

# Testimony by Otolaryngologists in Defense of Tobacco Companies 2009–2014

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**Objectives/Hypothesis:** To examine expert testimony offered by otolaryngologists in defense of the tobacco industry and to assess whether opinions rendered were congruent with evidence in the scientific literature.

**Methods:** Data sources include publically available expert witness depositions and trial testimony of board-certified otolaryngologists employed by the tobacco industry in defense of lawsuits brought by smokers suffering from head and neck cancer. The cases, adjudicated in Florida between 2009 and 2014, focused on whether smoking caused the plaintiff's cancer.

**Results:** The study includes nine legal cases of upper aerodigestive tract cancer involving six otolaryngologists serving as expert witnesses for the tobacco industry. Cancer sites included larynx (5), esophagus (2), mouth (1), and lung (1). Five of the six otolaryngologists consistently, over multiple cases, offered opinions that smoking did not cause the plaintiff's cancer. By highlighting an exhaustive list of potential risk factors, such as human papillomavirus (HPV), alcohol, asbestos, diesel fumes, salted fish, mouthwash, and even urban living, they created doubt in the minds of the jurors as to the role of smoking in the plaintiff's cancer. Evidence shows that this testimony, which was remarkably similar across cases, was part of a defense strategy shaped by tobacco's law firms.

**Conclusions:** A small group of otolaryngologists regularly serve as experts on behalf of the tobacco industry. Examination of their opinions in relation to the scientific literature reveals a systematic bias in interpreting the data relating to the role played by smoking in head and neck cancer causation.

**Key Words:** Tobacco, expert testimony, ethics.

**Level of Evidence:** N/A.

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## INTRODUCTION

For decades, tobacco companies were nearly invulnerable from consumer litigation in the United States. It took nearly four decades of litigation before the first legal victory in 1996.<sup>1</sup> Tobacco companies defended themselves by enlisting experts to testify that smoking was not related to disease. The 1962 case of *Ross v Philip Morris* was typical in that several leading otolaryngologists testified smoking was not a cause of head and neck cancer.<sup>2,3</sup> More recently, the tobacco industry defended against liability by arguing that consumers were adequately warned by package labels, and that their choice to smoke made any adverse consequence a matter of personal responsibility. As part of the Master Settlement Agreement of 1998, decades of internal

tobacco industry documents were released for public examination.<sup>4</sup> These documents revealed that the industry concealed information concerning the health effects of smoking and the addictiveness of nicotine. This material made it clear that the industry's knowledge of smoking's adverse effects differed widely from their public positions. It also revealed other forms of misconduct, such as pervasive youth-marketing strategies. This material provided new avenues for litigation.

A watershed 1999 Florida class-action suit (*Engle v Liggett*) yielded an unprecedentedly large award of \$145 billion.<sup>5</sup> Although the monetary award was reversed on appeal, this litigation led to a Florida Supreme Court decision in 2006 that upheld the Engle jury findings of widespread wrongdoing on the part of the tobacco industry. This enabled individual cases to proceed in Florida without need to relitigate settled issues under the dictum of *res judicata*: matters already judged. Thus, the addictiveness of nicotine, the dangers of tobacco, and the track record of industry deception and misconduct are considered factual in subsequent trials. The legacy of the Engle decision has been a series of some 8,000 individual Engle progeny cases. Because these cases have primarily focused on whether tobacco caused the plaintiff's disease, testimony by medical experts has been central to their adjudication.

As of early 2015, Florida courts have rendered approximately 120 decisions in Engle progeny cases, with some 70% of the awards favoring the plaintiff, including a \$23 billion in punitive damages in July 2014.<sup>6,7</sup> A fraction of these cases involved head and neck cancer, for which

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TABLE I.  
Recent Florida Cases in Which an Otolaryngologist Defended the Tobacco Industry.

2009	<i>Hess v R.J. Reynolds, Philip Morris</i>	Lung cancer
2009	<i>Brown v R.J. Reynolds</i>	Esophageal cancer
2010	<i>Hetzner v R.J. Reynolds et al.</i>	Oral cancer
2010–2012	<i>Mack v R.J. Reynolds</i>	Laryngeal cancer
2011	<i>Kirkland v R.J. Reynolds</i>	Laryngeal cancer
2011	<i>Syzmanski v R.J. Reynolds et al.</i>	Laryngeal cancer
2011	<i>Reese v R.J. Reynolds et al.</i>	Laryngeal cancer
2011	<i>Blitch v R.J. Reynolds</i>	Esophageal cancer
2014	<i>Cooper v R.J. Reynolds et al.</i>	Laryngeal cancer

board-certified otolaryngologists served as expert witnesses on behalf of tobacco companies, offering opinions that the plaintiff's tumors was caused by factors other than tobacco. Our purpose is to examine this expert testimony to assess whether opinions rendered were congruent with evidence in the published literature.

## STUDY DESIGN

Data sources included expert witness depositions and trial testimony of otolaryngologists employed by the tobacco industry via its law firms in defense of lawsuits by smokers. Cases involving head and neck cancer were retrieved from as early as the late 1940s, but the emphasis of the study is on Florida cases between 2009 and 2014. The legal documents were retrieved via online databases including the Tobacco Deposition and Trial Testimony Archive collection via the University of California at San Francisco Legacy database ([legacy.library.ecsf.edu](http://legacy.library.ecsf.edu)) and tobacco documents online ([tobaccodocuments.org/datta](http://tobaccodocuments.org/datta)).<sup>8</sup> These depositions and trial testimony are in the public domain and readily available online. Links to the documents cited in the text are provided to allow the interested reader to view the entire original document.

