Editorial

Global state of tobacco use: summary from the American Thoracic Society International Conference 2016

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Introduction

Some have estimated that in 2012 global smoking of any tobacco product in those ≥15 years of age was 13.8% in females *vs.* 17% in males (1). In children aged 13–15 years old between 2007–2014, 8.3% of females and 18.2% males smoked a tobacco product (1). Forty percent of children worldwide were exposed to second-hand smoke. Children from low-middle income families across Africa, Asia, Europe, the Mediterranean and Western Pacific regions had second-hand smoke exposures ranging from 12–68%, while high income countries reported 39% of children exposed (1). Many pulmonary inflammatory diseases are known to be caused by firsthand and secondhand cigarette smoke exposure (2). Also, the risk of lung cancer and sino-pulmonary infections directly associated with cigarette smoke exposure produce an on-going struggle in morbidity, mortality and increasing healthcare costs. Research examining the impact of tobacco related products on health risks and optimization of prevention and cessation of cigarette smoking is of critical importance.

Prevention of beginning tobacco use

As efforts to improve success with smoking cessation are made, prevention of initiation of tobacco-use is also necessary. Better understanding of various socio-economic factors that shape an individual's access to education, safe community and health care would provide more effective implementation of medical and behavioral change. Weir *et al.* showed a cohort study of those in an urban setting to examine better the prevalence of adverse health behaviors in addition to cigarette use and access to neighborhood walkability (3). Although it was based on a single city and variations in demographics will exist in different cities, the study provided insight on an urban, predominantly African American (79%) population with a mean age of 53.6 years with 95% of subjects who were active daily cigarette smokers (3). Almost half of the study population had a diagnosis of chronic obstructive pulmonary disease (COPD). Furthermore, there was a high prevalence of poor sleep, obesity, smoking and lack of exercise with predominantly limited access to community safety, public green space, and education (3).

Preventative counseling in tobacco use poses additional challenges in the younger generation as motivational incentives often times differ from those of adults. As communication via internet-based media increases (i.e., Facebook, Instagram and Twitter), utilizing social media for outreach to younger populations may be more effective than traditional counseling methods. Jayasinghe *et al.* reported that clearly stated and research-based campaign messages with intense broadcasting showed statistically significant reductions in smoking (4). However, further trials are recommended prior to sole use of social media in lieu of in-person education and counseling.

Tobacco cessation

Of the approximately 45 million Americans who smoke, the majority have reported a desire to quit. Smoking cessation requires a multi-dimensional approach that involves the individual, physician, counseling services and maintenance programs to ensure the best success in abstinence. Long-term abstinence rates may increase up to 30% in individuals with physician advice or encouragement

compared to 7% in those trying to quit alone (5). Physician perceptions and understanding the effectiveness of different smoking cessation agents also influence therapy of choice. Studies evaluating training physicians by Sedhom *et al.* observed that younger physicians who had formal medical education and training on tobacco were more likely than others to identify intervention effectiveness accurately. There were more positive perceptions of interventions in general in those with prior tobacco cessation training (6). However, trained physicians only performed slightly better than controls, indicating that areas of research on the development of comprehensive smoking cessation programs that incorporate education for medical providers must still be pursued (6).

Addressing smoking cessation while patients are hospitalized may also impact patient's smoking behavior significantly. Surprisingly, cessation rates at 6 months comparing inpatient *vs.* outpatient interventions show higher rates associated with inpatient (6). Addressing smoking cessation during inpatient hospitalizations especially when no other clinical or pharmacologic limitations are identified should be considered. In-person smoking cessation counseling by trained pharmacists, when combined with nicotine replacement therapy, led to improved abstinence (7). This finding demonstrates that education of smokers provided by a variety of trained healthcare providers may increase cessation success.

