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POSITION STATEMENT

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Position Statement on the Effects of Cigarette Smoke on Allergic Diseases*

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ABSTRACT

Environmental tobacco smoke exposure is one of the major causes of air pollution that has been suspected to play a role in the rise of allergic diseases worldwide. Studies have shown that exposure to environmental tobacco smoke during pregnancy and during childhood increases the risk of developing not only allergic airway diseases but also allergic skin diseases. Smokefree legislation has been shown to help in decreasing the burden of allergic diseases in the country. Thus, we fully support the implementation of smokefree legislation in the hope that it will continue to decrease the number of smokers in the country. We strive to educate our patients against the ill effects of environmental tobacco smoke and continue to urge current smokers to quit smoking, not only for themselves but for their entire family.

INTRODUCTION

Allergic disease is on the rise particularly in developing countries. There are two possible reasons for this: first, due to an increased disease awareness of physicians, patients, or caregivers; second, due to an abrupt exposure to unknown hazards.[1] Aside from known allergens, environmental triggers such as climate change and air pollution have long been suspected to play a role in the rise of allergic diseases. Pollutants, in particular, have been shown to modulate host response to allergic diseases by increasing IgE and IgG4 and in some cases, have been shown to increase sensitization to certain allergens.[2]

Cigarette smoke is one of the main contributors to air pollution. Based on the Global Adult Tobacco Survey done in 2021, there are about 1.3 billion people who smoke worldwide. Eighty percent (80%) of smokers live in low- to middle-income countries where the Philippines belong. In the 2021 survey of 18,708 Filipino respondents, 19.5% of all adults reported current tobacco use in any form, the majority of which are men (34.7% among men vs. 4.2% among women).[3] Overall, daily cigarette smokers smoked an average of 9.5 cigarettes per day (9.8 among men vs 6.7 among women). The overall average age of initiating daily cigarette smoking among daily smokers aged 15-34 was 19.5 years old (19.5 among men vs 20.1 among women).[3]

The Global Youth Tobacco Survey done in 2019 showed that among the 13- to 15-year-old age group, 12.5% of students – 18.3% of boys and 6.9% of girls – used any form of tobacco products. About 10% of students – 15.3% of boys and 5.1% of girls – smoked cigarettes while 14.1% of students – 20.9% of boys and 7.5% of girls – used electronic cigarettes at the time of the survey.[4] About 77.1% of students who currently smoke cigarettes were able to buy cigarettes from a store, shop, street vendor or kiosk. Of these students, 37.1% were not prevented from buying cigarettes despite their age.[4] For the 13- to 15–year old age group who have never smoked tobacco or any cigarette products, 10.6% of boys and 7.9% of girls – believe that they will enjoy smoking a cigarette later on. Exposure to tobacco smoke at home was at 29.3% while 41.3% of the students were exposed to tobacco smoke in public places that were enclosed. These findings imply that these students are not only exposed to the ill effects of first-hand smoke but to secondhand smoke as well.

Several studies have assessed the effects of cigarette smoke on allergic diseases, particularly asthma, allergic rhinitis and atopic dermatitis. Exposure to cigarette smoke during pregnancy is one of the known risk factors for developing early allergic airway diseases such as asthma and allergic rhinitis. It can also cause exacerbation of allergic airway diseases.

This consensus statement aims to present evidences on the effects of cigarette smoke on asthma, allergic rhinitis and atopic dermatitis and make recommendations based on these scientific evidences.

What happens when we smoke?

When a person smokes a cigarette, the part of the smoke that goes directly into the lungs is called mainstream smoke. The pollutants can directly enter the lungs and get absorbed quickly into the bloodstream as well as body tissues.[5] The smoke pollutants can also come out once exhaled as processed mainstream smoke.[5] Side stream smoke refers to the smoke coming out from the lighted end of the cigarette, cigar, pipe or tobacco burning in a hookah. This is where majority of the pollutants actually come from.[5]

Secondhand smoke is also called "environmental tobacco smoke." It is a mixture of exhaled mainstream smoke and side stream smoke.[4] It is also called passive smoking. It refers to the situation wherein a smoker exhales smoke in front of another person and that person gets to inhale the smoke coming from the mainstream and side stream smoke.

A lot of smokers think that when they smoke away from their loved ones, they are sparing them from the effects of smoking. They are not aware that the pollutants have a tendency to stick to their breath, skin and clothes. Recent studies show that pollutants from cigarette smoke have a tendency to accumulate in dust, objects, as well as in smoker's skin and clothing.[6] Thus, when they go inside their homes and hug their loved ones, the pollutants may transfer and stay there for weeks to months. Loved ones may also inhale the smoke sticking to the hair, breath and clothes of the smoker. This is commonly referred to as "third-hand smoke inhalation."[5-7] Young kids are highly susceptible to this since they are very curious and some have the tendency to put objects in their mouths.[5-7] A recent preliminary study measured nicotine levels in the hands and saliva of children whose parents are smokers and who presented in the emergency room with a potentially smoke-related illness. Nicotine was noted to be present in all children. Cotinine, the predominant metabolite of nicotine, was noted to be present as well except for one child. Younger children showed higher levels of cotinine regardless of nicotine levels.[7]

Another study done by Liu et al, measured children's exposure to smoke by a number of adult smokers living in the home and the level of cotinine, a by-product of nicotine in the children's urine.[8] Eighty percent of children who did not have smoking adults at home still tested positive for smoke exposure.[8] This study thereby gives supporting evidence to third-hand smoke exposure.

