, allowances—all of which require realistic budgeting and careful as well as continuous monitoring [18–21].

Peer education is a chance that affords young people the opportunity to access the necessary information and services about protecting their health. Peer education plays a crucial role in informing young people, about important subjects like smoking, drugs, alcohol, sexual health, contraception and healthy eating habits [22, 23].

4. Methods of peer education

Different methods of peer education have been proposed. The target audience can be reached through a variety of interactive strategies such as small group presentations, role-play or games [13]. Formal delivery of peer education in highly structured settings such as class teaching in schools is also possible. Other methods may include informal tutoring in unstructured settings during everyday interactions or individual discussions and counselling. Various methods are adopted based on the intended outcomes of the project (e.g. communicating information, behaviour modification or development of skills) [5].

Usually, two types of peer education models are applied. The first model is the peer tutoring (cross-age peer tutoring) model. In this model, the teaching peer is older than the learning peer is. The second model is the peer tutoring model in which peers in the same age group, including the whole class, teach each other (same age peer tutoring) [15].

Peer education can be planned in two ways according to the characteristics of peers. In cross-age peer tutoring, the peer tutor is older than the tutee, and in same age peer tutoring, both the tutor and the tutee are the same age [14, 20].

1. Reciprocal peer tutoring: both students are on the same level. In reciprocal peer tutoring, students can work at the same level and both students assume the roles of teacher and student. In the reciprocal peer tutoring model, both teachers and learners can benefit.

2. Cross-level peer tutoring: one of the students is more advanced than the other is. Cross-level peer matching occurs between more successful students and students with learning difficulties. The student with academic superiority always teaches and the other always learn [15, 24].

Tutoring may also take place between true 'peers', students with similar experiences and achievement levels. When student tutors and tutees are in the same class group, share similar levels of expertise and are at similar levels of development, the peer tutoring (PT) arrangement is referred to as being same-level. In same-level tutoring, roles of tutor and tutee may be fixed or may change. These distinctions are elaborated in **Figure 1**.