Infections and smoking exposure

Pulmonary tuberculosis (TB) cases in smokers have lower responses to TB treatment compared to non-smokers. In a cohort study by Reddy *et al.*, of those with smear-positive TB cases, those with positive 2-month post-treatment initiation sputum smears (thus indicating resistance to treatment), 64% were current or former smokers while 25% lived in households exposed to wood-fuel (8). The findings from this study suggest that subjects with both wood-fuel and cigarette smoke exposures were more likely to have a positive 2-month sputum smear although larger better powered studies are still needed (8). Prior studies have shown increased lung inflammation in cigarette smoke exposed mice infected with *Haemophilus influenzae* (9). Dhillon *et al.* showed that neonatal mice exposed to environmental tobacco smoke prior to influenza viral infection had increased lung inflammation compared to adult mice (10). These findings suggest that cigarette smoke alters the inflammatory state of lung cells more-so in juveniles, promoting inflammation and causing more collateral damage in the setting of pulmonary infections.

Cigarette smoke exposure in mice with chronic bronchitis led to changes in the microbiome (*Firmicutes*, *Proteobacteria* and *Bacteriodetes* species) that have been identified in COPD patients and thus may contribute to exacerbations or serve a role as biomarkers (11). Cigarette smoke was found to impact the lower airway DNA virome by decreasing bacteriophage diversity (12). There are limited data studying the physiologic and clinical effects of an altered virome, thus more research needs to be done in this area.

E-cigarettes—what is known?

Newer electronic tobacco-related products are emerging on the market with much need for research in short- and long-term health effects in individuals as well as in the community. E-cigarette use among teenagers tripled between 2013—2014 from 1.1% to 3.9% (13). The use of e-cigarettes is controversial as it is marketed as a "safe alternative" to smoking despite lack of studies evaluating their health effects, safety and efficacy. A study by Rankin *et al.* suggests that e-cigarette vapor and conventional cigarette extracts both decrease *in vitro* lung epithelial cell viability acutely, but e-cigarettes have less of an effect compared to conventional cigarettes (14). Lerner *et al.* found an increase in mitochondrial oxidative stress, elevated IL-6 and IL-8 release in human lung epithelial cells exposed to e-cigarette flavoring aerosols (15). *In vivo* and *in vitro* e-cigarette vapor exposures demonstrated airway epithelial changes comparable to tobacco cigarettes, with similar oxidative responses and down regulation of genes involved in the mucociliary complex (16). These data correspond well with another study in which human bronchial epithelial cells exposed to e-cigarettes had diminished mucociliary clearance, to a degree similar to tobacco smoke (17). In a murine model study by Laube *et al.*, chronic exposure (3 weeks) with combined nicotine and propylene glycol showed decreased mucociliary clearance compared to acute exposure (A1198) (18). Furthermore, Reidel *et al.* evaluated human sputum samples and found that e-cigarette users had more differentially expressed proteins (cell lysis and pulmonary innate immune response) compared to cigarette or hookah use (19). E-cigarette vapor liquid alone or in combination with nicotine decreased phagocytosis by bronchial macrophages and increased IL-1β levels while multiple components (vapor solution, propylene glycol, and vegetable glycerin) increased epithelial cell apoptosis (20).

E-cigarette liquids contain not only nicotine but also flavorings dissolved in propylene glycol and/or vegetable glycerin that can cause respiratory irritation. In a study by Jordt *et al.*, commercially available cinnamon-flavored e-liquid was aerosolized and tested in mice. They found that the cinnamon flavoring led to respiratory irritation *in vivo* and activation of both mouse and human TRPA1 sensory irritant receptors (21). In addition, propylene glycol and vegetable glycerin attenuated responses of TRPA1 to irritants, raising concern for blunted responses to other e-cigarette contents, namely nicotine, flavors and contaminants (21).

Conclusions

Smoking, whether firsthand or secondhand, has posed health risks for both youth and adults. The use of "alternative" modes of cigarette smoking such as e-cigarettes are on the rise despite limited data on safety profile and long term health effects. Efforts in studying comprehensive, more effective methods of smoking prevention and cessation that address both socioeconomic influences and evidence based use of cessation agents are needed. Varying prevalence of tobacco smoking throughout different parts of the world requires evaluation of social perceptions, role of media and education of community on the known harmful effects of cigarette smoke exposure and of less known products.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

