CHEST®

Official publication of the American College of Chest Physicians

Effects of Water-Pipe Smoking on Lung Function : A Systematic Review and Meta-analysis

Dany Raad, Swarna Gaddam, Holger J. Schunemann, Jihad Irani, Philippe Abou Jaoude, Roland Honeine and Elie A. Akl

Chest 2011;139;764-774; Prepublished online July 29, 2010;
DOI 10.1378/chest.10-0991

ISSN:0012-3692

CHEST

Original Research

COPD

Effects of Water-Pipe Smoking on Lung Function

A Systematic Review and Meta-analysis

Dany Raad, MD; Swarna Gaddam, MBBS, MPH; Holger J. Schunemann, MD, PhD, FCCP; Jihad Irani, MD, MPH; Philippe Abou Jaoude, MD; Roland Honeine, MD; and Elie A. Akl, MD, PhD, MPH

Background: **Although common in many Middle Eastern countries, water-pipe tobacco smoking, commonly known as water-pipe smoking (WPS), is increasingly popular in Western cultures. The primary objective of this study was to systematically review the effects of WPS on lung function. The secondary objective was to compare the effects of WPS and cigarette smoking on lung function.**

Methods: **We conducted a systematic review using the approach of the Cochrane Collaboration to search for, select, and abstract studies. We conducted two separate meta-analyses comparing water-pipe smokers with nonsmokers, and water-pipe smokers with cigarette smokers for each of three spirometric measurements (FEV_1, FVC, and FEV_1/FVC). We used the standardized mean difference (SMD) to pool the results.**

Results: **Six cross-sectional studies were eligible for this review. Compared with no smoking, WPS was associated with a statistically significant reduction in FEV_1 (SMD = −0.43; 95% CI, −0.58 to −0.29; equivalent to a 4.04% lower FEV_1%), a trend toward lower FVC (SMD = −0.15; 95% CI, −0.34 to 0.04; equivalent to a 1.38% reduction in FVC%), and lower FEV_1/FVC (SMD = −0.46; 95% CI, −0.93 to 0.01; equivalent to a 3.08% lower FEV_1/FVC). Comparing WPS with cigarette smoking, there was no statistically significant difference in FEV_1, FVC, and FEV_1/FVC. The six studies suffered from methodologic limitations.**

Conclusions: **WPS negatively affects lung function and may be as harmful as cigarette smoking. WPS, therefore, is likely to be a cause of COPD.** ***CHEST 2011; 139(4):764–774***

Abbreviations: GRADE = Grading of Recommendations Assessment, Development and Evaluation; SMD = standardized mean difference; WPS = water-pipe smoking

COPD is a preventable and treatable disease characterized by an abnormal inflammatory response of the lung to noxious particles or gases (eg, tobacco) leading to a progressive and nonreversible airflow obstruction.[1,2] COPD has become a leading cause of mortality and morbidity throughout the world. The Global Burden of Disease study has projected that it will become the third leading cause of death by 2020.[3] The World Health Organization classifies COPD as the fourth leading cause of mortality in the United States.[4]

For editorial comment see page 737

The causal relationship between long-term cigarette smoking and COPD is clearly established,[5-7]

Manuscript received April 18, 2010; revision accepted June 7, 2010.

Affiliations: From the Department of Medicine (Drs Raad, Schunemann, Abou Jaoude, Honeine, and Akl), and the Department of Family Medicine (Drs Gaddam and Akl), State University of New York at Buffalo, Buffalo, NY; the Departments of Clinical Epidemiology and Biostatistics and Medicine (Drs Schunemann and Akl), McMaster University, Hamilton, ON, Canada; and the Faculty of Health Sciences (Dr Irani), University of Balamand, Beirut, Lebanon.

Correspondence to: Elie A. Akl, MD, PhD, MPH, Department of Medicine, State University of New York at Buffalo, ECMC-CC 142, 462 Grider St, Buffalo, NY 14215; e-mail: elieakl@buffalo.edu

DOI: 10.1378/chest.10-0991

with cigarette smoking being the single most important risk factor.[6] A recent systematic review showed that the prevalence of COPD is highest among male smokers who are > 40 years old.[8]

Water-pipe smoking (WPS) is a form of tobacco consumption that is increasing on a global level at a remarkable pace (see e-Appendix 1 and Fig 1 for further details on WPS).[9] The American Lung Association has described it as an "emerging deadly trend."[10] In fact, a recent systematic review found WPS to be possibly associated with lung cancer, esophageal cancer, low birth weight, and periodontal diseases.[11]

The above-referenced systematic review identified no study assessing the association of WPS with airways diseases in general or COPD in particular. There are, however, published studies assessing the association of WPS with lung function measurements. Some of these measurements (ie, FEV_1, FVC, FEV_1/FVC) are considered valid surrogate measures of COPD.[12] In the absence of data on clinical disease, data on such surrogates may be useful for both clinical and research purposes. Thus, the primary objective of this study was to systematically review the effects of WPS on lung function. The secondary objective was to compare the effects of WPS and cigarette smoking on lung function.