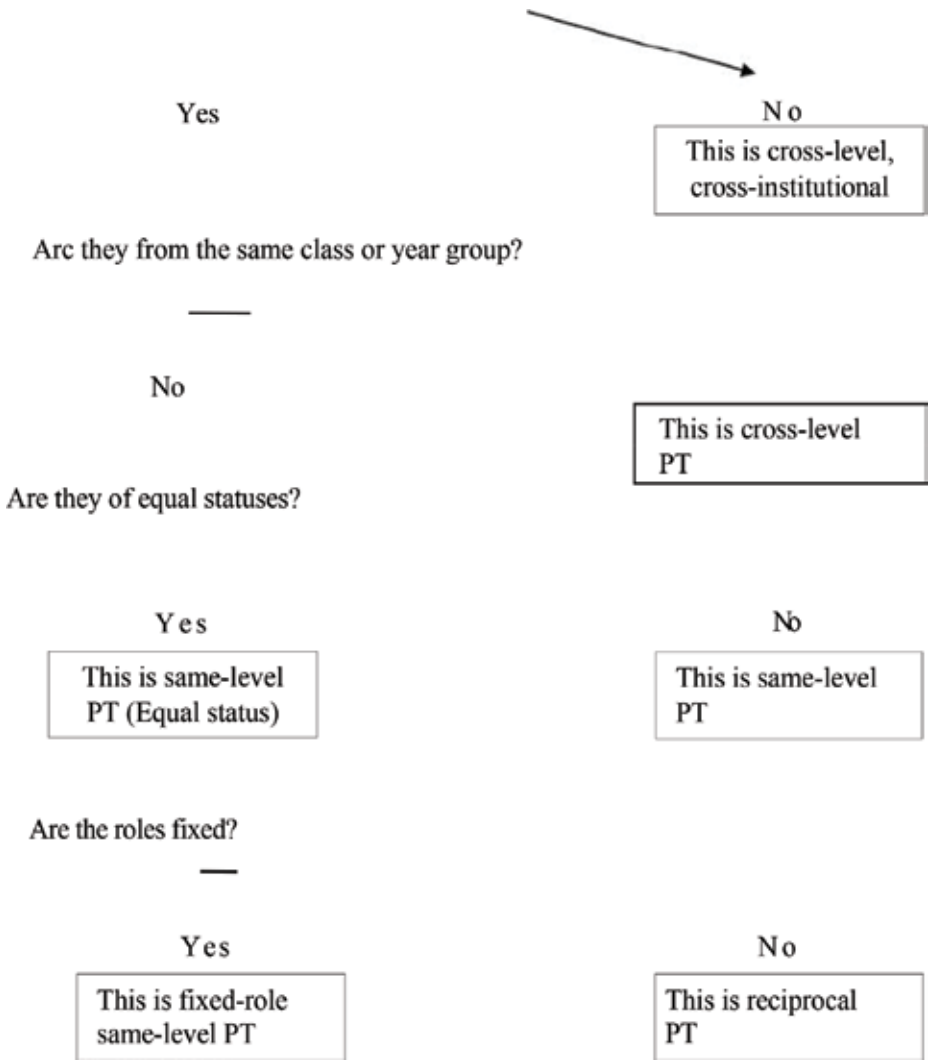

Figure 1. Varieties of peer tutoring ([20], pp. 8).

Topping (1996) identified nine different forms of peer education:

1. small group education at different ages,
2. personalised teaching system,
3. complementary teaching,
4. same age fixed role education,
5. same age peer education,
6. fixed role education between different ages,
7. same age group education,
8. writing with peer help and
9. distance learning with peer help.

5. How should we plan peer education?

The organisation of peer tutoring involves three key variables:

1. the status of participants,
2. the location of the activity and
3. the roles assumed [20].

Peer education programs should be systematically planned. The expectations and methods that would be used in peer education should be clearly explained. Explicitly defined roles and responsibilities of peer tutors during education, their functions and behaviours as role models and feedback and/or reinforcement processes are of tantamount importance. Peer tutors should undergo a planned, well-organised education program for both developing and embracing their teaching skills. It has been determined that the teaching skills of peer tutors will be enhanced if they are promoted [25].

Participants of the peer group should be carefully selected so that the peer group formed will consist of individuals who are enthusiastic and volunteer for participation.

Peer group education should be carried out in a certain systematic way. In interim meetings held with the peer group, primarily the questions 'which activity will be done by whom', 'for what duration (how long)' and 'how' should be answered. In subsequent meetings, These things that can be done and solutions for activities that cannot be performed should be determined again [6, 13, 14, 24].

Problems that may arise before and during peer education should be determined beforehand and the peer tutor should be supported in solving these prospective as well as subsequent problems. After peer education is completed, an assessment should be performed (**Table 1**).

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Organising peer tutoring		
Context	1. Problems	
	2. Support	
Objectives		
Curriculum area		
Selection and matching of participants	1. Background factors	7. Participant partner preference
	2. Age	8. Standby helpers
	3. Numbers of participants	9. Recruiting
	4. Contact constellation	10. Parental agreement
	5. Ability	11. Incentives/reinforcement
	6. Relationships	12. Social reinforcement and modelling
Helping technique	1. General peer tutoring skills	6. Master reference source
	2. General social skills	7. Praise
	3. Drill & practice vs. conceptual challenge	8. Troubleshooting
	4. Combinations of the above	9. Behavioural methods
	5. Correction procedure	10. Mutual gains
		11. Evidential basis
Contact	1. Time	4. Frequency
	2. Place	5. Project period
	3. Duration	
Materials	1. Structure	4. Access
	2. Difficulty and choosing	5. Progression criteria
	3. Availability and Sources	6. Recording
Training	1. Staff training	6. Verbal instruction
	2. Participant training organisation	7. Written instruction
	3. Venue and space	8. Demonstration
	4. Materials and equipment	9. Guided practice and feedback
	5. Participant training content	10. Checking and coaching
		11. Organising and contracting
Process monitoring	1. Self-referral	5. Project process
	2. Self-recording	6. Recognition of successful students
	3. Discussion	7. Fraud
	4. Direct observation	
Assessment of Students		
Evaluation		
Feedback	1. Reassurance	

Table 1. Structured planning for organising peer tutoring.

Situations such as the absence of pre-defined aims and targets, discrepancies between planning and implementation and budget shortfalls negatively affecting peer education may lead to the failure of peer education. In addition, the complexity of the management process, lack of enthusiastic and talented personnel, insufficient equipment and lack of appreciation of tutors may result in the failure of peer education [10].