News reports relating to recent Florida tobacco cases were retrieved via Google and Google News using search terms such as tobacco litigation, Florida tobacco

cases, and Engle progeny cases—as well as specialty terms such as larynx, pharynx, oral, throat, head & neck, otolaryngology, and otolaryngologist. Case selection criteria included: 1) litigation in Florida against a tobacco company alleging smoking-related malignancy of the upper aerodigestive tract; 2) board-certified otolaryngologist providing compensated testimony on behalf of one of the three major US tobacco companies (R.J. Reynolds, Philip Morris, and/or Lorillard); and 3) trial date between 2009 to 2014.

Published literature from the medical, legal, and tobacco control literature was utilized to evaluate the scientific basis for the expressed expert opinions. Because the literature on cancer causation is voluminous, preference was given to recent meta-analysis and comprehensive reviews. Laryngeal cancer was the leading type among these cases, so literature on its causation was a special emphasis.

## RESULTS

### *Otolaryngologists Defending the Tobacco Industry*

The study included nine cases of upper aerodigestive tract cancer involving six otolaryngologists serving as expert witnesses for the tobacco industry between 2009 and 2014. The cases were laryngeal cancer (5), esophageal cancer (2), oral cancer (1), and lung cancer (1) (Table I). Two physicians were senior (certified by the American Board of Otolaryngology in the 1970s) and four physicians were midcareer (certified in the 1980 to 1990s) (Table II). Five of the six physicians are in private practice, whereas one is a retired full-time academician. Two of the six physicians describe themselves as subspecialist head and neck oncological surgeons. None had completed a formal head and neck surgery fellowship, although one physician is a former fellowship director of long standing. Below is a sample of testimony by each witness, with representative quotes gleaned from thousands of pages of deposition and trial transcripts.

### *Witness 1*

Witness 1 is a recently retired, senior professor (boarded 1974) at a major university who is one of the

TABLE II.  
Otolaryngologists Serving as Expert Witness on Behalf of the Tobacco Industry

Witness	ABO*	Practice Setting	Faculty Appointment	Head and Neck Specialty <sup>†</sup>	Tobacco Testimony <sup>‡</sup>
1	1974	University	Full Time (retired)	Yes	10–15
2	1974	Private practice	Clinical	No	20+
3	1997	Private practice	None	No	7
4	1995	Private practice	None	No	3–4
5	1987	Private practice	Clinical	Yes	> 4
6	1992	Private practice	Former full time	No	6

\*Year of certification by the American Board of Otolaryngology (ABO).

<sup>†</sup>Describes special interest in aerodigestive tract cancer. None completed head and neck fellowships.

<sup>‡</sup>Includes trial testimony, deposition, and/or request to review legal case)

12 But other confounding things that he had that  
 13 were operational that are known to produce larynx cancer  
 14 are urban living, exposure to gasoline fumes, possible  
 15 exposure to diesel fumes, machinery fluid, polycyclic  
 16 aromatic hydrocarbons, asbestos, and reflux. And all of  
 17 those are known etiologic agents in the production of  
 18 larynx cancer.  
 19 And those in toto combined with hereditary  
 20 influences are much more important in producing his  
 21 cancer, than was his smoking history.

Fig. 1. Opinion of witness 1 in 2010 case *Mack v R.J. Reynolds*.<sup>10</sup>

nations' leading head and neck surgeons. In testimony, witness 1 disclosed that he had served as expert witness some 125 times over many years and started working for tobacco circa 2007. In a 2014 trial, the witness described involvement in some 10 to 15 tobacco industry cases.<sup>9</sup> All of witness 1's cases included in this article were in Florida for the law firm Shook, Hardy & Bacon on behalf of either R.J. Reynolds or Philip Morris. Testimony reveals that the lawyers provided witness 1 with scientific literature to help guide opinion formation.

In *Mack v R.J. Reynolds* (2010), the plaintiff started smoking at age 16, had a 40- to 80-pack history, and developed laryngeal cancer and chronic obstructive pulmonary disease.<sup>10,11</sup> He had quit smoking some 10 to 15 years prior to diagnosis. In witness 1's deposition, he opined, "In this case he has an etiological soup" and provided an extensive list of potential causes (Table III) (Fig. 1). He went on to say: "Doctors are lazy . . . we don't take great toxicological histories." When pressed by plaintiff's counsel regarding some of the more exotic causes on the list, he vehemently defended their inclusion. For example, regarding gasoline, he replied: "I can tell you that there are many, many, many, many articles talking about gasoline and larynx cancer. It is not just one. Many, many, many." Witness 1 goes on to say that it is "common knowledge." The witness went on to say, "I do not know what caused his larynx cancer. My opinion is that Mr. Mack, indeed, had squamous cell carcinoma of the larynx that was most certainly not due to smoking." He based this opinion by stating that: "After you have quit for 10 years . . . risk of cancer of the larynx approaches never smokers." He went on to say, "And I am not entirely certain what it was due to, but he had a number of occupational exposures to known carcinogens that are known to produce larynx cancer."

After citing that the plaintiff's mother and sisters had breast and/or uterine cancer, whereas two brothers had prostate or bladder cancer, he concluded that, "The predominant influencing factor for this man was hereditary." The jury awarded \$2,900,000 compensatory damages, which was successfully appealed and remanded for retrial.