Materials and Methods

Eligibility Criteria

We included studies that assessed the association between water-pipe tobacco use and lung function. Eligible studies had to include a group of individuals smoking a water pipe exclusively. They also had to include at least one of the following: (1) a group of nonsmokers, (2) a group of individuals practicing exclusively cigarette smoking. Our outcomes of interest were the following three spirometric measurements: FEV_1, FVC, and FEV_1/FVC.

Search Strategy

In June 2008, we searched the following electronic databases starting with their dates of inception: MEDLINE, EMBASE, and ISI the Web of Science. e-Appendix 2 provides the detailed search strategies. We also reviewed the reference lists of included and other relevant papers and used the Related Articles function in PubMed and applied no language restrictions.

Selection Process

Two reviewers independently screened titles and abstracts resulting from the search using a standardized screening guide. We obtained the full text of citations considered as potentially eligible by at least one of the two reviewers. Next, the two reviewers independently screened the full texts for eligibility using a standardized and pilot tested form. Disagreements were resolved by discussion or by a third reviewer.

Data Abstraction

One reviewer abstracted data from each eligible study using a standardized and pilot-tested data abstraction form. A second reviewer verified data abstraction. They resolved disagreements with the help of a third reviewer. The abstracted data included information about:

1. Study design and funding;
2. Population: setting and period, and participants' characteristics;
3. Exposure: type, measurement tool, and exposure levels of participants;
4. Outcomes: measurement tool and blinding of outcome adjudicator;
5. Methodologic features: selection method, information collection (measurement of exposure and outcome), handling of confounding, participation rate, and rate of complete data;
6. Statistical results.

We collected data separately for the three different exposure groups (water-pipe smokers, cigarette smokers, nonsmokers). FEV_1, FVC, and FEV_1/FVC were calculated as percentages of predicted values and reported at the group level as mean and SD of these percentages.

Data Analysis

We calculated the κ statistic to evaluate the agreement between the two reviewers assessing full texts for eligibility. For each of the three spirometric measurements (FEV_1, FVC, and FEV_1/FVC),

Figure 1. Annotated figure of a water-pipe device.

we conducted two separate meta-analyses comparing water-pipe smokers with nonsmokers and water-pipe smokers with cigarette smokers. Because the populations and lung function measures differed across studies, we first calculated the standardized mean difference (SMD) and 95% CI for each outcome in the individual studies. The SMD expresses a measurement in standard units rather than the original units of measurement. We then pooled the SMDs across studies using a random effects model. In a sensitivity analysis, we excluded studies in which the nonsmokers were described as passive smokers. We translated the pooled SMD back into mean differences using the standard deviation for the respective spirometric outcomes derived from the National Health and Nutrition Examination Survey (NHANES) III data.[13]

We tested results for homogeneity across studies using the I^2 test[14] and used the following interpretation of the value of I^2: 0 to 50 = low, 50 to 80 = moderate and worthy of investigation, 80 to 100 = severe and worthy of understanding, 95 to 100 = aggregate with major caution (Julian Higgins, PhD, personal communication). We rated the overall quality of evidence using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach.[15]

Results

Description of Included Studies

Figure 2 shows the study flow. Of 1,658 identified citations, we included six studies (Table 1)[16-21] One study reported results separately for men and women.[18] All studies included a group of nonsmokers, described as passive smokers in two of the studies.[19,21] All but one study included a group of cigarette smokers.[19] All studies reported spirometric measurements. Countries in which the studies were conducted were Turkey (n = 3), Kuwait (n = 1), Saudi Arabia (n = 1), and Syria (n = 1).

Methodologic Quality of Included Studies

The six included studies were cross-sectional and suffered from a number of methodologic limitations. All the studies used an objective outcome evaluation (measurement by spirometry). Authors calculated the percent predicted spirometric values using the Communité Europeenne du Carbon e de l'Acier method in two studies,[17,20] and the Knudson and Hankinson methods in one study.[21] The remaining studies did not report any method.[16,18,19] None of the studies reported using a standardized exposure assessment tool. Selection of subjects was either done by visiting local coffee shops,[16,17,21] by volunteer recruitment,[18,19] or by a field survey.[20] Only one study reported handling confounding by matching for gender[17]; two other studies reported no difference between mean age for the groups involved.[19,21] Only one study reported blinding of outcome adjudicator,[16] and only one study reported the percentages of participation (88%) and complete data (96%).[20]