Various activities can be organised within the scope of peer education. These can be standing activities, concerts, promotional merchandise, theatre, sports events, film screenings, conferences, seminars, workshops, individual or small group information sessions, keeping a diary, radio/TV programs and festivals. Also, print materials-based activities including brochures, booklets and posters can be helpful. In addition, information and communication technologies such as internet-based consulting services, social media sites and telephone helplines can be used [26, 27]. The use of popular people and leaders as peer educators for community-based programs can have significant effects [26].

The success of peer education is closely linked to both good planning and organisation. Peer education application steps are:

1. determination of the subject for peer education,

2. determination of peer tutors and learners,

3. determination of the environment for peer education [skill lab/clinic],

4. education of peer tutors,

5. application and

6. evaluation and feedback [12].

6. What should be the characteristics of peer tutors?

Peer tutors should be chosen from among individuals who have both listening and interpersonal communication skills. They should be individuals who are accepted as well as respected by the group. Furthermore, peer tutors should have leadership qualities; the potential to serve as a role model; have time, energy and desire to work as a volunteer; have the ability to intervene in any positive or negative situation in the group; and not display any judgemental attitude [5, 28].

A peer tutor should be a model to peers by his/her respectable appearance and compliant behaviours. Peer tutors who exhibit healthy behaviours positively affect the same behaviours of peers. Peer tutors and their students should speak a language that is more similar than that spoken by teachers, and traditional mentoring relationships should not be used in peer tutoring [16, 29].

Peer groups encourage young people's interest in peer education programmes and thereby increase their participation in these programmes.

Peers indicate that, away from authority, they can speak, act, discuss and learn more easily in the group. They can complete activities they cannot perform near authority figures, gain independence and express themselves much better. Peers entertain, not threaten their friends. Peers' point of view is magnified in the group: they gain new positive behaviours by interacting with each other. The newly acquired knowledge and skills are also useful in adulthood. Peer education provides young people with not only leadership experience but with leadership qualities and develops both teamwork skills and team spirit. In this way, peer education helps them to affect each other by serving as role models. It allows young people to take responsibility for themselves as well as their actions [16, 27, 29, 30].

In peer education, the educator and the target audience are speaking the same language. Peer education provides easy access to hard-to-access groups. In peer groups, peers feel that they have equal status; a collaborative learning environment is established and they try to help each other [6, 27].

7. Strengths of peer education

In recent years, the many positive benefits of peer education programmes have been recognised. This recognition may be responsible in large part for the increasing popularity of peer education as an alternative to classical educational approaches [12].

The difference between peer education and classical education methods is that classical educational methods involve a hierarchical relationship, creating a power imbalance between educators and students. In classical education, the flow of knowledge tends to be one-sided from the educator to the student. In peer education, on the other hand, peers are not in positions to reward or punish each other; they use the same language and influence each other to create a suitable learning environment [12, 15]. In peer education, it is beneficial that peers encourage each other, individuals feel comfortable with their peers. In peer groups, peers can do what they cannot do near authority, and express their own attitudes more easily [15, 26].

Peer education is described as a less costly method than other training techniques conducted by professionals [26]. Peer education is an important benefit to community health in many areas, including smoking cessation, alcohol and drug abuse reduction, violence prevention and awareness, cancer prevention and early diagnosis, nutrition adequate and balanced food intake and sexually transmitted disease prevention and family planning strategies [27]. A successful peer education programme should be adapted to changing environmental conditions, cultural and economic changes and both health and social conditions.

8. What are the limitations of peer education?

In the classical education approach, knowledge transfer is from the educator to the student. As classical educators oppose using peer education, it cannot be widely used. Besides, impatience of peer educators, disorganisation during the selection of both peer volunteers and their school programme during peer education and insufficient success of peers in chosen subjects

prevent the use of peer education. On the other hand, undesirable interactions between peer tutors and tutees, and the desire of peer tutors to exercise power and authority over tutees also negatively affect peer education [15, 16, 27, 29].

9. The theoretical basis for peer education

When undertaking a peer education programme, objectives are often developed to reinforce positive behaviours, to adopt new, recommended behaviours or to change risky behaviours in a target group. Why and how do people adopt new behaviours? The fields of health psychology, health education and public health provide relevant behavioural theories that explain this process. It is important to be aware of these theories because they provide a theoretical basis that explains why peer education is beneficial. Moreover, these theories can help guide both the planning and design of peer education interventions. The following theories and models of behavioural change are of particular relevance to peer education [22, 30].