How harmful is cigarette smoke?

The cigarette that we have right now is different from the cigarette of the early part of the 20th century. During that time, cigarettes had no filters, no vent holes and fewer added chemicals.[6] What we have now is far more complex with more added chemicals, thus, increasing the risk for cigarette smoke-related diseases. The added filters and vent holes from modern cigarettes make the smoker inhale deeper, thereby, allowing chemicals to reach farther areas of the lungs.[6]

It is estimated that cigarette smoke has more than 7,000 chemicals and chemical compounds, with at least 70 of these chemicals known to cause cancer.[6] Aside from nicotine and tar, cigarettes also contain lead, acetone, battery fluid, ammonia, carbon monoxide and other harmful chemicals. Pollutants are released from chemical combustion, that is the burning of tobacco and paper; some may still be in the gaseous phase while some are released as particulate matter.[5] Particulate matter consists of tiny particles of diverse chemical composition, which may or may be not visible to the naked eye.[5] Particles smaller than 2.5 micrometers (PM2.5) are major components of cigarette smoke and can enter deep into the lung where they can cause serious health problems.[5]

Needless to say, all cigarettes are harmful and there is no level of inhalation or exposure that is safe.

Link of Nicotine to Cannabis and Liquor

Nicotine use has also been linked to increase likelihood to engage in more risk taking behavior such as use of cannabis and liquor. According to a study done at the Columbia University Mailman School of Public Health, which included 50,000 US adolescents, vaping nicotine is strongly linked with increased likelihood of high levels of engaging in binge drinking and cannabis use. Study results showed that those who smoked traditional cigarettes were 8.03 times more likely to use cannabis, while those who used vapes were 20.3 times more likely to use cannabis.[9] Adolescents who are both traditional cigarette smokers and vapers were found to be 40.1 times more likely to use cannabis.

The association between smoking and binge drinking is also high. Those who smoked tobacco were said to be 5.6 times more likely to engage in binge drinking while those who used vapes were 20.6 times more likely to engage in binge drinking. Dual users were said to 36.5 times more likely to engage in binge drinking. Binge drinking, cannabis and nicotine use in combination has been shown to lead to deleterious effects in health.[9]

The association between smoking, cannabis and binge drinking leads to more deleterious health conditions and may increase the risk for development of allergic diseases.

EFFECTS OF SMOKING ON ALLERGIC DISEASES

Babies who were exposed to environmental tobacco smoke during pregnancy have not only been shown to have more respiratory infections during their first 2 years of life, but have also been shown to be more prone to developing allergic diseases early in life. They have not only been shown to be more prone to allergic respiratory diseases, but may be more prone to skin allergies as well.

Atopic Dermatitis

Atopic Dermatitis is a chronic relapsing inflammatory skin disease and a growing health concern, especially in children, because of its high prevalence and associated low quality of life.[10] The etiology of atopic dermatitis (AD) is multifactorial with interactions between genetic predisposition, environmental triggers and the immune system, contributing to the pathophysiology of AD.[10,11]

Prenatal exposure

Environmental tobacco smoke (ETS) exposure during the critical period (i.e., during pregnancy and early life) is considered as a potential cause of the development of childhood allergic diseases.[1] Children are more vulnerable to ETS exposure than adults are. Atopic dermatitis was found to be highly correlated with ETS, especially among children whose mothers had smoked during pregnancy and/or in the first year after birth. According to the study by Wang et al., the effect of prenatal tobacco smoke exposure on the risk for AD may be mediated through cord blood DNA methylation wherein they measured cord blood cotinine levels which correlated with prenatal smoke exposure.[12]

The probable mechanism by which ETS lead to AD development is via inducing oxidative stress in the skin, leading to skin barrier dysfunction or immune dysregulation. Benzene is one of the chemicals, implicated in the etiology of AD. Albeit an ubiquitous environmental chemical, non-occupational exposure to benzene mainly results from active or passive tobacco smoke exposure in homes or public places.[13] Prenatal benzene exposure is associated with decreased cord blood regulatory T (Treg) cell numbers and a subsequent increased risk for the development of AD in early childhood.

Treg cells, or regulatory T cells, are a type of immune cell that plays a vital role in maintaining equilibrium and preventing hyperreactivity of the immune system.[14] By inhibiting the activity of other immune cells and preventing them from responding to benign substances, they serve as mediators.