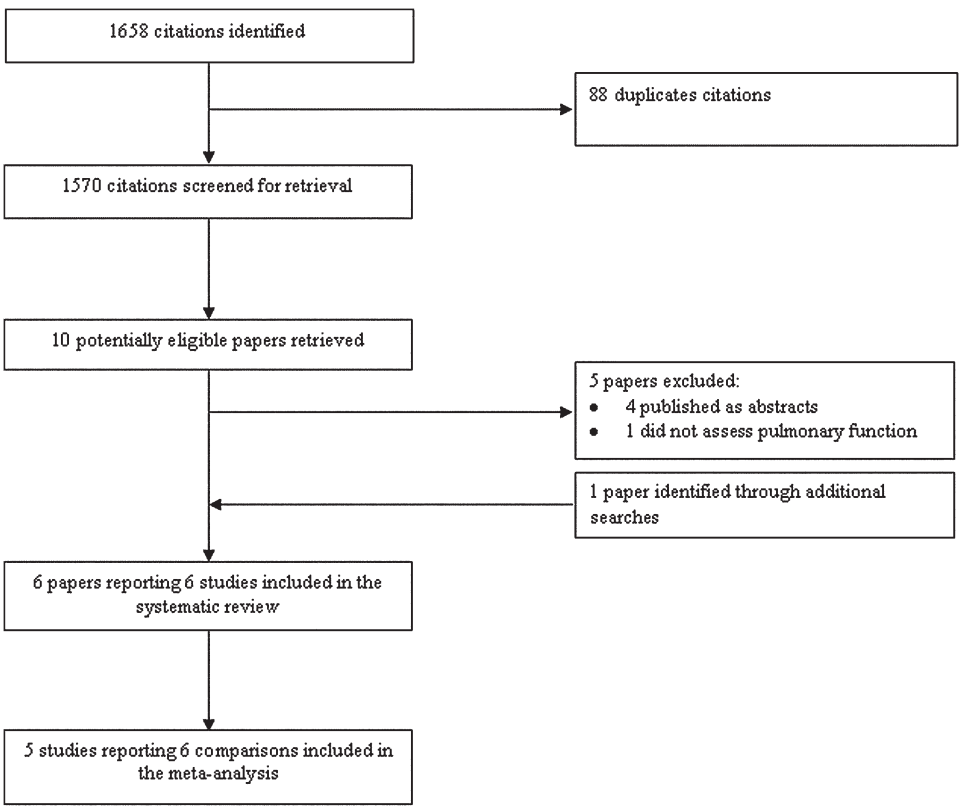

Figure 2. Study flow diagram.

Table 1—*Characteristics of Included Studies Measuring the Association Between WPS and Lung Function*

Study/Year	Population	Exposure	Outcome	Methodologic Characteristics	Results
Kiter et al[17]/2000 Study design: cross-sectional study Funding: not reported	Setting and period: special cafes in Izmir City (Turkey), period not reported Participants' characteristics: 302 subjects, 100% men Water-pipe smokers: 82 subjects, aged 56 ± 10 y Cigarette smokers: 103 subjects, aged 46 ± 14 y Water-pipe smokers who quit cigarette smoking: 95 subjects, aged 54 ± 12 y Nonsmokers: 117 subjects, aged 46 ± 16 y	Type: water pipe, cigarette Measurement: self-developed tool, no standardization reported; personal interview Water-pipe cumulative consumption measured as number of jurak-years = amount of jurak smoked/d × number of smoking years Cigarette cumulative consumption measured as pack-years = number of cigarette packs smoked/d × number of smoking years Exposure levels of participants: Water-pipe smokers: 47 ± 33 jurak-years Water-pipe smokers who quit cigarette smoking: 37 ± 42 jurak-years and 38 ± 32 pack-years Cigarette smokers: 38 ± 30 pack-years	Pulmonary function: Measurement tool: portable spirometer according to the standards of the American Thoracic society; percentage predicted values calculated automatically according to the Communité Europeenne du Carbon e de l'Acier method Blinding of outcome adjudicator: not reported	Selection: water-pipe smokers selected from special cafes, cigarette smokers and nonsmokers selected randomly from cafes and outside cafes Information collection: objective outcome evaluation, nonstandardized exposure assessment tool Confounding handling: matching done for gender only % Participation: not reported % Complete data: not reported	FEV_1, FVC, and FEV_1/FVC nonsignificantly lower in water-pipe smokers than nonsmokers FEV_1 and FEV_1/FVC significantly lower in cigarette smokers than water-pipe smokers FVC nonsignificantly lower in cigarette smokers than water-pipe smokers There was negative correlation of pack-years with FEV_1 in cigarette smokers, but no correlation between FEV_1 and jurak-years in water-pipe smokers
Al-Fayez et al[18]/1988 Study design: cross-sectional study Funding: King AbdulAziz City for Science and Technology	Setting and period: Saudi Arabia, period not reported Participants' characteristics: 595 subjects, men aged 20-59 y and women 17-59 y. Subjects with history of asthma, chronic bronchitis, or any cardiopulmonary diseases excluded. Water-pipe smokers: 344 subjects, 73% men Cigarette smokers: 251 subjects, 75% men Nonsmokers: 283 subjects, 58% men	Type: water pipe, cigarette Measurement: self-developed tool, no standardization reported; personal interview Exposure levels of participants: not reported	Pulmonary function: Measurement tool: time revolving spirogram (Vitalograph); method of calculation of percent predicted values not reported Blinding of outcome adjudicator: not reported	Selection: volunteer recruitment Information collection: objective outcome evaluation, nonstandardized exposure assessment tool Confounding handling: no matching or adjustment in the analysis reported % Participation: not reported % Complete data: not reported	FEV_1, FVC, and FEV_1/FVC significantly lower in water-pipe smokers than nonsmokers FEV_1 and FVC significantly lower in men and nonsignificantly lower in women in cigarette smokers than water-pipe smokers FEV_1/FVC significantly lower in both men and women in cigarette smokers than water-pipe smokers Heavy water-pipe smokers (> 2 water pipe/d) exhibited

