REVIEW ARTICLE

https://doi.org/10.1590/1806-9282.2023S124

The challenge of tobacco and nicotine use among women

Paulo César Rodrigues P. Corrêa[1,2*], Roberta Karla B. de Sales[3], Marli Maria Knorst[4], Suzianne Ruth H. Lima Pinto[5], Laura Fonseca Queiroz Ragnini[6], Cristiane Almeida Pires Tourinho[7], Karin Mueller Storrer[8], Maria Enedina C. De A. Scuarcialupi[9], Maria Vera C. De O. Castellano[10], Aldo Agra De Albuquerque Neto[11], Sofia Belo Ravara[12,13,14]

Brazilian Society of Pulmonology/Thisiology

INTRODUCTION

There is growing concern about smoking among women, considering the trend toward reducing the gender gap in some WHO regions. In high-income countries, female smoking is declining but is increasingly concentrated among disadvantaged women[1]. In low-/middle-income countries, the pattern is more complex[1]. Women began smoking after men, lagging behind around 20–30 years[2]. Over time, the increasing female smoking trend narrowed the gender gap, and even more so among youth[1]. This gender gap is narrowest in high- and upper-middle-income countries; in the region of the Americas and Europe, where 1 in 3 tobacco users are female[3]. In contrast, in most low- and low-middle-income countries, particularly in Africa, the Eastern Mediterranean, and the Western Pacific regions, female smoking remains under 5% and is expected to decline[3].

All WHO regions are on track to decrease female prevalence by at least 30% by 2025, with the exception of Europe, which remains the region with the highest tobacco use among females[3].

Female prevalence is lower than males in most countries. According to WHO estimates, in 2020, worldwide prevalence of tobacco use among people aged ≥15 years was 22.3% in both sexes (male: 36.7%; female: 7.8%)[3]. Furthermore, tobacco use will continue to decrease, reaching 20.4% in 2025 (male: 34.3%; female: 6.6%)[3]. Nevertheless, female smoking-attributable mortality is estimated to increase, following the tobacco-epidemic model[2].

Women who smoke have a relatively greater risk of smoking-related diseases than men, such as heart disease, stroke, decreased lung function, COPD, and LC in earlier ages[4]. Remarkably, women face unique problems linked to tobacco and their biological/reproductive life-cycle: female-specific cancers (cancer of the cervix); coronary heart disease, stroke, and thromboembolism (increased risk with oral contraceptives); menstruation (irregular cycles and dysmenorrhea); early menopause; osteoporosis; and impact on fertility/pregnancy and fetus/child development, including the damaging effects of nicotine on brain development. Furthermore, women tend to face more difficulty to quit smoking[4] and are more exposed to SHS[5].

The tobacco companies have targeted women by marketing light, mild, and menthol cigarettes, tailoring their advertisements to women. The greatest health challenge is to avert the increase in smoking among disadvantaged women, which fosters health inequalities[1]. Moreover, the launch of novel nicotine/tobacco products may menace the decreasing worldwide

[1]Universidade Federal de Ouro Preto, Faculdade de Medicina – Ouro Preto (MG), Brazil.
[2]Brazilian Thoracic Society, Tobacco Control Section – Brasília (DF), Brazil,
[3]Universidade de São Paulo, Faculdade de Medicinaa, Hospital das Clínicas, Instituto do Coração, Departamento de Pneumologia – São Paulo (SP), Brazil.
[4]Universidade Federal do Rio Grande do Sul, Programa de Pós-Graduação em Ciências Pneumológicas, Faculdade de Medicina – Porto Alegre (RS), Brazil.
[5]Universidade Federal do Rio Grande do Norte, Faculdade de Medicina – Natal (RN), Brazil.
[6]Instituto do Pulmão de Rondônia – Cacoal (RO), Brazil.
[7]Universidade do Estado do Rio de Janeiro, Ambulatório de Cessação de Tabagismo Pneumologia – Rio de Janeiro (RJ), Brazil.
[8]Universidade Federal do Paraná – Curitiba (PR), Brazil.
[9]Ciências Médicas da Paraíba, Faculdade de Medicina – João Pessoa (PB), Brazil.
[10]Hospital do Servidor Público Estadual de São Paulo (Hospital do Servidor Público Estadual – Instituto de Assistência Médica ao Servidor Público Estadual) – São Paulo (SP), Brazil.
[11]Centro de Estudos Superiores de Maceió – Maceió (AL), Brazil.
[12]Universidade da Beira Interior, Centro de Investigação em Ciências da Saúde – Covilhã, Portugal.
[13]Centro Hospitalar Universitário da Cova da Beira – Covilhã, Portugal.
[14]Universidade NOVA, Escola Nacional de Saúde Pública, Centro de Investigação em Saúde Pública (CISP) – Lisboa, Portugal.
*Corresponding author: paulocrpcorrea@yahoo.com.br
Conflicts of interest: the authors declare there is no conflicts of interest. Funding: none.
Received on March 14, 2023. Accepted on March 22, 2023.

trend of cigarette consumption. Remarkably worrying is the narrower gender gap on e-cigarette use[3].