In *Hetzner v R.J. Reynolds et al.* (2010), the plaintiff smoked 30 plus pack years but quit as many as 20 years (duration disputed) before developing oral cancer.<sup>12</sup> The tumor itself was negative for HPV, but the mandibular margin was positive for HPV 52. Witness 1

concluded, "That this lady's cancer was, in all medical certainty, not related to her tobacco consumption." The opinion was based upon the HPV finding and the viewpoint that "The preponderance of evidence says that the relation of tobacco and oral cavity cancer disappears after ten years cessation." The witness went on to opine regarding head and neck cancer: "40% of our patients never smoked or drank" and "if you ask my opinion about the causation of cancer almost all of it is gene produced."

In the deposition, witness 1 described mouthwash users being at risk: "You should not use alcohol-based mouth wash that is greater than 14%." Listerine was cited as an example. The Hetzner case resolved as a summary judgment for the defense

In *Cooper v R.J. Reynolds et al.* (2014), the plaintiff smoked 25 to 45 pack years, beginning at age 12, before developing supraglottic laryngeal cancer.<sup>9,13,14</sup> Witness 1 opined that keratinization of the larynx was not caused by exposure to tobacco smoke. When asked the question, "the irritation caused by exposure to carcinogens in cigarettes can cause development of normal cells to dysplastic cells to cancerous cells, correct?" the witness replied, "Well, that mechanism has not been worked out and is not uniformly agreed upon. So the answer to your question is no." In terms of causation, the witness emphasized concomitant marijuana and alcohol use and possible asbestos exposure in a cotton mill. Because she worked as a bookkeeper in an automotive repair facility, the witness cited diesel fume exposure as a risk factor. In testimony, the witness opined that neither Cumming's textbook on otolaryngology-head and neck surgery textbook nor the Surgeon General's reports on tobacco are authoritative sources. The case was dismissed on technical grounds.

TABLE III.  
 Nontobacco Laryngeal Cancer Risks Cited by Otolaryngologists  
 Defending Tobacco Companies.

Alcohol
Anemia
Asbestos
Chromium
Cleaning solvents
Gasoline and diesel fumes
Gastroesophageal reflux disease
Genetic predisposition
Human papilloma virus
Immunosuppression
Machinery fluid
Mouthwash
Nickel
Polycyclic aromatic hydrocarbons
Radiation
Salted fish and meat
Smoking
Urban living

## Witness 2

Witness 2 is a private practice otolaryngologist, boarded in 1974, with a clinical appointment at a local medical school. This witness testified for tobacco companies for some 20 years in numerous cases, including the original 1999 Engle trial. In the 1994 *Allgood v R.J. Reynolds* case, witness 2 testified that smoking did not cause laryngeal cancer.<sup>15</sup> In 2011, responding to a plaintiff attorney's during a trial, witness 2 admitted that during his long history to testifying on behalf of the tobacco industry: "I have never given a sworn statement, deposition, live or otherwise, in court that in those cases where I have been named an expert that I have been able to identify smoking as the cause of disease."<sup>16</sup>

In the 2009 case *Brown v R.J. Reynolds*, the patient with 50 pack years of smoking died of a midesophageal cancer.<sup>16</sup> When asked about qualifications, the witness stated that, as an otolaryngologist, he saw 20 cases of esophageal cancer per year, and over his 40-year career claimed to have cared for 800 to 1,000 cases. Of note, in deposition, the witness described that his practice was 50% to 60% pediatric. Regarding causation of Brown's cancer, the witness described: "There are 25 (risk) factors in esophageal carcinoma." He cited alcohol, poor oral hygiene, chemical exposures, cleaning fluids, asbestos (the plaintiff was in the building trade), HPV (tumor never tested for HPV), reflux, and genetic predisposition. Regarding tobacco in Brown's esophageal cancer, "there is no way, in my opinion, to a medical degree of certainty, that you can say it was a contributing factor." He also opined, "Just because you smoke and get cancer doesn't necessarily mean that the smoking is the cause of cancer." The jury awarded Brown's heirs \$1.2 million in compensatory damages, which the court later reduced to \$600 thousand based on the jury's apportionment of fault.

In the 2011 case, *Kirkland v R.J. Reynolds*, the plaintiff had a 60-pack year smoking history and supraglottic laryngeal cancer.<sup>17</sup> In this case, witness 2 gave the opinions: 1) "I never saw a patient with throat cancer who had a solitary risk factor"; 2) "Patients that have supraglottic carcinoma, the cause is much more likely to be alcohol as opposed to tobacco or other causes"; and 3) "You aren't ruling out smoking as having an influence. It's just not the predominant cause, in my opinion." The jury awarded damages of only \$100 thousand in compensatory damages (reduced to \$10,000 by 90% apportionment of fault to the plaintiff) and added a punitive damage award of \$250 thousand.

## Witness 3

Witness 3 is a private practice otolaryngologist who was boarded in 1997. In 2011 testimony, he stated that he had reviewed seven cases in 2 years for tobacco-related cancers.<sup>18</sup>

In the 2011 case, *Szymanski v R.J. Reynolds et al.*, the 72-year-old patient, who started smoking at age 10 or 11 with a 100 pack year, developed laryngeal cancer that eventually required total laryngectomy.<sup>18,19</sup> The tumor was not tested directly for HPV, but the biomarker for HPV p16 was negative. When asked if

cigarettes caused the cancer, he stated: "Well, I think, you know, he smoked a lot; he drank a lot. So I tend to look at those as basically one risk factor." And I think he had reflux, which, as I said earlier, we think about 10 percent of patient(s) with those type of symptoms can develop laryngeal cancer. And we still haven't accounted for the possibility of HPV. So it's impossible for me to say that smoking alone was a substantial risk." He went on to say that: "I can't partition out the specific risk factors and say this one was more causative than the other." Verdict in this case was for the defense.