Treg cells are crucial in the context of atopic dermatitis, a prevalent skin disorder characterized by itchy, irritated

skin. Immune system imbalances, including overreacting to certain triggers like allergens, are common in people with atopic dermatitis.[14] The signs of atopic dermatitis can be brought on by this heightened immune response, which can result in chronic skin inflammation.

Researchers and physicians are concentrating on Treg cells in atopic dermatitis to better understand how these cells work and whether their activity might be increased. Finding strategies to increase Treg cells' suppressive activity is the aim in order to restore immunological balance and lessen the severity of atopic dermatitis symptoms. In other words, by encouraging the "peacekeeping" function of Treg cells, they hope to reduce the symptoms of atopic dermatitis by taming the heightened immune response.

Low levels of the reactive benzene metabolite 1, 4_BQ can significantly impair the suppressive function of Treg cells by reducing their potential to secrete the anti-inflammatory cytokine IL-10. This functional impairment most likely reduces the Treg-cell suppressive capacity, resulting in higher GrzB (Granzyme B) production by conventional T cells and a subsequent further loss in Treg-cell numbers presumably due to higher apoptosis rates induced by GrzB. Winter et al demonstrated an additional direct inhibitory effect of the benzene metabolite 1, 4-BQ on Treg-cell functional activity, providing further mechanistic insight into how benzene/tobacco smoke exposure can contribute to early childhood AD by reducing Treg-cell activity.[13]

This reduction in immunocompetent cells is likely a contributing factor to the increased risk of AD described in relation to benzene/environmental tobacco smoke.[13]

International studies on ETS and AD in children

Israel

Graif et al., investigated the association of smoking and exposure to ETS with prevalence of atopic eczema in a national sample of 13- to 14-year-olds in Israel. The research was conducted as part of the International Study on Childhood Allergies and Asthma. Smoking and atopic eczema were substantially more common in smokers than in nonsmokers, and there was a dose-response relationship between the two. A new finding is the dose-response relationship between active smoking and atopic eczema in teens.[15]

South Africa

Using the International investigation of Asthma and Allergies in Childhood questionnaire, Shirinde et al. conducted a cross-sectional investigation on environmental cigarette smoke and the risk of eczema symptoms among school children in South Africa. Eczema symptoms were positively correlated with ETS exposure at home and at school. The findings are consistent with the idea that ETS is crucial to understanding the onset of eczema.[16]

Lebanon

To assess the relationship between exposure to waterpipe passive smoking and asthma and allergy in Lebanese children, Waked et al. conducted an investigation. Mothers who smoked waterpipes were considerably and moderately more likely to develop allergy conditions such dermatitis, rhinitis, and possible asthma. On the contrary, none of the ailments were linked to the father's water pipe smoking. Parental cigarette smoking revealed some favorable effects: the mother's smoking only showed a positive link with possible asthma, whereas the father's smoking did not show an association with dermatitis or asthma identified by a doctor.[17]

Lifetime exposure

Smoking exposure in the past or present may have a cumulative effect on the development of adult-onset AD. The onset of adult-onset AD is linked to early-life exposure to ETS. To avoid adult-onset AD in themselves and their families, adults should be discouraged from smoking. Packs per year were substantially related with adult-onset AD, suggesting a lifetime cumulative risk in current smokers. Additionally, those with adult-onset AD who are not smokers reported much increased exposure to ETS.[18]

Atopic Dermatitis

Traditional cigarette smoke exposure

There were four systematic reviews that have elucidated well the association of smoking and asthma.[19-21] Multiple studies were done to explain the effects of tobacco smoke on the airway epithelium, smooth muscles, and gene expression, and its mechanism on asthma causation.

Inhaled smoke that is in direct contact with the airway epithelium increases the permeability of the epithelial lining.[22, 23] With the increase in permeability, there is an increase in allergic sensitization to inhaled allergens[24,25] and increase in inhaled steroid clearance in the airway.[26,27] Moreover, exposure to tobacco smoke worsens allergic airway responses to simultaneously inhaled aeroallergens.[28]

Smokers have decreased histone de-acetylase activity which is necessary for corticosteroids to fully inhibit cytokine production, and can lead to corticosteroid resistance.[26,27] Current smokers with asthma were more likely to have a non-eosinophilic type of airway inflammation, thus, more likely to be poorly responsive to corticosteroids.[27] Therefore smoking is associated with severe uncontrolled asthma that is poorly responsive to inhaled steroids.