Female smoking trends are influenced by age, psychological, socioeconomic, demographic, and cultural factors, as well as the tobacco industry's underhanded marketing and promotion strategies[6]. Women are targeted using the same themes as tobacco companies have for decades: their use is tied to independence, stylishness, sophistication, and power[6].

A comprehensive tobacco control program requires coordinated efforts by state and community partners to provide public education and support for policies that work to reduce disparities. There is overwhelming evidence that the tobacco industry understands, exploits, and shapes gender norms. Strikingly, the design and delivery of tobacco control policies/programs remain mostly gender-blind and gender-unresponsive, failing a key determinant not only of risk but also of effective interventions[6].

WOMEN'S HEALTH AND PREGNANCY

Tobacco/nicotine use during pregnancy is the leading preventable cause of maternal and fetal health hazards, and the epigenetic changes caused may be transmitted transgenerationally[7]. TS exposure affects all stages of human reproduction[8]. TS contains thousands of compounds with known toxic effects on reproductive health, such as carbon monoxide, nicotine, and heavy metals, including lead, mercury, and cadmium[9]. Smoking reduces fertility in women, with an apparent conception delay for first-time pregnancies. Smoking women require twice as many *in vitro* fertilization attempts and are more likely to enter menopause earlier than non-smokers[9]. Findings from studies evaluating the effects of in-utero exposure to maternal smoking on later fertility/fecundability are mixed[9].

Conception delays reflect a range of possible adverse effects on reproduction: interference with gametogenesis/fertilization, difficulty on implanting the fertilized egg, or subclinical loss after implantation. Animal studies suggest that tobacco compounds interfere with all early pregnancy events[10,11]. Furthermore, female mice exposed to benzopyrene, a cigarette component, have impaired fertility, potentially related to primary oocyte destruction[12].

Maternal smoking has also been associated with increased risk of ectopic pregnancy, premature membrane rupture, abruptio/previa placentae, and miscarriage; stillbirth, preterm birth, low birth weight, and gestational age small size; and congenital anomalies (such as cleft lip, cardiac septal defects, pulmonary, tricuspid valve and great arteries malformations, pyloric stenosis, and clubfoot) and multiple malformations[8,12,13].

Smoking during pregnancy is responsible for 20% of low-birth-weight babies, 8% of premature births, and 5% of all perinatal deaths[14]. Economic estimates indicate that the costs of perinatal complications are 66% higher for smoking mothers than for non-smokers[15]. Although the most significant benefits for fetal development occur with early smoking cessation, abstinence at any stage of pregnancy or in the postnatal period remarkably improves family health.

Uteroplacental insufficiency is the primary mechanism for fetal growth retardation in pregnant smokers. Nicotine causes uterine/placental vasoconstriction, reducing blood flow, oxygen, and nutrients for fetal delivery.

Smoking also contributes to several causes of premature membrane rupture and miscarriage. Tobacco toxicity decreases macrophage phagocytic capacity, alters mucous membrane immunoglobin A levels, and interferes with local infection control mechanisms. Additionally, it reduces ascorbic acid concentration in amniotic fluid and amino acid transport through the placenta, influencing amniochorionic membrane development[16,17].

Decreased placental synthesis of nitric oxide, a potent myometrial relaxant, increases spontaneous abortion risk. Moreover, decreased platelet activating factor can cause uterine contractions and premature birth since it is involved in initiating/maintaining labor through prostaglandin synthesis[16,17].

SECONDHAND TOBACCO SMOKE HAZARDS IN PREGNANT WOMEN AND FETUS

Secondhand tobacco smoke (SHS) results from the burning of a tobacco product (side stream) and the smoke exhaled by smokers (mainstream). It is estimated that 85% of SHS in an enclosed smoking environment arrives from the tip of a lit cigarette[18]. Cigarettes, pipes, cigars, and hookahs generate SHS. TS contains more than 7,000 toxic chemicals[18]. Exposure to secondhand aerosol from electronic cigarettes also poses harmful effects to bystanders[19].

Despite smoking bans in public places, pregnant women may be exposed to SHS in the home, especially in subpopulations with higher smoking prevalence[15].

Little is known about SHS exposure during pregnancy. SHS surveys in pregnant women in low-/middle-income countries found that daily exposure ranged from 6 to 73%, which was higher than mother smoking in all countries[20]. Being wealthier, having a mother's job, having a higher education, and living in an urban area were associated with lower SHS exposure[20].

The association between social determinants and SHS during pregnancy varies according to the type of exposure[21]. Women at risk of any exposure to smoke during pregnancy include unmarried women who allow indoor smoking[7]. Those most exposed include the younger ones and in early pregnancy[7].