In the 2011 case, *Blich v R.J. Reynolds*, the plaintiff died of esophageal cancer in 1998.<sup>20</sup> When asked to allocate among the various risks, witness 3 stated: "*She drank significantly, she smoked. She did have a prior HPV cancer. And she does have reflux. . . . It is difficult for me to determine how to allocate how much risk there is for each entity. However, there are certain studies that where they have tried to factor out smoking and look at alcohol use and these studies have shown that alcohol by itself can cause esophageal cancer, that HPV infection is associated with the causation of esophageal cancer, and reflux can cause esophageal cancer. So I can't determine in Ms. Blich's case specifically which one has more weight or if they are all similar effect.*"

Even though the HPV status of the patient's esophageal cancer was unknown, because she had cervical cancer decades earlier, witness 3 opined there was a high probability that she also had HPV of the head and neck.

The jury found that Ms. Blich was addicted to smoking, and the addiction to smoking was the legal cause of the cancer that caused her death. Nevertheless, the jury blamed the plaintiff rather than the tobacco company; thus, the verdict in this case was for the defense.

## Witness 4

Witness 4 is a private practice otolaryngologist who was boarded in 1995. In 2011 testimony, she stated she had been retained by attorneys representing R.J. Reynolds 3 to 4 times in the preceding 5 years.<sup>21</sup>

In the 2011 case, *Reese v R.J. Reynolds*, the plaintiff smoked one pack per day for 55 years, since age 10, before developing supraglottic laryngeal cancer.<sup>21,22</sup> In deposition, the witness admitted that the law firm wrote her written legal opinion on risk factors in cancer causation and then she approved it. She opined that supraglottic tumors were not considered laryngeal, only glottic. Regarding the frequency by which smoking causes laryngeal cancer, the witness said: "20% to 50% of laryngeal cancer patients are nonsmokers." With regard to the Mrs. Reese's cancer etiology, she gave the opinion that: "*Most patients don't just smoke . . . therefore, you can't determine that smoking is the only risk factor. . . . I haven't had someone whose only risk factor was tobacco because they seem to partake in numerous things that increase their risk for developing cancer. . . . I look at the patient as a whole and consider the*

*multifactorial etiology or risk factors associated with their cancer because they all play a role*"

When asked about various studies in the published literature and whether they applied to the plaintiff, the witness stated: "I would agree that in the population they studied and as this knowledge is evolving, that is reflective of the population that they studied." The award was \$3.5 million for damages apportioned 70% plaintiff-30% R.J. Reynolds, yielding about a \$1 million payment to the plaintiff.

#### **Witness 5**

Witness 5 is a private practice otolaryngologist boarded in 1987. He described himself as a specialist in head and neck cancer, director of head and neck oncology at a local cancer center, and associate clinical professor in his local medical school. In a 2011 testimony, he indicated that he had reviewed at least three other tobacco defense cases for R.J. Reynolds in the past.<sup>23</sup>

In the 2011 case, *Syzmanski v R.J. Reynolds et al.*, the 72-year-old patient, who started smoking at age 10 or 11 with a 100-pack year, developed laryngeal cancer, which eventually required total laryngectomy (note: witness 3 also testified in this case).<sup>23</sup> Witness 5 indicated that the lawyers had engaged his services "to evaluate the risks of smoking in relation to laryngeal cancer." Regarding the risk factors for laryngeal cancer, he stated: "They include smoking. They would include chronic reflux. They would include HPV exposure disease. Previous radiation to the area. Toxic environmental exposures. Those would be the common ones. And alcohol is a contributing one. So it's a risk factor in combination with smoking. . . . Family history would also be a contributor."

With regard to the effects of quitting, the witness opined, if a person that smokes quits smoking ten years later, their risk of developing laryngeal cancer returns to that of a nonsmoker. The verdict in this case was for the defense.

#### **Witness 6**

Witness 6 was board certified in otolaryngology in 1992 and spent 7 years as a full-time faculty member before going into private practice. During a 2009 trial, witness 6 described testifying for Philip Morris in a 2003 case and giving opinions on at least four cases for the company by 2009.<sup>24</sup>

In 2009, this witness 6 testified in *Hess v RJ Reynolds and Philip Morris*. The plaintiff was a 40-year-old, chain-smoking, 3-pack-a day smoker who died of lung cancer in 1997.<sup>24</sup> The witness's testimony related to whether the plaintiff had a smoking hiatus in the early 1990s. This was part of a defense strategy to question the notion that he was addicted to nicotine. When the plaintiff was admitted for severe bronchitis several years before he died, endoscopy showed laryngeal leukoplakia. In the weeks following discharge, the leukoplakia turned to ulcerations and later improved. Witness 6 argued that this meant that the plaintiff had either quit smoking or

cut down to less than 1 pack a day. That he could halt or reduce smoking, even for a short period, was used to argue that the plaintiff was not addicted to the nicotine in the tobacco company's cigarettes and thus they were not liable.

The jury awarded \$8 million, of which \$5 million was for punitive damages. In 2012, the punitive damages were revoked on appeal.

### **Scientific Literature on Head and Neck Cancer Causation**

**Tobacco and Cancer Causation: The Legal Standard for Expert Testimony.** In civil actions, an expert witness is instructed to base their opinion on the preponderance of evidence, often explained with the phrase *more likely than not*. In the Florida Engle progeny cases, the central issue is whether the probability that smoking caused the plaintiff's cancer was > 50%.

