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.
In vivo
in vitro.

et al.
chronic exposure.
Firmicutes
Proteobacteria
Bacteriodetes
strain
have shown increased lung inflammation in cigarette smoke
better powered studies are still needed (8). Prior studies
wood-fuel and cigarette smoke exposures were more likely than
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