Maternal smoking during pregnancy and SHS exposure are associated with mother/child health hazards, such as infant stillbirth, congenital and respiratory illnesses, neonate lower mean birth weight, length, and head circumference[7,15,20].

A study found that non-smoking women exposed to SHS had an increased risk of stillbirth (23%) and congenital malformations (13%), but not spontaneous abortions[7].

There was a greater risk of discontinuing breastfeeding before 6 months among women exposed to SHS during pregnancy[15]. Postnatal maternal smoking doubled lower respiratory infection risk compared to prenatal smoking[15]. The risk of orofacial clefts was accentuated by 200% when pregnant mothers were exposed to SHS[7], similar to active smoking risk[7].

Women exposed to SHS have a 20% greater risk of giving birth prematurely[15]. SHS exposure in pregnant women may be linked to a 70% increase in mental health disorder risk (depression and suicide ideation)[15].

SMOKING AND CARDIOVASCULAR DISEASE IN WOMEN

Among women, CVDs are the leading cause of death[22]. While tobacco and hypertension are the most preventable CVD risk factors, smoking also increases the risk of arterial hypertension, diabetes, and dyslipidemia, reinforcing CVD risk. Worryingly, tobacco is the main cardiocerebrovascular risk factor in young women, especially if associated with oral contraception, increasing the risk by 30 times compared to non-smoking women without it[23].

The association of smoking with CVD occurs through several mechanisms: endothelial damage/dysfunction, oxidative stress, changes in hemostatic factors, fibrinolysis, inflammation, lipid changes, and vasomotor function; directly influencing pathways related to atherogenesis and thrombosis. Smoking activates the sympathetic nervous system, increasing heart rate and blood pressure and leading to cardiac hypoxia; its effects are more pronounced in women[22].

Women have an increased gender-related risk for CVD. A large sample cohort demonstrated a 25% greater risk for coronary heart disease in female smokers than in male smokers[24]. Female smoking is associated with an increased risk of premature myocardial infarction (<66 years) and higher stroke risk, increasing with higher consumption[25]. A review on hormonal contraception among e-cigarette users has identified no evidence on cardiovascular outcomes, but further research is needed[25]. E-cigarettes expose users to high levels of ultrafine particles that penetrate deep into the lungs[26], triggering inflammatory mechanisms and causing CVD and acute cardiovascular events[26].

Vigitel 2021 showed that 6.4% of adult Brazilian women were exposed to SHS in their homes. SHS exposure is significantly associated with cardiovascular risk; exposed women have a 24% increased risk for CVD, 24% for coronary disease, and 21% for stroke[28].

Smoking cessation has a direct and rapid effect on CVD risk, fostering reductions in inflammatory markers and hypercoagulability, rapid changes in HDL levels, and possibly improving endothelial function. Moreover, a study following 104,519 nurses (1980–2004) showed that women who continued to smoke had higher mortality from coronary and cerebrovascular disease than those who quit less than 5 years ago[28].

The dose–response relationship between smoking and SHS exposure and cardiovascular mortality is nonlinear: light/intermittent smoking, frequent among women, poses similar CVD-risk as daily or higher cigarette consumption; reducing consumption does not warrant cardiovascular health benefits[23].

There is overwhelming evidence supporting smoking cessation and smoke-free environments as key interventions in the prevention and management of CVD[22,27]. Smoking cessation is the single most effective intervention for improving prognosis after a cardiac event, resulting in larger reductions in CVD mortality when compared with secondary prevention[22,29]. Nonetheless, smoking cessation remains neglected in CVD clinical practice[29].

RESPIRATORY HEALTH EFFECTS

The inhalation of harmful substances (such as cigarette smoke and environmental pollutants) is associated with an increased airway inflammatory activity[30].

Tobacco stands as the main and greatest preventable cause of respiratory diseases. The "big five" respiratory diseases (such as asthma, COPD, lung cancer, tuberculosis, and pneumonia/acute lower respiratory tract infections), as well as ILDs, are caused or aggravated by tobacco use or exposure to SHS[30].

ENDS aerosol is not harmless "water vapor"; it contains nearly 2,000 chemicals, mostly ignored[31], including heavy metals, ultrafine particulates, and cancer-causing agents[31].

LUNG CANCER

Tobacco causes 55% of lung cancer deaths in women. Although past research had suggested that women were more likely to

develop cancer at a younger age and with lower smoking rates, recent epidemiologic studies failed to demonstrate this[33]. Remarkably, the tobacco epidemic is not yet fully mature in women, eventually underestimating lung cancer risk in women.

Since 1987, lung cancer has overtaken breast cancer as the leading cause of cancer death among women in the United States and other 28 industrialized countries, due to increased[32] smoking in women. Although LC mortality has reduced over time, it is still higher than deaths from breast, prostate, and colon cancers combined[33].

Nevertheless, studies suggest that sex hormones play an important role in tobacco-induced LC[34] and that disease is not the same in both sexes, especially in terms of modifiable/non-modifiable biological risk factors of carcinogenesis[34].