Overwhelming evidence exists for dominant role of tobacco smoking in causation of upper-aerodigestive tract squamous cell carcinoma (SCC). In an extensive meta-analysis among those smoking more than 20 cigarettes per day, the odds ratio for all upper aerodigestive tract cancer was 4.42 and for laryngeal cancer was 19.0.<sup>25</sup> For smokers and drinkers, the odds ratio for laryngeal cancer was 27.9, highest among upper aerodigestive tract cancers. For expert witnesses to offer testimony exculpating tobacco from causing a smoker's cancer requires reaching the conclusion, based upon the scientific evidence, that more likely than not other factors were causative.

It is relevant to examine the epidemiology of the subset of head and neck SCC, which are not related to tobacco use. Tumors in nonsmokers tend to be located in the oropharynx, occur in younger men (< age 50), and be HPV-16 positive.<sup>26</sup> Those who don't smoke, chew, or have secondhand exposure to tobacco lack grounds to bring suit against the tobacco industry. All of the plaintiffs in this study were long-term, heavy cigarette smokers.

### **Human Papilloma Virus**

Human papilloma virus is often cited in testimony as a nontobacco cause of head and neck cancer. In a 2014 meta-analysis of 12,163 cases of head and neck squamous cell carcinomas, HPV was identified in 40.6% of oropharynx, 24.2% of oral cavity, and 22.1% of larynx cancers.<sup>27</sup> HPV is thought to have only a minor role in laryngeal cancer etiology, with HPV-16 present in only 6.5%.<sup>28</sup> Because tobacco use increases both HPV-associated and HPV-independent head and neck cancer, smoking history is relevant even in HPV positive cases. In a major University of Iowa study, significant interaction was found between tobacco and HPV in oropharyngeal cancer.<sup>29</sup>

### **Gastroesophageal Reflux**

Witnesses cited gastroesophageal reflux (GERD) as a causation of laryngeal cancer. Two recent reviews of

the literature concluded that there is insufficient evidence to link GERD with laryngeal cancer.<sup>30,31</sup> Reflux may be more common in laryngeal cancer patients, but this could well be because smokers and drinkers have more GERD. Because it is extremely common in the population, were GERD more than a minuscule risk factor, we would expect to see large number nonsmoking GERD patients developing laryngeal cancer.

### **Alcohol**

Alcohol is an important risk factor in head and neck cancer. The principal effect of ethanol is that it facilitates the effect of tobacco smoking.<sup>32</sup> In a large size study, drinking was not independently associated with oral/oropharyngeal cancer, only the joint effect of alcohol and tobacco together.<sup>33</sup> A witness described alcohol-containing mouthwash use as a risk factor, with Listerine (Johnson & Johnson, New Brunswick, NJ) cited as an example. Two recent comprehensive reviews showed no association between alcohol-containing mouthwash and oral cancer.<sup>34,35</sup>

### **Asbestos**

Witnesses often included exposure to asbestos as an alternative explanation for a smoker's head and neck cancer, alleging potential occupational exposure such as in the building trades, auto shops, or cotton mills. Although some studies suggest an additive effect with smoking, a meta-analysis concluded that the weight of the evidence does not support a causal association.<sup>36,37</sup>

### **Heredity**

Witnesses also cited heredity factors as an important cause in head and neck cancer. Whereas familial clusters of head and neck SCC have been observed, these are not common, are more frequent in smokers, and may be syndromic (e.g., Fanconi's anemia).<sup>38</sup> In a large series of laryngeal cancer patients, no significant increase in risk was found for family history of cancer at other sites.<sup>39</sup>

Testimony by one witness related uterine and breast cancer in the plaintiff's mother and sister and prostate and bladder cancer in his brothers as evidence justifying the witnesses opinion that: "The predominant influencing factor for this man was hereditary." We were unable to find literature to support the contention that a family history of women's or male urological cancers makes a relative prone to spontaneously developing laryngeal cancer absent tobacco exposure.

### **Environment, Diet, and Occupation**

The literature contains scattered articles evaluating possible associations between head and neck cancer and diet (e.g., meat consumption, coffee and tea drinking), occupational exposures (e.g., heavy metals, machinery fluid, polycyclic aromatic hydrocarbons, silica, cotton dust), home exposures (e.g., wood stoves, radon), and even urban versus rural living. Witnesses cited exposure

to gasoline and diesel fumes as potentially causative, even in a bookkeeper who worked in the back office of an automotive center. The relationship of diesel fume exposure to laryngeal cancer has been discounted.<sup>40</sup>

It is important to interpret these studies with caution; association does not imply causation. For example, analysis of occupational factors shows enhanced risk among blue collar workers, but the incidence of smoking is also higher in this demographic.<sup>41</sup> Coffee drinking was associated with a decreased risk of oral and oropharyngeal cancer, perhaps because frequent coffee drinkers quench their thirst with nonalcoholic drinks.<sup>42</sup> A large fraction of mankind is exposed to these factors. Were such widespread exposures significant causes of the head and neck cancer, a much larger cohort of nonsmoking cancer cases than observed would be expected.

### **Smoking Marijuana**

A 2015 comprehensive review of the literature on marijuana and cancer found that the data on association with head and neck cancer was limited and inconclusive.<sup>43</sup> Adjusting for other exposures, such as concomitant cigarette use and prevalence of HPV infection, complicates the study of this possible risk factor. The nearly universal reported use of cigarettes among heavy cannabis users was noted.

### **Effect of Smoking Cessation on Decline of Cancer Risk**

It was testified by multiple witnesses that the risk of laryngeal cancer dropped to the level of nonsmokers 10 years after smoking cessation. A review of four cohort and 15 case-control studies regarding postcessation reduction of risk of laryngeal cancer concluded that risk declined by about 60% 10 to 15 years postcessation, and to a greater degree after 20 years.<sup>44</sup> Even after 15 years, postcessation the risk is still three- to four-fold higher than in nonsmokers. In addition, the rate of risk decline is related to the amount smoked before cessation with heavy smokers, such as the plaintiffs with 40 to 80 pack years, at greater long-term risk.