References

1. World Health Organization. Global Health Observatory Data Repository, 2016, Web 2 June 2016. Available online: http://www.who.int/en/
2. Crotty Alexander LE, Shin S, Hwang JH. Inflammatory Diseases of the Lung Induced by Conventional Cigarette Smoke: A Review. Chest 2015;148:1307-22.
3. Weir M, Cuesta H, Kolman D, et al. Environmental Influence On Tobacco Use In Pulmonary Patients. Am J Respir Crit Care Med 2016;193:A2036.
4. Jayasinghe H, Carson KV, Van Agteren J, et al. Mass media interventions for preventing smoking in young people: a Cochrane systematic analysis. Am J Respir Crit Care Med 2016;193:A2030.
5. Sedhom R, Sarkar A, Parikh S, et al. Do physicians address smoking status in patients admitted to the hospital? Am J Respir Crit Care Med 2016;193:A6495.
6. Sedhom R, Parikh S, Sarkar A, et al. Internal Medicine Resident Receptions and Knowledge Regarding Smoking Cessation. Am J Respir Crit Care Med 2016;193:A6494.
7. Carson KV, To-A-Nan R, Roberts M, et al. Community Pharmacy Personnel Interventions for Smoking Cessation: A Cochrane systematic review and meta-analysis. Am J Respir Crit Care Med 2016;193:A6497.
8. Reddy D, Sahu S, Roy G, et al. Association between Biomass Fuel, Tobacco Use and Two-Month Sputum Smear conversion among pulmonary tuberculosis cases in India. Am J Respir Crit Care Med 2016;193:A7666.
9. Gaschler GJ, Skrtic M, Zavitz CC, et al. Bacteria challenge in smoke-exposed mice exacerbates inflammation and skews the inflammatory profile. Am J Respir Crit Care Med 2009;179:666-75.
10. Dhillon JK, Wang L, Kaur M, et al. Age related modulation of pulmonary and systemic innate immunity against influenza A/PR/8/34 by environmental tobacco smoke in mice. Am J Respir Crit Care Med 2016;193:A1191.
11. Engle M, Zorn B, Jania C, et al. Changes in the lung microbiome induced by exposure to tobacco smoke in healthy mice and mice with chronically inflamed airways. Am J Respir Crit Care Med 2016;193:A1072.
12. Keller BC, Zhao G, Wu BG, et al. The effects of smoking on the lower airway DNA virome. Am J Respir Crit Care Med 2016;193:A1056.
13. Centers for Disease Control and Prevention, 16 April 2015, Web 1 June 2016. Available online: http://www.cdc.gov/media/releases/2015/p0416-e-cigarette-use.html
14. Rankin GD, Wingfors H, Ekstrand-Hammarstrom B, et al. Toxicological effects of electronic cigarette extracts on lung epithelial cell lines. Am J Respir Crit Care Med 2016;193:A1197.
15. Lerner CA, Rutagarama P, Sundar IK, et al. E-cigarette

flavoring aerosols trigger mitochondrial stress associated with mitophagy in lung cells. Am J Respir Crit Care Med 2016;193:A1193.
16. Nitzberg M, Corbett S, Moses E, et al. Molecular impact of electronic cigarette exposure on bronchial airway epithelium. Am J Respir Crit Care Med 2016;193:A1192.
17. Grosche A, Baumlin-Schmid N, Krick S, et al. Effects of nicotine-containing E-cigarette vapor on mucociliary clearance in NHBE cells. Am J Respir Crit Care Med 2016;193:A2032.
18. Laube B, Bang C, Chen G, et al. Acute and chronic effects of second-hand exposure to nicotine and propylene glycol from an E-cigarette on mucociliary clearance in a murine model. Am J Respir Crit Care Med 2016;193:A1198.
19. Reidel B, Radicioni G, Ford AA, et al. The impact of new and emerging tobacco products on the airway mucosal barrier. Am J Respir Crit Care Med 2016;193:A1195.
20. Ween M, Hodge G, Reynolds P, et al. The new Kid on the block: E-cigarettes can cause damage to airway cells and cause airway macrophage dysfunction. Am J Respir Crit Care Med 2016;193:A1194.
21. Jordt SE, Jabba S, Ghoreshi K et al. Propylene Glycol and Glycerin E cigarette liquids modulate murine respiratory irritation response and human sensory irritant receptor function. Am J Respir Crit Care Med 2016;193:A7767.

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