There is a higher LC incidence in non-smoking young females (female: 15–20%; male: 7–9%)[35]. Adenocarcinoma is the most common histological pattern in women. Finally, women have better five-year survival, regardless of age, staging, and treatment.

TUBERCULOSIS

Smoking is a risk factor for both active and latent TB. Several epidemiological studies have shown that, even after adjusting for sex, age, and educational level, TB is more common in smokers than in nonsmokers[36]. Additionally, smokers with TB tend to have a more severe disease course and worse treatment response and relapse. This association occurs for several reasons, including the reduction in mucociliary clearance and ciliary dysfunction caused by smoking. Furthermore, smoking negatively impacts the function of neutrophils, dendritic cells, and T lymphocytes, which are responsible for controlling the dissemination of tuberculosis[36].

INTERSTITIAL LUNG DISEASES

ILDs are a heterogeneous group of lung disorders; several may lead to progressive pulmonary fibrosis[37]. There is a higher ILD prevalence in women related to connective tissue diseases and hypersensitivity pneumonitis (HP); home environmental antigens are mostly responsible for this gender-difference. Because they can have extremely aggressive behavior, treatable traits for ILDs have been proposed[37]. Smoking is definitely one of them, being linked to more severe HP, worse survival in idiopathic pulmonary fibrosis, and other progressive ILDs. Furthermore, there are some ILDs that are closely smoking-related, such as Langerhans cell histiocytosis, respiratory bronchiolitis, desquamative pneumonitis, and combined pulmonary fibrosis and emphysema.

ASTHMA

In adults, asthma is more frequent in females, and smokers have increased asthma prevalence and incidence. In addition, ENDS use increases symptoms, self-reported diagnoses, and asthma exacerbations. Smoking during pregnancy increases the risk of developing asthma in childhood[38]. Asthma is characterized by airway inflammation and BHR. BHR is higher in smokers compared to nonsmokers and in women than in men[38].

Smoking and SHS exposure significantly impact asthma, worsening disease severity, and control. In asthmatics, smoking accelerates lung function loss, decreases the response to inhaled and systemic corticosteroids, and increases exacerbation risk, hospitalizations, and mortality. Importantly, smoking cessation improves asthma control, reduces inflammation and exacerbations, and improves lung function[38].

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

COPD is diagnosed in the presence of respiratory symptoms, identification of a risk factor, and confirmation of airflow obstruction by spirometry. Smoking is the main COPD risk factor in Brazil, with other factors being SHS, occupational exposure to particulate matter or gases, environmental pollution, alpha-1-antitrypsin deficiency, and low lung growth[39].

A lower lung growth rate was recorded in female adolescent smokers compared to males. Non-smokers lose about 20–25 mL/year of forced expiratory volume in 1 s after the age of 25 years, due to the aging process. In smokers at risk for developing COPD, loss of lung function is more accelerated. Women may be more susceptible to smoking than men, with more symptoms and exacerbations, greater loss of lung function, and more small airway disease with the same smoking history[39].

Quitting smoking improves symptoms such as cough and sputum, reduces exacerbations, and accelerates lung function loss. While early cessation prevents COPD, later cessation increases survival in established disease[39].

GENDER-SPECIFIC TOBACCO/NICOTINE USE CESSATION TREATMENT

TNU is a chronic, relapsing disorder. Treatment combines pharmacological therapy to overcome withdrawal symptoms and behavioral counseling to deal with smoking triggers and behavioral aspects[40].

Women often visit health services due to their biological/reproductive life-cycle and also being caregivers for their children/relatives. This is a golden opportunity to systematically

ask and advise about TNU and SHS exposure and offer support to quit whenever women contact healthcare, even if it is not tobacco-related. Importantly, healthcare providers should record both TNU and the counseling intervention in the clinical files so that they can be followed at a later visit[23,40].

Pregnancy is commonly seen as a unique window of opportunity since women are more motivated to quit and smoking cessation at any pregnancy time results in health benefits. Early smoking cessation during the first trimester obtains the greatest benefits and should be strongly encouraged[16].

Smoking cessation counseling during pregnancy is effective[41]. It should ideally be offered while planning pregnancy to maximize maternal-fetal health benefits, and when pharmacological therapy may be used without restrictions[7]. Behavioral counseling, pregnancy-specific self-materials reinforcing benefits, and psychosocial support are first-line treatment[7,16]. The woman's family, environment, and partner should be approached and involved[7,23]. Regarding the efficacy and safety of cessation pharmacotherapy when used during pregnancy, the evidence is inconclusive. In some countries, nicotine replacement therapy (NRT) is recommended for women who do not succeed in quitting without pharmacotherapy[16].

There are recent literature reviews and smoking cessation guidelines for intervening in pregnancy[7,16,41,42].