## **DISCUSSION**

For many decades, the tobacco industry publicly denied that smoking caused adverse health consequences even though internal documents confirm that companies were well aware that the evidence was convincing.<sup>45,46</sup> As a result of legal proceedings in the late 1990s, the industry was forced to admit that its products cause cancer. Despite this admission, in litigation the industry routinely instructs its lawyers to put on vigorous defenses intended to deny that tobacco was causative of the individual plaintiff's cancer.<sup>47</sup> This tactical approach is abetted by legions of well-compensated "experts," who provide testimony proffering alternative causations for smokers who develop cancers of types typically caused by tobacco. Tobacco legal defense teams have honed methods to implant the notion in the jury that tobacco's role in a plaintiff's cancer is controversial

and that the scientific evidence is debatable. Their strategy is to manufacture doubt in the mind of jurors, who lack the technical background needed to critically analyze testimony on cancer causation. With extensive case experience, tobacco lawyers have crafted arguments carefully tailored to persuade those lacking scientific background.

This study involved examination of the opinions expressed by six otolaryngologists hired by tobacco companies to defend against allegations that their products caused the plaintiff's head and neck cancer. The legal standard in civil litigation is preponderance of the evidence. In these tobacco lawsuits, the issue was whether it is more likely than not (> 50%) that smoking caused the plaintiff's cancer. Otolaryngologists in this study routinely expressed the opinion that, more likely than not, tobacco did not cause the smoker's head and neck cancer.

Comments made in the legal records indicate that hired experts were specifically engaged "to look at risk factors." Testimony revealed that tobacco defense lawyers sometimes went so far as to write the opinion for the hired expert. The resulting testimony from multiple experts reads like peas in a pod—well coached and faithful to the tactical narrative that there are many, many causes of head and neck cancer—and that factors other than smoking must have caused the plaintiff's disease. During deposition and trial testimony, these witnesses cited a wide variety of nontobacco causes for the cancers, characterized by one witness as an "etiological soup" (Table III).

The tobacco industry strategy is based upon the assumption that juries may be swayed by long lists of theoretical causes, which tend to resonate with the public's belief that a wide variety of environmental, occupational, and dietary exposures cause cancer. An obvious fallacy of this argument lies in the fact that literally billions of nonsmoking people are exposed regularly to gasoline fumes, use cleaning solvents, eat salted fish, or live in urban environments. Were these causative factors for head and neck cancer, with even a minute fraction of the potency of tobacco, the rate of head and neck cancer among nonsmokers would be much greater than what has been observed. Among habitual smokers, it is not credible to opine that rare and hypothetical causes, taken singly or as a lengthy list, are more likely than causative than tobacco to a degree remotely approaching >50%.

In another example of bias, witnesses were selective in their claims of expertise willing to opine on subjects such as environmental and industrial toxicology, domains not typically within the expertise of an otolaryngologist. During testimony, multiple witnesses refused to agree that nicotine is addictive, citing lack of expertise, even though it has been widely accepted by the medical profession for decades. A central pillar of tobacco defense legal strategy is to maintain that the smoker has cancer due to personal choice rather than having been ensnared by addiction. Witnesses also denied the validity of widely recognized authoritative sources such as Surgeon General's reports

## CONCLUSION

Ethical experts provide testimony that is based upon a fair and balanced consideration of the scientific evidence, regardless of which of the litigants has solicited their opinion. Unethical experts bias their testimony to bolster the position of the side who hired them. Most often this is done by cherry-picking literature, contorting its meaning, or citing personal experience as though anecdote were more important than scientific facts. In writing about testimony in tobacco litigation, Maggi comments: "Most reputable scholars will not give partial and highly selective accounts of the available evidence depending upon who is paying freight," and goes on to say, "The tobacco industry pays generously and gets its money worth."<sup>48</sup> Discussing how tobacco friendly research escapes scrutiny in the courtroom, Friedman et al. opined: "They spend large sums cultivating experts and manufacturing evidence to confuse jurors about the relevant scientific issues."<sup>49</sup> Tobacco witness are typically well compensated. In the 2014 Cooper case, the witness's payment totaled approximately \$100 thousand.<sup>9</sup> In a European study, 33 of 45 medical expert witnesses testifying for tobacco companies received research funding before or after testifying.<sup>50</sup>

Some medical ethicists question whether it could ever be ethical for a physician to testify on behalf of the tobacco industry. In the opinion of Alderman: "Physicians never should serve as apologists for an industry whose product kill or injure large numbers of patients and pose a major threat to public health."<sup>51</sup> The tobacco industry continues to win a substantial fraction of cases, in part because of an asymmetry of resources between the industry and plaintiffs. They identify the best experts that money can buy, train them in their well honed narrative to manufacture doubt in the minds of the jury, and make use of them over and over in case after case. Together, the six otolaryngologists in this study helped to defend the tobacco industry in over 50 cases (Table II).

Numerous specialty societies have guidelines regarding ethical conduct among expert witnesses.<sup>52,53</sup> Since 2003, the American Academy of Otolaryngology–Head and Neck Surgery (AAO–HNS) has had such a policy in place.<sup>54</sup> Among other provisions it states: "Physician expert witnesses should not adopt a position as an advocate or partisan in the legal proceedings" and "the physician expert witness should be aware that transcripts of their deposition and courtroom testimony are public records, subject to independent peer review." When a formal review process has determined that an ethics code violation has occurred, the AAO–HNS policy specifies remedies that include possible censure, suspension, or expulsion. Noble organizations in otolaryngology should take stands to resist tobacco industry manipulation of the legal process by enforcing ethical standards in expert testimony to insure that it is unbiased and based on fair and balanced interpretation of the scientific literature.