Studies are evaluating the effect of peer education programmes on adolescents. The implementation of peer education is based on several cognitive-behavioural models and theories on both the regulation and rehabilitation of preventive health behaviours. Some of the models and institutions that shed light on the studies of health behaviour are the social learning theory, diffusion of innovation theory, theory of participatory education, health belief model, planned behaviour theory, reasoned action theory and transtheoretical model.

9.1. Theory of reasoned action

Developed by Fishbein and Ajzen (1901) to explain behaviour from the perspective of social psychology, and to clarify the intentions of attitudes, along with subjective norms, towards people's behaviour, this theory states that intention is the direct predictor of behaviour. On the other hand, intention refers to tendencies/plans of individuals to exhibit or not to exhibit the behaviour concerned. Ajzen (1991) speaks of intention as a level of desire that the individual has to exhibit a behaviour and the intensity of the effort he/she tends to put forth [31, 32].

This theory states that the intention of a person to adopt a recommended behaviour is determined by:

- A person's subjective beliefs, that is, his or her own attitudes towards this behaviour and his or her beliefs about the consequences of the behaviour.
- A person's normative beliefs, that is, how a person's view is shaped by the norms and standards of his or her society and whether people important to him or her approve or disapprove of the behaviour [18].

9.2. Social learning theory

Asserts that some individuals function as role models of behaviour due to their aptitude for stimulating behaviour changes in other individuals [5]. This theory is largely based on the work of psychologist Albert Bandura [18, 23]. He states that people learn:

- Through direct experience.
- Indirectly, by observing and modelling the behaviour of others with whom the person identifies [for example, how young people see their peers behaving].
- Through training that leads to confidence in being able to carry out behaviour.

This specific condition is called self-efficacy, which includes the ability to overcome any barriers to performing the behaviour.

9.3. Diffusion of innovation theory

This theory argues that social influence plays an important role in behavioural change. The theory considers an innovation as new information, an attitude, a belief or a practice that is perceived as novel by an individual and that can be diffused to a particular group. This theory employs 'opinion leaders' to propagate information, influence group norms and finally act as change agents within the population to which they belong [5]. The role of opinion leaders in a community, acting as agents for behavioural change, is a key element of this theory. Their influence on group norms or customs is predominantly seen because of person-to-person exchanges and discussions [18].

9.4. Theory of participatory education

This theory states that empowerment and full participation of the people affected by a given problem is key to behavioural change [18]. The theory of participatory education has also played an important role in the development of peer education. According to participatory or empowerment models of education, powerlessness at the community or group level along with socioeconomic conditions caused by the lack of power are major risk factors for poor health [5].

9.5. Health belief model

The health belief model was developed in the early 1950s by social psychologists, Godfrey Hochbaum, Stephen Kegels and Irwin Rosenstock. It was used to both explain and predict health behaviour, mainly through perceived susceptibility, perceived barriers and perceived benefits. This model suggests that if a person has a desire to avoid illness or to get well (value) and the belief that a specific health action would prevent illness (expectancy), then a positive behavioural action would be taken concerning that behaviour. An application of this model, the social ecological model for health promotion, views behaviour as being determined by the following:

- Intrapersonal factors—characteristics of the individual such as knowledge, attitudes, behaviour, self-concept and skills.
- Interpersonal processes and primary groups—formal and informal social networks and social support systems, including families, work groups and friendships.
- Institutional factors—social institutions with organisational characteristics and both formal and informal rules and regulations for operation.

- Community factors—relationships among organisations, institutions and informal networks within defined boundaries.
- Public policy—local, state and national laws and policies. This theory acknowledges the importance of the interplay between the individual and the environment and considers multilevel influences on unhealthy behaviours. In this manner, the importance of the individual is de-emphasised in the process of behavioural change [18, 31, 32].

9.6. IMBR model (information, motivation, behavioural skills and resources model)

The IMBR model addresses health-related behaviour in a way that can be applied to and across different cultures. It focuses largely on the information (what), motivation (why), behavioural skills (how) and resources (where) that can be used to target at-risk behaviours [18, 26, 33].