The greatest challenge is how to engage and promote smoking cessation among socially disadvantaged women who are more likely to smoke during pregnancy, perceive a less negative attitude toward their smoking, are more tempted in habit-related situations, and profit less from valuable empirical processes of change[43].

Although women smoke fewer cigarettes and with lower nicotine content than men, they have a higher dependence risk, reporting greater physical and emotional dependence on smoking[4]. Particularly, women may be less receptive to nicotine reinforcement effects but more sensitive to non-nicotine conditioned-smoking cues and sensory aspects. Furthermore, women may be less likely to report readiness to quit and less confident in quitting success, usually reporting more difficulty to cope with stress and withdrawal symptoms, especially anxiety and negative mood[23,44].

Women face gender-related barriers and more difficulty to quit: weight gain concerns; menstrual/hormonal cycles influencing withdrawal and metabolization of nicotine/NRT; greater likelihood of depression and mood variability; lack of social support; family work; and caregiver burden. These barriers require tailored behavior interventions[4,23,40,44]. Treatment programs should use a patient-centered approach, focusing on gender-specific barriers, individual concerns, and beliefs; consider the complexity of variables influencing smoking behavior in women; and include intensive multicomponent interventions, i.e., motivational interviewing, problem-solving skills strategies, and cognitive behavior therapy addressing smoking cues, negative mood, weight management, and social support. Group therapy may enhance motivation, self-efficacy, and social support[23].

Women respond less to NRT than men, requiring more intensive behavior counseling[4]. Nevertheless, NRT in women is effective and should be used in combination therapy with other NRT or non-nicotine medications[4,23,40].

Bupropion and varenicline clinical trials demonstrate no treatment-gender interaction, benefiting both sexes equally, indicating that these drugs may be more effective in female patients. Non-nicotine medication reduces craving and desire to smoke and may help weaken smoking-conditioned cues[23]. While varenicline is more effective than bupropion[45], bupropion helps to delay weight gain, as does oral NRT[40]. For women with weight concerns, bupropion and oral NRT are pharmacotherapy options. Additionally, regular, moderate exercise and a healthy diet should be encouraged.

Research addressing gender and pregnancy-specific smoking cessation interventions, developing tailored behavior change strategies, and targeting socially disadvantaged women is much needed.

COGNITIVE BEHAVIOR TREATMENT AND RELAPSE PREVENTION

"When you want to see me again
You'll find me redone, believe me
Eye to eye, I want to see what are you gonna do
When I feel that without you I'm doing too well"
(Chico Buarque, Olhos nos olhos)

Smoking Cessation Outpatient Clinic of the State University of Rio de Janeiro (UERJ) is a multidisciplinary team. All patients undergo individual consultations; participation in group sessions, made up of 10 patients of both sexes and different ages, is optional. Meetings occur every Wednesday. In the first month, there are four weekly meetings of 90 min each, and then a monthly meeting until completing 1 year of follow-up.

Since January 2022, 130 patients have attended UERJ, mostly women (71%), with an average age of 59.7 years. They have been prescribed NRT (patch and/or gum) and Bupropion, available at the Brazilian Public Health Service. Giving voice to these women reveals a rich and challenging universe. They

know why they are there and that they should not smoke; they want to stop but still continue to, i.e., ambivalence.

In the first meeting, tobacco-related diseases are discussed. It is emphasized what is gained by quitting. Addiction is debated without prejudice; the "belief system" is presented, and beliefs that hinder decision-making are discussed, such as "it's too hard and I won't make it". Patients are engaged in choosing the quit method and the treatment plan, reducing drop-out, and strengthening the patient–physician relationship. This shared decision-making builds autonomy and commitment.

Relapse prevention is a cognitive-behavioral intervention designed to prevent or manage relapse. The goal is to teach individuals how to anticipate and cope with high-risk relapse situations. Once relapse has occurred, UERJ's team identifies *when, with whom, where, and what you were feeling when you smoked*.

Patients with psychiatric comorbidities are referred for specialized care; they report fear of failing and a lack of social/family support. Many have low self-efficacy. It is not uncommon to hear: "*I was so nervous that my family ended up buying me cigarettes.*" Many men arrive with their wife or mother. Women come alone.

Post-smoking cessation weight gain (PSCWG) is a concern in both sexes and an independent predictor of quitting failure, mainly among teenage girls and women[4]. A real-world prospective cohort study conducted at the Outpatient Smoking Cessation Clinic of São Lucas Hospital (Porto Alegre, Brazil) between 2010 and 2016 found that 64.6% of the patients who achieved biochemically confirmed continuous abstinence maintained their weight or changed no more than 5% in relation to their baseline weight[46]. PSCWG is not reported by UERJ's patients as a barrier to quitting; many of them lost weight while suffering from COPD or cancer, considering it positive.