## BIBLIOGRAPHY

- Daynard RA, LeGesley E. Strategic directions and emerging issues in tobacco control: Product liability. *Tob Control* 2012;21:227–228.
- Trial Testimony. *Ross v Philip Morris*. July 1962. <http://legacy.library.ucsf.edu/tid/idu47h00>. Accessed December 24, 2014.
- Jackler RK, Samji HA. The Price Paid—Mid 20th Century Tobacco funded research designed to obfuscate the emerging truth that smoking causes head and neck cancer. *Laryngoscope* 2012;122:75–87.
- Master Settlement Agreement 1998. <http://publichealthlawcenter.org/sites/default/files/resources/master-settlement-agreement.pdf>. Accessed February 24, 2015.
- Douglas CE, Davis RM, Beasley JK. Epidemiology of the third wave of tobacco litigation in the United States, 1994–2005. *Tob Control* 2006;15(suppl IV):9–16.
- Robles F. Jury award \$23.6 billion in Florida smoking case. *New York Times* July 19, 2014. [http://www.nytimes.com/2014/07/20/business/jury-awards-23-6-billion-in-florida-smoking-case.html?\\_r=0](http://www.nytimes.com/2014/07/20/business/jury-awards-23-6-billion-in-florida-smoking-case.html?_r=0). Accessed June 12, 2015.
- Sweda E. Public Health Advocacy Institute: Florida jury holds Philip Morris accountable for its actions in Federal Court. <http://www.PHAOnline.org>; Sept 25, 2014. Accessed January 2, 2015.
- Davis RM, Douglas CE, Beasley JK. The tobacco deposition and trial testimony archive (DATTA) project: origins, aims, and methods. *Tob Control* 2006;15(suppl IV): iv4–iv8.
- Trial Testimony. *Cooper v R.J. Reynolds*, et al. August 27, 2014. <http://legacy.library.ucsf.edu/tid/vsg37j00>. Accessed February 11, 2015.
- Deposition. *Mack v R.J. Reynolds*. March 9, 2010. Vol. 1: <http://legacy.library.ucsf.edu/tid/hyu20j00>. Vol. 2: <http://legacy.library.ucsf.edu/tid/iyu20j00>.
- Trial testimony. *Mack v R.J. Reynolds*. December 12, 2012. Circuit Court of Alachua County, Florida. CASE NO. 2008-CA-3256.
- Deposition. *Hetzner v R.J. Reynolds*, et al. November 13, 2009. <http://legacy.library.ucsf.edu/tid/gyu20j00>. Accessed December 26, 2014.
- Deposition. *Cooper v R.J. Reynolds*, et al. 17th judicial court of Broward County, Florida. May 8, 2014. CASE NO. 08-026350.
- Deposition. *Cooper v R.J. Reynolds*, et al. 17th judicial court of Broward County, Florida. August 1, 2014. CASE NO. 08-026350.
- Deposition. *Allgood v R.J. Reynolds*. March 4, 1994. <http://legacy.library.ucsf.edu/tid/ngt07a00>. Accessed December 21, 2014.
- Trial Testimony. *Brown v R.J. Reynolds*. May 14, 2009. <http://legacy.library.ucsf.edu/tid/yr020j00>. Accessed January 2, 2015.
- Trial testimony. *Kirkland v R.J. Reynolds*. February 4, 2011. <http://legacy.library.ucsf.edu/tid/zru20j00>. Accessed December 22, 2014.
- Deposition. *Syzmanski v R.J. Reynolds*, et al. May 13, 2011. <http://legacy.library.ucsf.edu/tid/oqv20j00>. Accessed December 26, 2014.
- Trial testimony. *Syzmanski v R.J. Reynolds*, et al. October 18, 2011. <http://industrydocuments.library.ucsf.edu/tobacco/docs/krpk0191.#.27#>.
- Deposition. *Blich v R.J. Reynolds*. February 15, 2011. <http://legacy.library.ucsf.edu/tid/nqv20j00>. Accessed December 26, 2014.
- Deposition. *Reese v R.J. Reynolds*, et al. January 5, 2011.#.29#. <http://legacy.library.ucsf.edu/tid/wxu20j00>. Accessed December 27, 2014.
- Trial testimony. *Reese v R.J. Reynolds*, et al. May 13, 2011. <http://legacy.library.ucsf.edu/tid/mtw20j00>. Accessed December 22, 2014.
- Deposition. *Syzmanski v R.J. Reynolds*, et al. May 6, 2011.#.32#. <http://industrydocuments.library.ucsf.edu/tobacco/docs/gmhl0191>. Accessed December 22, 2014.
- Trial testimony. *Hess v R.J. Reynolds, Philip Morris*. February 10, 2009. <http://industrydocuments.library.ucsf.edu/tobacco/docs/jxcl0154>. Accessed December 26, 2014.
- Ansary-Moghaddam A, Huxley RR, Lam TH, Woodward M. Risk of upper aerodigestive tract cancer associated with smoking with and without alcohol consumption. *Mt Sinai Med J* 2009;76:392–403. Accessed January 1, 2015.
- Curado MP, Boyle P. Epidemiology of head and neck squamous cell carcinoma not related to tobacco or alcohol. *Curr Opin Oncol* 2013;25:229–234.
- Ndiaye C, Mena M, Alemany L, et al. HPV DNA, E6/E7 mRNA, and p16INK4a detection in head and neck cancers: a systematic review and meta-analysis HPV DNA, E6/E7 mRNA, and p16INK4a detection in head and neck cancers: a systematic review and meta-analysis. *Lancet Oncol* 2014;15:1319–1331.