Smoking also alters gene expression apparently through its influence on the messenger RNA and microRNA[29,30] as well as DNA methylation.[31,32] In another study between genes and active tobacco smoking, novel polymorphisms were identified in non-annotated intergenic regions on chromosomes 9 and 12, that showed suggestive evidence for interaction with active tobacco smoking in the onset of adult asthma.[31,32]

Chronic tobacco smoke exposure has been reported to have an effect on immune regulations33,34 particularly in the production of IL-13, a cytokine important in the pathophysiology of asthma, which increases airway respon-siveness.[25] In vitro data suggest that cigarette smoke also affects smooth muscle in the airway through the induction of neurotrophin and neurotrophin receptor, thereby increasing neurotrophin signaling, which may contribute to cigarette smoke-induced airway hyper responsiveness.[25]

In a systematic review by Jayes L et al., they reviewed studies from 1985 until 2013 and reported increased risk in developing asthma, poor asthma control, increased exacerbations in smokers and those exposed to tobacco smoke, both in the adult and pediatric populations.[19]

<u>*Adult asthmatic smokers*</u>

- Smokers were 1.61 to 1.81 times (61% to 81%) more likely to develop asthma than adults who had never smoked. Adults with asthma who were current or ex–smokers had a 1.71 times higher risk of subsequent asthma exacerbations than adults with asthma who had never smoked.
- A study on 80 pregnant asthmatic women showed that current or past smokers significantly increased the number of severe asthma exacerbations annually and poorer asthma control was seen on current smokers compared to non-smokers.

<u>*Adult passive smokers*</u>

- Passive smoking was defined as being in contact with second hand smoke from any source: domestic, occupational or other sources. Three studies showed that non-smokers exposed to passive smoke increased significantly the risk of being restricted in daily activities.

<u>*Pediatric population exposed to passive smoking*</u>

- In 140 studies of asthmatic children exposed to passive smoke in the household or by the mother, the risk of multiple hospital admissions for asthma per year more than doubled the risk. In addition, exposure to maternal smoking more than tripled the risk of multiple hospital admissions for asthma per year.
- Across age groups (less than 2 years old to 18 years of age), children exposed to household smoking 1.14 to 1.3 times (14-30%) more likely to develop asthma.
- For all sources of exposure to smoking (prenatal, postnatal, paternal and household), the effect of size for the increased risk of asthma was similar sensitivity analyses except in studies done in Europe where the magnitude of the risks of asthma from postnatal maternal smoking in 5-18 years old was larger (1.48 times) than all studies (1.20 times). In 5–18 years old exposed to household smoking, the risk of asthma was even larger 2.03 times than all studies (1.3 times).

E-cigarette smoke exposure

Vaping is associated with worsening of immune and respiratory functions. There is upregulation of aldehyde-detoxification and oxidative stress leading to increased airway obstruction, tissue damage, airway hyperactivity and remodeling.[35] There are decreased host defense genes and elevated pathologic mucin, neutrophil elastase, and matrix metalloproteinases MP 2 and 9.[35]

Physical appearance of bronchial airways during bronchoscopy reported gross physical damage described as friable and erythematous. Nicotine has been shown to activate macrophage, disrupt airway mucociliary clearance, disrupts mucus properties leading to persistent infection and inflammation of the lungs.

Cinnamaldehyde, a flavoring agent, impairs mitochon-drial respiration and glycolysis, reduces intracellular ATP level in human bronchial epithelial cells and suppresses ciliary beat frequency.[35] Furthermore, cinnamaldehyde, benzaldehydes, and vanillin impair neutrophil function leading to poor immune response.

Summary

In summary, exposure to tobacco smoke increases the risk of developing asthma in both adults and children. Smoking also increases symptom severity, exacerbation, restrictions in daily activities, multiple hospital admissions and non-response to steroid inhalers in asthmatics. This is true for all types of exposures, whether as current or previous smoker, or those exposed to second hand smoke, including prenatal, postnatal, paternal and household.

Likewise, electronic cigarette smoke or vaping, is associated with deterioration of immune and respiratory function leading to inflammation, increased airway obstruction, hyperactivity, remodeling and susceptibility to infection.

Allergic Rhinitis

Allergic Rhinitis or hay fever is an inflammatory condition of nasal passages which presents as sneezing, nasal pruritus, eye itchiness, runny nose, eye tearing, nasal congestion,

post nasal drip, as well as loss of smell and taste. It is one of the chronic conditions affecting the patient's quality of life greatly. Children with severe rhinitis are unable to breathe through their nose and do mouth breathing instead. Thereby, the lungs are more exposed to environmental allergens and irritants like tobacco smoke. Inflammation and congestion of nasal passages causes coughing and directly irritates the lungs.[35] In the one disease one airway concept, the nasal status mirrors the bronchial status. Thus, triggers like pollutants, smoke from traditional and electronic cigarettes, exacerbates allergic rhinitis and bronchial asthma, too.[35,36]

Active Smoking

Cigarette smoke exposure was associated with increased prevalence of rhinitis symptoms.[36] It alters the structure and function of central and peripheral airways, alveoli and capillaries. Pulmonary function abnormalities and respiratory symptoms are greatly increased among cigarette smokers and greatly aggravate condition of patients.[36] According to Zhou et al., higher exposure to smoke causes pathologic changes to sinonasal epithelial cells that serve to adversely reduce mucociliary clearance function.[37]

Smoke alters the locally expressed pattern receptors such as toll like receptors and innate immune effector proteins such as beta defensins and complement components in chronic rhinosinusitis (CRS) sinonasal epithelium.[38] It has immunosuppressive effects by suppressing monocyte-derived macrophage function as well as inhibiting inflammatory cytokines by suppressing toll like receptor (TLR) mediated pathways in human bronchial epithelial cells.[39]