Justifying cessation benefits from our point of view does not work. It is their own reasons that will motivate them to quit: whether it is getting fragrant, saving money, using clear nail polish, improving health, or "*I want to hold my grandson*". Treatment should be individualized. The patient-centered approach and active listening with reflection of feelings, among other techniques, elicit the smoker to seek internal motivation to change, i.e., eye to eye. Any doctor can do this, and it is based on the therapeutic doctor–patient relationship.

Cigarettes steal their youth, health, and freedom, and they ask "*How am I going to live without smoking? I smoked all my life*!" In front of us, reality is imitating fiction: "*Do I want to die healthy after being sick all my life?*" Yet, they seek comfort in their tormentors.

Some received brief counseling, mostly guilt-generating or not "supportive": "T*he doctor said I had to stop smoking, but didn't explain how to do it*". When doctors omit advising on tobacco hazards, smoking is allowed. Doctors often fail due to negligence, or poor knowledge.

Group sessions allow participants to understand the behavior change process, create complicity, and foster engagement. They need to learn to live and face daily challenges without cigarettes. Sessions' content includes strategies to manage craving, enhance self-esteem, and make choices. We recognize each person's strengths and celebrate baby steps. Patients associate relapse with a relative's death or illness, divorce, unemployment, stress, and negative mood.

Partnership, compassion, and evocation are also part of our daily lives. Helping them to achieve long-term abstinence is our biggest challenge. They move on with their lives.

"Despite of you, tomorrow will be another day" (Chico Buarque, Apesar de você).

ABBREVIATIONS

BHR: bronchial hyperreactivity; COPD: chronic obstructive pulmonary disease; ENDS: electronic nicotine delivery systems; ILDs: Interstitial lung diseases; LC: lung cancer; SHS: secondhand smoke; TB: tuberculosis; TNU: tobacco and nicotine use; TS: tobacco smoke; WHO: World Health Organization.

WARNING SIGN

This review results from a collaboration between the tobacco control committee of the Brazilian Respiratory Society (SBPT) and the Portuguese Respiratory Society (SPP), with the International Network of Women Against Tobacco (INWAT), Europe.

Paulo Corrêa is the coordinator of the SBPT tobacco control committee.

Sofia Ravara is the coordinator of the SPP tobacco control committee and a board member of INWAT-Europe.

AUTHORS' CONTRIBUTIONS

PCRPC: Conceptualization, Methodology, Project administration, Supervision, Validation, Writing – original draft, Writing – review & editing. **SBR:** Conceptualization, Methodology, Project administration, Supervision, Validation, Writing – original draft, Writing – review & editing. **RKBS:** Project administration, Writing – original draft, Writing – review & editing. **MMK:** Writing – original draft. **SRHLP:** Writing – original draft. **LFQR:** Writing – original draft. **CAPT:** Writing – original draft. **KMS:** Writing – original draft. **MECDAS:** Writing – original draft. **MVCDOC:** Writing – original draft. **AADAN:** Writing – original draft.