- Young RJ, Urban D, Angel C, Corry J, et al. Frequency and prognostic significance of p16INK4A protein overexpression and transcriptionally active human papillomavirus infection in laryngeal squamous cell carcinoma. *Br J Cancer* 2015;112:1098–1104. doi: 10.1038/bjc.2015.59.
- Smith EM, Rubenstein LM, Haugen TM, et al. Tobacco and alcohol use increases the risk of both HPV-associated and HPV-independent head and neck cancers. *Cancer Causes Control* 2010;21:1369–1378.
- Zhang D, Zhou J, Chen B, et al. Gastroesophageal reflux and carcinoma of the larynx and pharynx: a meta-analysis. *Acta Otolaryngol* 2014;134:982–989.
- Coca-Pelaz A, Rodrigo JP, Takes RP, et al. Relationship between reflux and laryngeal cancer. *Head Neck* 2013;35:1814–1818.
- La Vecchia C, Zuo Feng Zhang ZF, Altier A, et al. Alcohol and laryngeal cancer: an update. *Euro J Cancer Prev* 2008;17:116–124.
- Antunes JLF, Toporcov TN, Haye Biazevic MGH, et al. Joint and independent effects of alcohol drinking and tobacco smoking on oral cancer: a large case-control study. *PLoS ONE* 2013;8:e68132. doi: 10.1371/journal.pone.0068132.
- Currie S, Farah CS. Alcohol-containing mouthwash and oral cancer risk: a review of current evidence. *OA Alcohol* 2014;2:4.
- Gandini S, Negri E, Boffetta P, La Vecchia C, Boyle P. Mouthwash and oral cancer risk—quantitative meta-analysis of epidemiologic studies. *Ann Agricultural Environ Med* 2012;19:172–180.
- Griffiths H, Molony NC. Does asbestos cause laryngeal cancer? *Clin Otolaryngol Allied Sci* 2003;28:177–182.
- Browne K, Gee JBL. Asbestos exposure and laryngeal cancer. *Ann Occup Hyg* 2000;44:239–250.
- Lacko M, Braakhuysen BJM, Sturgis EM, et al. Genetic susceptibility to head and neck squamous cell carcinoma. *Int J Radiation Oncol Biol Phys* 2014;89:38–45.
- Garavello W, Turati F, Bosetti C, Talamini R, et al. Family history of cancer and the risk of laryngeal cancer: a case-control study from Italy and Switzerland. *Int J Cancer* 2012;130:665–670.
- Muscat JE, Wynder EL. Diesel exhaust, diesel fumes, and laryngeal cancer. *Otolaryngol Head Neck Surg* 1995;112:437–440.
- Paget-Bailly S, Cyr D, Luce D. Occupational exposures and cancer of the larynx-systematic review and meta-analysis. *J Occup Environ Med* 2012;54:71–84.
- Galeone C, Tavani A, Pelucchi C, Turati F, et al. Coffee and tea intake and risk of head and neck cancer: pooled analysis in the international head and neck cancer epidemiology consortium. *Cancer Epidemiol Biomarkers Prev* . 2010;19:1723–1736.
- Huang Y-H, Zhang Z-F, Tashkin DP, et al. An epidemiologic review of marijuana and cancer: an update. *Assn Am Cancer Res* 2015;24:15–31. doi: 10.1158/1055-9965.EPI-14-1026.
- Bosetti C, Garavello W, Gallus S, La Vecchia C. Effects of smoking cessation on the risk of laryngeal cancer: overview of published studies. *Oral Oncol* 2006;42:866–872.
- Proctor R. *Golden Holocaust*. Oakland, CA: University of California Press; 2011.
- Oreskes N, Conway EM. *Merchants of Doubt*. Bloombury Press; 2011.
- Milberger S, Davis RM, Gouglas CE, et al. Tobacco manufacturers' defense against plaintiffs' claims of cancer causation: throwing mud at the wall and hoping some of it will stick. *Tob Control* 2006;15(suppl 15):17–26.
- Maggi L. Bearing witness for tobacco. *J Public Health Policy* 2000;21:296–302.
- Friedman LC, Daynard RA, Banthin CN. How tobacco friendly science escapes scrutiny in the courtroom. *Am J Public Health* 2005;95(suppl 1):S16–S20.
- Hilamo, HT. The impact of strategic funding by the tobacco industry of medical expert witnesses appearing for the defence in the Aho Finnish product liability case. *Addiction* 2007;102:979–988.
- Alderman J. Ethical implications of physician involvement in lawsuits on behalf of the tobacco industry. *J Law Med Ethics* 2007;35:692–698, 513.
- Svider PF, Eloy JA, Bareded S, et al. Expert witness testimony guidelines: identifying areas for improvement. *Otolaryngol Head Neck Surg* 2015;152:207–201.
- Ward NO. The essential and ethical expert: serving as a medical expert witness in the 21st century. *Otolaryngol Head Neck Surg* 2007;136:4–7.
- American Academy of Otolaryngology—Head and Neck Surgery expert witness policy (revised October, 2012). [https://www.entnet.org/sites/default/files/Expert%20Witness%20Testimony%20Policy\\_0.pdf](https://www.entnet.org/sites/default/files/Expert%20Witness%20Testimony%20Policy_0.pdf). Accessed February 12, 2015.