Lee et al., reported that acrolein, a volatile component of tobacco smoke, inhibits the cytokine IL-8 and human B defensin(HBD-2) in sinonasal epithelial cell culture derived from patients with chronic rhinosiusitis.[39]

Yamin et al., collected primary nasal epithelial cells from middle turbinates of patients with chronic sinusitis. They exposed these nasal epithelial cells to cigarette smoke. They found exaggerated RANTES (regulated upon activation, normal T-cell expressed and secreted) production in response to cigarette smoke. When this condition is added with viral infection, inappropriate inflammation in chronic sinusitis can be seen.[40] The role of RANTES in epithelial innate immunity is not well known, but increased expression of this cytokine has been seen in patients diagnosed with asthma and nasal polyp.[41]

E-cigarettes

A Korean youth study among middle and senior high school students, involving 60,040 participants, was conducted using large survey data by the team of Dr Soo Jie Chung. Results revealed conventional cigarettes and heated tobacco products utilization was significantly associated with current asthma and allergic rhinitis.[42]

Rha showed in their 2022 nationwide cross-sectional study that E-cigarette use has been significantly associated with high prevalence of chronic rhinosinusitis in adult population in Korea. This study revealed that the consumption of E-cigs may directly increase the risk of upper airway disease.[43]

Passive Smoking

Passive smoking has harmful effects in the respiratory health of children such as higher rates of respiratory illness during first year of life, higher rates of middle ear effusion and higher rates of sudden infant deaths.[42] Children with asthma and/or rhinitis, upper respiratory allergy whose parents smoke have more severe symptoms and more frequent exacerbations.[44] Tobacco smoke is involved in uncontrolled asthma with or without allergic rhinitis, diminished response to anti-allergy drugs, nasal obstruction and deregulation of immune system.[45]

SMOKEFREE INTERVENTIONS IN THE PHILIPPINES

Republic Act 9211, otherwise known as the Tobacco Regulation Act of 2003 regulates smoking in public places, tobacco advertising as well as promotions and sponsorships and restricts minors to sell or buy any tobacco-related product.[46] This law also prohibits sale of cigarettes within 100 meters from schools or public playgrounds. It was further reinforced by the signing of Executive Order 26 in 2017, banning smoking from all public places.

The Tobacco regulation act also required tobacco companies to put text health warnings on their packages. This is not in line, however, with the guidelines of the WHO Framework Convention on Tobacco Control. Thus, in 2010, the Department of Health issued an administrative order requiring tobacco companies to put graphic health warnings on cigarette packages. This later led to the filing of the Graphic Health Warning Bill in 2014 which requires tobacco companies to print twelve graphic health warning templates on cigarette packages being sold in the country. It took a while for its' implementation due to interference from tobacco companies but it was finally enforced in 2016.

Republic Act 10351 or the Sin Tax Reform Law was also enacted in 2012 to reduce the purchase and use of tobacco and alcohol products. The Sin Tax Reform Law helps fund the Universal Health Care Program of the government and simplifies the excise tax system on tobacco and alcohol products. This law also helps to address public health

issues that are related to tobacco and alcohol product consumption with the aim of reducing consumption of these products in the long run.[47] Its' success paved the way for more policies like Republic Act 11346 of 2019 which increases the excise tax imposed on all tobacco-related products, including e-cigarettes.[48]

Laws and policies regulating e-cigarette use have also been enacted such as Republic Act 11900 or the Vaporized Nicotine and Non-Nicotine Products Regulation Act of 2022. This law regulates the production, purchase and use of vaping nicotine, non-nicotine products and novel tobacco products.[49] It is expected that by June 2024, these products will also show graphic and text health warnings as seen in other tobacco-related products like cigarettes.

All of these laws and policies have led to a decrease in the number of smokers in the country as shown in the results of the Global Adult Tobacco Survey in 2021. A comparison of results from 2009 to 2021 showed a 34.4% relative decrease in use of any tobacco-related product.[50] On the other hand, the percentage of adults who currently smoke cigarettes and tried to quit smoking in the past 12 months, increased from 55.5% in 2015 to 68% in 2021.[50] The average age of smoking initiation also increased from 17.3 years in 2009 to 19.5 years in 2021.[50] This shows that continued efforts in decreasing smokefree exposure through strict implementation of smokefree laws may decrease the burden of allergic diseases and other ill effects of smoking in the country.[48,51] Likewise, increased public awareness on effects of smoking and its impact on health and diseases may encourage the public to discontinue smoking.

RECOMMENDATIONS

We strongly recommend avoiding exposure to tobacco products in any form. Exposure to tobacco products can lead to development as well as exacerbation of allergic diseases.

We support the implementation of smokefree legis-lation and revision of the sin tax reform law.

We strive to educate our patients against the ill effects of tobacco products and advice those who are currently using tobacco products to quit as soon as possible, not only for themselves but for their whole family.