REFERENCES

1. Amos A, Greaves L, Nichter M, Bloch M. Women and tobacco: a call for including gender in tobacco control research, policy and practice. Tob Control. 2012;21(2):236-43. https://doi.org/10.1136/tobaccocontrol-2011-050280
2. Thun M, Peto R, Boreham J, Lopez AD. Stages of the cigarette epidemic on entering its second century. Tob Control. 2012;21(2):96-101. https://doi.org/10.1136/tobaccocontrol-2011-050294
3. World Health Organization. WHO global report on trends in prevalence of tobacco use 2000-2025, fourth edition; 2021. https://www.who.int/publications/i/item/9789240039322
4. Perkins KA. Smoking cessation in women. CNS Drugs. 2001;15(5):391-411. https://doi.org/10.2165/00023210-200115050-00005
5. Öberg M, Jaakkola MS, Woodward A, Peruga A, Prüss-Ustün A. Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. Lancet. 2011;377(9760):139-46. https://doi.org/10.1016/S0140-6736(10)61388-8
6. World Health Organization (WHO). Gender-responsive tobacco control: evidence and options for policies and programmes; 2018. https://fctc.who.int/publications/m/item/gender-responsive-tobacco-control-evidence-and-options-for-policies-and-programmes
7. Karadag B, Dağlı E, Yildiz F. Preventing tobacco use and exposure to second-hand tobacco smoke in pregnancy. In: Belo Ravara S, Dağli E, Katsaounou P, et al., editors. Supporting tobacco cessation. European Respiratory Society; 2021:273-86. https://doi.org/10.1183/2312508X.10003520
8. World Health Organization. Recommendations for the prevention and management of tobacco use and second-hand smoke exposure in pregnancy; 2013. http://apps.who.int/iris/bitstream/handle/10665/94555/9789241506076_ionid=0DC301C453D03012873C42714CF1783A?sequence=1
9. U.S. Department of Health and Human Services. How tobacco smoke causes disease: the biology and behavioral basis for smoking-attributable disease; 2010. https://www.ncbi.nlm.nih.gov/books/NBK53017/pdf/Bookshelf_NBK53017.pdf
10. Marom-Haham L, Shulman A. Cigarette smoking and hormones. Curr Opin Obstet Gynecol. 2016;28(4):230-5. https://doi.org/10.1097/GCO.0000000000000283
11. Cooper A, Moley K. Maternal tobacco use and its preimplantation effects on fertility: more reasons to stop smoking. Semin Reprod Med. 2008;26(2):204-12. https://doi.org/10.1055/s-2008-1042959
12. Corrêa PCRP. Tabagismo, hipertensão e diabetes : Reflexões. Rev Bras Clínica Ter. 2003;29(1):19-24.
13. Leite M, Albieri V, Kjaer SK, Jensen A. Maternal smoking in pregnancy and risk for congenital malformations: results of a Danish register-based cohort study. Acta Obstet Gynecol Scand. 2014;93(8):825-34. https://doi.org/10.1111/aogs.12433
14. Schilling L, Spallek J, Maul H, Tallarek M, Schneider S. Active and passive exposure to tobacco and e-cigarettes during pregnancy. Matern Child Health J. 2021;25(4):656-65. https://doi.org/10.1007/s10995-020-03037-8
15. Gould GS, Havard A, Lim LL, Kumar R. Exposure to tobacco, environmental tobacco smoke and nicotine in pregnancy: a pragmatic overview of reviews of maternal and child outcomes, effectiveness of interventions and barriers and facilitators to quitting. Int J Environ Res Public Health. 2020;17(6):2034. https://doi.org/10.3390/ijerph17062034
16. Havard A, Chandran JJ, Oei JL. Tobacco use during pregnancy. Addiction. 2022;117(6):1801-10. https://doi.org/10.1111/add.15792
17. Rogers JM. Tobacco and pregnancy: overview of exposures and effects. Birth Defects Res Part C - Embryo Today Rev. 2008;84(1):1-15. https://doi.org/10.1002/bdrc.20119
18. Fielding JE, Phenow KJ. Health effects of involuntary smoking. N Engl J Med. 1988;319(22):1452-60. https://doi.org/10.1056/NEJM198812013192205
19. Amalia B, Liu X, Lugo A, Fu M, Odone A, Brandt PA, et al. Exposure to secondhand aerosol of electronic cigarettes in indoor settings in 12 European countries: data from the TackSHS survey. Tob Control. 2021;30(1):49-56. https://doi.org/10.1136/tobaccocontrol-2019-055376
20. Reece S, Morgan C, Parascandola M, Siddiqi K. Secondhand smoke exposure during pregnancy: a cross-sectional analysis of data from Demographic and Health Survey from 30 low-income and middle-income countries. Tob Control. 2019;28(4):420-6. https://doi.org/10.1136/tobaccocontrol-2018-054288
21. Do EK, Green TL, Prom-Wormley EC, Fuemmeler BF. Social determinants of smoke exposure during pregnancy: findings from waves 1 & 2 of the Population Assessment of Tobacco and Health (PATH) Study. Prev Med Reports. 2018;12:312-20. https://doi.org/10.1016/j.pmedr.2018.10.020
22. Papadakis S, Pipe A, Vlachopoulos C, Ioakeimidis N, Katsaounou P. Cardiovascular patients. In: Belo Ravara S, Dağli E, Katsaounou P, et al, editors. Supporting tobacco cessation (ERS monograph). Sheffield, European Respiratory Society, 2021; p. 208-28. https://doi.org/10.1183/2312508X.10003020
23. Ravara SB, Matias D, Calheiros JM. Cessação tabágica na Mulher-considerações para uma abordagem de género [Smoking cessation in women: towards a gender-approach]. In: Precioso J, Macedo M, Samorinha C, Araújo C, Eds. Associação Para a Prevenção e Tratamento Do Tabagismo de Braga (APTTB), Braga; 2011:85-107.
24. Tsao CW, Aday AW, Almarzooq ZI, Alonso A, Beaton AZ, Bittencourt MS, et al. Heart disease and stroke statistics-2022 update: a report from the American Heart Association. Vol. 145, Circulation. Lippincott Williams and Wilkins; 2022. p. E153-639.
25. Allagbé I, le Faou AL, Thomas D, Airagnes G, Limosin F, Chagué F, et al. Tobacco-related cardiovascular risk in women: new issues and therapeutic perspectives. Arch Cardiovasc Dis. 2021;114(11):694-706.
26. Glantz SA, Bareham DW. E-cigarettes: use, effects on smoking, risks, and policy implications. Annu Rev Public Health. 2018; 39:215-35. https://doi.org/ 10.1146/annurev-publhealth-040617-013757.
27. Lv X, Sun J, Bi Y, Xu M, Lu J, Zhao L, et al. Risk of all-cause mortality and cardiovascular disease associated with secondhand smoke exposure: A systematic review and meta-analysis. Int J Cardiol. 2015;199:106-15.
28. Kenfield SA. Smoking and smoking cessation in relation to mortality in women. JAMA [Internet]. 2008;299(17):2037. Available from: http://jama.jamanetwork.com/article.aspx?doi=10.1001/jama.299.17.2037
29. Critchley JA, Capewell S. Mortality risk reduction associated with smoking cessation in patients with coronary heart disease: a systematic review. JAMA. 2003;290(1):86-97. https://doi.org/10.1001/jama.290.1.86
30. Hedman L, Ashley D, Filippidis F, Gezer T, Pisinger C. Tobacco is still the most important preventable cause of respiratory diseases. In: Belo Ravara S, Dağli E, Katsaounou P, et al., editors. In: Supporting tobacco cessation. European Respiratory Society; 2021:1-17. https://doi.org/10.1183/2312508X.10001920