9.7. Transtheoretical model

This model was developed by Prochaska and DiClemente in 1983. Prochaska and DiClemente started to work on a theoretical model involving behavioural change in the 1970s. They observed people who tried to quit smoking without a professional initiative and discovered that people who change themselves went through specific stages when they were trying to reduce or eliminate a high-risk behaviour. A theoretical model is an intentional behavioural change model that focuses on individual decisions. First, the theoretical model used in smoking cessation programs was used for different health behaviours in health improvement programs over time (e.g. overeating and weight control, exercise, coping with stress, substance abuse). This model focuses on helping individuals make willing behavioural changes and understand the process of change. Both preparations and approaches to changing health behaviours are appropriate for the individual's change stage. In traditional behavioural approaches, Prochaska and Velicer (1997) define change as a gradual, continuous and dynamic structure, while change is a sharp and direct conclusion. They argue that individuals do not go directly to new behaviours (smoking cessation) from old behaviours (e.g. smoking) but progress in stages. Using the theoretical model, the stages of change, the process of change, decision-making balance and an organisational scheme of self-efficacy, the individual reveals problem-interaction patterns and problem-solving strategies [34, 35].

10. The importance of peer education in cessation of tobacco use and providing protection

Along with increased knowledge about the harms of tobacco, a variety of studies and control programs should be developed, especially for children and adolescents, to prevent using tobacco products and smoking in particular [30]. Initiatives to prevent tobacco product testing by children and adolescents should be planned to avoid the gradual increase of cigarette use among young people [1–3].

Training programs for cessation of the use of tobacco products and for establishing protection programs may be more effective if applied before children start using tobacco products or leave school. The general aims of these training programs are to increase the number of people who grow up without ever using tobacco products, to reduce illnesses caused by tobacco products, to delay the decision to start using tobacco products, to reduce the risk of addiction to tobacco products and to speed up the cessation of current use of tobacco products [12].

Education establishes non-smoking as an attitude provides an environment without cigarette smoke and creates opportunities for positive role models.

When starting to use tobacco products, peers and role models are quite effective. Peer education models should be used in health projects that aim to reduce the frequency of tobacco product use among young people. The inclusion of peers in anti-smoking training programs will enhance the effectiveness of these programs by ensuring that the main focus is reduced tobacco product use among young people.

There is no hierarchical relationship and power imbalance among peers in classical education methods. One-way communication occurs between peers. In peer education, the fact that peers do not have a position to reward or punish each other helps to create an appropriate environment for implementation, learning and development. Through peer education, enthusiastic young peers are planning activities with their friends to increase their awareness of protecting their own health.

Peer groups encourage young people to take part in a tobacco-related training program and increase young people's participation in the program. Peers are more comfortable talking, moving, discussing and learning about the harms of tobacco and tobacco products by finding a comfortable environment in the group and by being away from the authorities.

Peers who do not use tobacco products and participate in their programs to avoid using their friends are not seen as threats to their parents and school officials by their friends. Peers who are role models to each other and have the ability to influence each other are more easily accessible to power groups.

Peers are beginning to talk about the harms of tobacco use in conversations with their friends on the way, in the garden, in the classroom, in and out of school. Talking about the harms of cigarette with friends after peer education is increasing at the end of the study period. The posters and brochures they prepare inside and outside the school are hanging on the school board, class panels that all students can see. She shares her experiences with her friends and tries to find out what they think. Peers set up groups on social networking sites and share their experiences with their friends over these sites.

Peers are planning a variety of activities to improve their wellness skills, other than conversations about the harms of tobacco use. Peers exchange information about the proposed and implemented solutions to the disruptions that occur in the educational process.

In peer groups, female students are more willing to work in the program and are more active during the program. While girls are more successful in recording the conversations they have made with their friends, especially on the panels, presentations, presentations, and men, men are more successful in creating and maintaining the page in social media.

After peer education, the behaviour of peers changes positively, the ability to cope with the challenges of no-promises, and the difficulties experienced during smoking cessation are developing.

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Smoking was and remains one of the most important public healthcare issues. It is estimated that every year six million people die as a result of tobacco consumption. Several diseases are caused or worsened by smoking: different cancer types, heart disease, stroke, lung diseases and others. In this book we describe the different toxic effects of smoke on the human body in active and in passive smokers. It is also well known that many people who smoke wish to quit, but they rarely succeed. Smoking prevention and cessation are of utmost importance, thus we also describe different strategies and aspects of these issues. We hope that this book will help readers to understand better the effects of smoking and learn about new ideas on how to effectively help other people to stop smoking.

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