REFERENCES

1. Yang HJ. Impact of perinatal environmental tobacco smoke on the development of childhood allergic diseases. Korean J Pediatric. 2016;59(8):19-27. PMID: 27610180 PMCID: PMC5014911 DOI: 10.3345/kjp.2016.59.8.319
2. Saulyte J, Regueira C, Montes-Martínez A, Khudyakov P, Takkouche B. Active or passive exposure to tobacco smoking and allergic rhinitis, allergic dermatitis, and food allergy in adults and children: a systematic review and meta-analysis. PLoS Medicine. 2014;11(3):e1001611. PMID: 24618794 PMCID: PMC3949681 DOI: 10.1371/journal.pmed.1001611
3. World Health Organization. 2021 GATS Fact Sheet Philippines. 2021 GATS Fact Sheet Philippines. https://www.who.int/publications/m/item/2021-gats-fact-sheet-philippines
4. World Health Organization. 2019 GYTS Fact Sheet Philippines. 2019 GYTS Fact Sheet Philippines. https://www.who.int/publications/m/item/2019-gyts-fact-sheet-philippines
5. Repace J. Exposure to secondhand smoke. In: Ott WR, Steinemann AC, Wallace LA (eds). Exposure analysis, 1st ed, ch. 9. USA: CRC Press, Taylor & Francis Group; 2009. http://www.repace.com/pdf/EXPOSURE_TO_SECONDHAND_SMOKE.pdf
6. US Department of Health and Human Services. Let's make the next generation tobacco-free: your guide to the 50th anniversary surgeon general's report on smoking and health; 2014. https://www.hhs.gov/sites/default/files/consequences-smoking-consumer-guide.pdf
7. Mahabee-Gittens EM, Merianos AL, Matt G. Preliminary evidence that high levels of nicotine on children's hands may contribute to overall tobacco smoke exposure. Tob Control. 2018;27(2)217-9. PMID:28360145 PMCID: PMC5623162 DOI: 10.1136/tobaccocontrol-2016-053602
8. Liu AH, Babineau DC, Krouse RZ, et al. Pathways through which asthma risk factors contribute to asthma severity in inner – city children. J Allergy Clin Immunol. 2016;138(4):1042-50. PMID: 27720018 PMCID: PMC5381517 DOI: 10.1016/j.jaci.2016.06.060
9. Kresk N, Ankrum A, Cerdá M, Chen Q, Hasin D, Martins S, Olfson M, Keyes K, Nicotine Vaping and Co-occurring substance use among adolescents in the United States from 2017-2019, Substance use and misuse, 2023:58(9), 1075-79, DOI:10.1080/10826084.2023.2188462
10. Ahn K. The role of air pollutants in atopic dermatitis. J Allergy Clin Immunol. 2014;134(5):993-9. PMID: 25439225 DOI: 10.1016/j.jaci.2014.09.023
11. Kantor R, Silverberg JI. Environmental risk factors and their role in the management of atopic dermatitis. Expert Rev Clin Immunol. 2017;13(1):15-26. PMID: 27417220 PMCID: PMC5216178 DOI: 10.1080/1744666X.2016.1212660
12. Wang IJ, Chen SL, Lu TP, Chuang EY, Chen PC. Prenatal smoke exposure, DNA methylation, and childhood atopic dermatitis. Clin Exp Allergy. 2013;43(5):535-43. PMID: 23600544 DOI: 10.1111/cea.12108
13. Winter M, Thürmann L, Gu Z, et al. The benzene metabolite 1, 4-benzoquinone reduces regulatory T-cell function: a potential mechanism for tobacco smoke–associated atopic dermatitis. J Allergy Clin Immunol. 2017;140(2):603-5. PMID: 28274748 DOI: 10.1016/j.jaci.2017.01.034
14. Adkinson NF, Bochner BS, Burks AW, et al. Middleton's Allergy: Principles and Practice, 8th ed. Elsevier Inc; 2013.
15. Graif Y, German L, Ifrah A, Liyne I, Shohat T. Dose-response association between smoking and atopic eczema: results from a large cross-sectional study in adolescents. Dermatology, 2013;226(3):195-9. PMID:23711459 DOI:10.1159/000348333
16. Shirinde J, Wichmann J, Voyi K. Environmental tobacco smoke and the risk of eczema symptoms among school children in South Africa: a cross sectional study. BMJ Open. 2015; 5(8) e008234. PMID 26310401 PMCID: PMC4554914 DOI: 10.1136/bmjopen-2015-008234
17. Waked M, Salameh P. Maternal water pipe smoke exposure and the risk of asthma and allergic diseases in childhood: a post hoc analysis. Int J Occup Med Environ Health. 2015:28(1):147-56. PMID: 26159955 DOI: 10.13075/ijomeh.1896.00316
18. Lee CH, Chuang HY, Hong CH et al. Lifetime exposure to cigarette smoking and the development of adult-onset atopic dermatitis. Br J Dermatol. 2011;164(3):483-9. PMID:21054333 PMCID:PMC3062947 DOI:10.1111/j.1365-2133.2010.10116.x
19. Jayes L, Haslam PLG, Powell P, et al. SmokeHaz: systematic reviews and metaanalyses of the effects of smoking on respiratory health. Chest 2016; 50 (1): 164 – 79. PMID: 27102185 DOI: 10.1016/j.chest.2016.03.060
20. Genuneit J, Seibold A, Apfelbacher C, et al. The state of asthma epidemiology: an overview of systematic reviews and their quality. Clin Transl Allergy. 2017; 7:12. PMID:284000946 PMCID:PMC5387188 DOI: 10.1186/s13601-017-0146-y
21. Zhang L, He L, Gong J et al. Risk factors associated with Irreversible airway obstruction in asthma: a systematic review and meta-analysis. Biomed Res Int. 2016;9868704. MID:27119087 PMCID: PMC4828538 DOI: 10.1155/2016/9868704