31. Corrêa PCRP. No controversy: e-cigarettes are not a treatment for tobacco/nicotine cessation. J Bras Pneumol. 2022;48(5):1-2. https://doi.org/10.36416/1806-3756/e20220283
32. Tanoue LT. Women and Lung Cancer. Clin Chest Med. 2021;42(3):467-82. https://doi.org/10.1016/j.ccm.2021.04.007
33. O'Keeffe LM, Taylor G, Huxley RR, Mitchell P, Woodward M, Peters SAE. Smoking as a risk factor for lung cancer in women and men: a systematic review and meta-analysis. BMJ Open. 2018;8(10):e021611. https://doi.org/10.1136/bmjopen-2018-021611
34. Stapelfeld C, Dammann C, Maser E. Sex-specificity in lung cancer risk. Int J Cancer. 2020;146(9):2376-82. https://doi.org/10.1002/ijc.32716
35. Jemal A, Miller KD, Ma J, Siegel RL, Fedewa SA, Islami F, et al. Higher lung cancer incidence in young women than young men in the United States. N Engl J Med. 2018;378(21):1999-2009. https://doi.org/10.1056/nejmoa1715907
36. Quan DH, Kwong AJ, Hansbro PM, Britton WJ. No smoke without fire: the impact of cigarette smoking on the immune control of tuberculosis. Eur Respir Rev. 2022;31(164):210252. https://doi.org/10.1183/16000617.0252-2021
37. Hoffman TW, Grutters JC. Towards treatable traits for pulmonary fibrosis. J Pers Med. 2022;12(8):1275. https://doi.org/10.3390/jpm12081275
38. Global Initiative for Asthma (GINA). Global strategy for asthma management and prevention, 2022. Published online 2022. https://ginasthma.org/wp-content/uploads/2022/07/GINA-Main-Report-2022-FINAL-22-07-01-WMS.pdf
39. Global initiative for chronic obstructive lung disease (GOLD). Global strategy for diagnosis, management and prevention of COPD 2023 update; 2022. https//goldcopd.org/2023-gold-reports/ 9
40. Fiore MC, Jaén CR, Baker TB, Bailey WC, Benowitz NL, Curry SJ et al. Treating tobacco use and dependence: 2008 update. Content Last Reviewed February 2020. Agency for Healthcare Research and Quality, Rockville, MD. https://www.ahrq.gov/prevention/guidelines/tobacco/index.html
41. Chamberlain C, O'Mara-Eves A, Porter J, Coleman T, Perlen SM, Thomas J, et al. Psychosocial interventions for supporting women to stop smoking in pregnancy. Cochrane Database Syst Rev. 2017;2020(3). https://doi.org/10.1002/14651858.CD001055.pub5
42. Tsakiridis I, Oikonomidou AC, Bakaloudi DR, Dagklis T, Papazisis G, Chourdakis M. Substance use during pregnancy: a comparative review of major guidelines. Obstet Gynecol Surv. 2021;76(10):634-43. https://doi.org/10.1097/OGX.0000000000000943
43. Ruggiero L, Tsoh JY, Everett K, Fava JL, Guise BJ. The transtheoretical model of smoking : comparison of pregnant and nonpregnant smokers. Addict Behav. 2000;25(2):239-51. https://doi.org/10.1016/S0306-4603(99)00029-5
44. Gajos JM, Hawes ES, Chana SM, Mrug S, Wolford-Clevenger C, Businelle MS, et al. Daily adherence to nicotine replacement therapy in low-income smokers: the role of gender, negative mood, motivation, and self-efficacy. Addict Behav. 2023;138:107543. https://doi.org/10.1016/j.addbeh.2022.107543
45. Liakoni E, Benowitz NL. Evidence of the effectiveness and safety of first-line smoking cessation pharmacotherapy. In: Supporting Tobacco Cessation. European Respiratory Society; 2021:97-117. https://doi.org/10.1183/2312508X.10002420
46. Jeremias-Martins E, Chatkin JM. Does everyone who quit smoking gain weight? A real-world prospective cohort study. J Bras Pneumol. 2019;45(1):1-7. https://doi.org/10.1590/1806-3713/e20180010