22. Rusznac C, Mills PR, Devalia JL, Sapsford RF, Davies RJ, Lozewicz S. Effect of cigarette smoke on the permeability and IL – 1 beta and sICAM – 1 release from cultured human bronchial cells of never-smokers, smokers and patients with chronic obstructive pulmonary disease. Am J Respir Cell Mol Biol 2000;23(4):530-6. PMID:11017919 DOI: 10.1165/ajrcmb.23.4.3959
23. Rusznack C, Sapsford RJ, Devalia JL, et al. Cigarette smoke potentiates house dust mite allergen – induced increased in the permeablity of human bronchial epithelial cells in vitro. Am J Respir Cell Mol Biol 1999;20(6):1238-50. PMID:10340943 DOI:10.1165/arjcmb.20.6.3226
24. Lanchacker EA, Tournoy KG, Hammad et al. Short cigarette smoke exposures facilitates sensitization and asthma development in mice. Eur Respir J 2013;41(5):1189-99. PMID:22903968 DOI:10.1183/09031936.00096612
25. Trimble NJ, Botelho FM, Bauer Cm, Fattouh R, Stampfli MR. Adjuvant and anti-inflammatory properties of cigarette smoke in murine allergic airway inflammation. Am J Respir Cell Mol Bio.I 2009;40(1):38-46. PMID:18635815 DOI:10.1165/rcmb.2008.0107OC
26. Stapleton M, Howard – Thompson A, George C, Hoover RM, Self TH. Smoking and asthma. Am Board Fam Med, 2011; 24(3):313-22. PMID: 2155404 DOI: 10.3122/jabfm.2011.03.100180
27. Esteban-Gorgojo I, Antolin-Amergio D, Dominguez-Ortega J, Quirce S. Non- eosinophilic asthma: current perspectives. J Asthma Allergy. 2018;11:267-81. Dario 2 Javier, PMID:30464537 PMCID: PMC6211679 DOI: 10.2147/JAA.S153097
28. Diaz-Sanchez D, Rumold R, Gong H Jr. Challenge with environmental tobacco smoke exacerbates allergic airway disease in human beings. J Allergy Clin Immunol 2006; 118(2):441-6. PMID:16890770 DOI:10.1016/j.jaci.2006.04.047
29. Schembri F, Sridhar S, Perdomo C, et al. MicroRNAs as modulators of smoking-induced gene expression changes in human airway epithelium. Proc Natl Acad Sci USA. 2009;106(7):2319-24. PMID: 19168627 PMCID:PMC2650144 DOI: 10.1073/pnas.0806383106
30. Spira A, Bane J, Shah V, et al. Effects of cigarette smoke on human airway epithelial cell transcriptome. Proc Natl Acad Sci USA 2004;101:10143-8. PMID: 15210990 PMCID: PMC454179 DOI: 10.1073/pnas.0401422101
31. Joubert BR, Felix JF, Yousefi P, et al. DNA methylation in newborns and maternal smoking in pregnancy: genome-wide consortium meta-analysis. Am J Hum Genet. 2016;98(4):680-96. PMID:27040690 PMCID: PMC4833289 DOI: 10.1016/j.ajhg.2016.02.019
32. Vonk JM, Scholtens S, Postman DS, et al. Adult onset asthma and interaction between genes and active tobacco smoking: the Gabriel consortium: Plos One. 2017; 12(3):eo0172716. PMID: 28253294 PMCID: PMC5333809 DOI:10.1371/journal.pone.0172716
33. Hallstrand TS, Hackett TL, Altemeier WA, Matute-Bello G, Hansbro PM, Knight DA. Airway epithelial regulation of pulmonary immune homeostasis and inflammation. Clin Immunol. 2014;151(1):1-15. PMID:24503171 DOI:10.1016/j.clim.2013.12.003
34. Coogan PF, Castro-Webb N, Yu J, O'Connor G, Palmer J, Rosenberg L. Active and passive smoking and the incidence of asthma in the black women's health study. Am J Respir Crit Care Med. 2015;191(2):168-76. PMID:25387276 PMCID: PMC4347433 DOI: 10.1164/rccm.201406-1108OC
35. Hernandez ML, Burbank AJ, Alexis NE, et al. Electronic Cigarettes and Their Impact on Allergic Respiratory Diseases: a Work Group Report of the AAAAI Environmental Exposures and Respiratory Health Committee J Allergy and Clinical Immunology Pract. 2021;9(3):1142-51. PMID:33547027 PMCID: PMC8232370 DOI: 10.1016/j.jaip.2020.12.065
36. Shargorodsky J, Garcia-Esquinas E, Galán I, Navas-Acien A, Lin SY. Allergic sensitization, rhinitis and tobacco smoke exposure in US adults. PLoS One. 2015:10(7):eo0131957. PMID:26172447 PMCID: PMC4501790 DOI:10.1371/journal.pone.0131957
37. Zhou H, Wang X, Brighton L, Hazucha M, Jaspers I, Carson JL. Increased nasal epithelial ciliary beat frequency associated with lifestyle tobacco smoke exposure. Inhal Toxico. 2009;21(10):875-81. PMID: 19555226 PMCID: PMC2721908 DOI: 10.1080/08958370802555898
38. Vandermeer J, Sha Q, Lane AP, Schleimer RP. Innate Immunity of sinonasal cavity: expression of messenger RNA for complement cascade components and toll like receptor. Arch Otolaryngolo Head and Neck Surg. 2004;130(12):1374-80. PMID: 15611395 DOI: 10.1001/archotol.130.12.1374
39. Lee WK, Ramanathan Jr M, Spannhake EW, Lane AP. The cigarette smoke component acrolein inhibits expression of the innate immune components IL8 and human beta-defensin 2 by sinonasal epithelial cells. Am J Rhino. 2007;21(6):658-63, PMID: 18201443 DOI: 10.2500/ajr.2007.21.3094
40. Yamin M, Holbrook EH, Gray ST, et al. Cigarette smoke combined with Toll like receptor 3 signaling triggers exaggerated epithelial regulated upon activation, normal T cell expressed and secreted / CCL5 expression in chronic rhinosinusitis. J Allergy Clin Immunol. 2008;122(6):1145-53.e3. PMID: 18986692 DOI: 10.1016/j.jaci.2008.09.033
41. Beck LA, Stellato C, Beall LD, et al. Detection of chemokine RANTES and endothelial adhesion molecules in nasal polyps. J Allergy Clin Immunol. 1996;98(4):766-80. PMID: 8876553 DOI: 10.1016/s0091-6749(96)70126-4
42. Chung, SJ, Kim BK, Oh JH, et al. Novel tobacco products including electronic cigarette and heated tobacco products increase risk of allergic rhinitis and asthma in adolescents: analysis of Korean youth survey. Allergy. 2020;75(7):1640-8. PMID: 32003899 DOI: 10.1111/all.14212
43. Rha, MS, Cho HJ, Yoon JY, Kim CH. Association between the use of electronic cigarettes and the prevalence of chronic rhinosinusitis and allergic rhinitis: a nationwide cross-sectional study. Rhinology 2022;60(1):20-8. PMID: 34941973 DOI:10.4193/Rhin21.287
44. Koop CE. The health consequences of involuntary smoking: a report of the surgeon general. DHHS publication. no. (CDC) 87-8398. 1986. https://stacks.cdc.gov/view/cdc/20799/cdc_20799_DSI.pdf
45. American College of Allergy, Asthma and Immunology. Tobacco smoke has harmful impact on asthma, rhinitis and immunity. News Wise. October 29, 2009. https://www.newswise.com/articles/tobacco-smoke-has-harmful-impact-on-asthma-rhinitis-and-immunity
46. Republic Act 9211. June 23, 2003. http://www.officialgazette.gov.ph/2003/06/23/republic-act-n0-9211
47. Republic Act No. 10351. Implementing Guidelines: Revenue Regulation No. 17-2012. December 19, 2012. http://www.officialgazette.gov.ph/2012/12/19/republic-act-no-10351/. http://www.officialgazette.gov.ph/downloads/2012/12dec/20121219-RA-10351-BSA.pdf
48. Rodrigo P. 10 years since the signing of the sin tax law. Business World Online. January 15,2023. https://www.bworldonline.com/opinion/2023/01/15/498372/10-years-since-the-signing-of-the-sin-tax-law/
49. Talavera, C. DTI urges e-commerce platform to follow vape law. Philstar.com. March 7, 2023. https://www.philstar.com/business/2023/03/07/2249751/dti-urges-e-commerce-platform-follow-vape-law
50. World Health Organization. 2021 GATS Comparison Fact Sheet Philippines (2021, 2015, 2009). 2021. https://www.who.int/publications/m/item/2021-gats-comparison-fact-sheet-philippines-(2021-2015-2009)
51. Faber T, Been JV, Reiss IK, Mackenbacj JP, Sheikh A. Smoke-free legislation and child health. NPJ Prim Care Respir Med. 2016;26:16067. PMID: 27853176 PMCID: PMC5113157 DOI: 10.1038/npjpcrm.2016.67