(Continued)

Table 1—*(Continued)*

Study/Year	Population	Exposure	Outcome	Methodologic Characteristics	Results
					a marked decline in FEV_1 and FEV_1/FVC compared with light smokers (1-2 water pipe/d)
Al Mutairi et al[16]/2006 Study design: cross-sectional study Funding: Kuwait University Research Administration Grant	Setting and period: local coffee shops and university students in Kuwait, period not reported Participants' characteristics: 168 subjects, all subjects who smoked more than one kind of tobacco, had chronic cardiopulmonary or renal diseases, or were on regular medications were excluded from the study Water-pipe smokers: 77 subjects, 90% men, aged 36.97 (34.79-39.16) y Cigarette smokers: 75 subjects, 93% men, aged 37.73 (35.54-39.95) y Nonsmokers: 16 subjects, 56% men, aged 33.3 (28.9-37.76) y	Type: water pipe, cigarette Measurement: self-developed tool, no standardization reported; constructed questionnaire that identifies smoking behavior of both groups, detailing rate of consumption of tobacco/d Objective measurement tool: urinary cotinine and nicotine level Exposure levels of participants: Water-pipe smokers: Age of starting smoking: 24.89 (22.61-27.17) y Urinary nicotine = 440.48 (197.06-683.91) ng/mL Urinary cotinine = 677.62 (458.89-896.34) ng/mL Cigarette smokers: Age of starting smoking: 20.36 (18.55-22.17 = 8) y Urinary nicotine = 1487.30 (839.26-2135.37) ng/mL Urinary cotinine = 1321.35 (1003.73-1638.98) ng/mL	Pulmonary function: Measurement tool: Jaeqar Masterlab spirometry; method of calculation of percentage predicted values not reported Blinding of outcome adjudicator: yes	Selection: subjects selected from local coffee shops and among university students Information collection: objective outcome evaluation, nonstandardized exposure assessment tool Confounding handling: no matching or adjustment in the analysis reported % Participation: not reported % Complete data: not reported	FEV_1 and FEV_1/FVC nonsignificantly lower in water-pipe smokers than nonsmokers FEV_1, FVC, and FEV_1/FVC nonsignificantly lower in water-pipe smokers than cigarette smokers There was no significant difference in FEV_1 and FEV_1/FVC values in relation to duration of smoking (< 10 y vs > 10 y) in both cigarette and water-pipe smokers
Aydin et al[19]/2004 Study design: cross-sectional study Funding: not reported	Setting and period: Turkey, period not reported Participants' characteristics: 25 subjects, all subjects had no significant lung or other system disease or cigarette smoking history Water-pipe smokers: 14 subjects, 100% men, aged 53.7 ± 9.8 y	Type: water pipe Measurement: self-developed tool, no standardization reported; personal interview Exposure levels of participants: Water-pipe smokers: 23.7 ± 8.7 y Passive smokers: 21.3 ± 5.2 y	Pulmonary function: Measurement tool: Sensor Media V max 22; method of calculation of percentage predicted values not reported Blinding of outcome adjudicator: not reported	Selection: volunteer recruitment Information collection: objective outcome evaluation, nonstandardized exposure assessment tool Confounding handling: matching not reported but no significant difference between the mean age of the two groups	FEV_1, FVC, and FEV_1/FVC nonsignificantly lower in water-pipe smokers than nonsmokers

(Continued)

Table 1—*(Continued)*

Study/Year	Population	Exposure	Outcome	Methodologic Characteristics	Results
	Passive smokers: defined as individuals who do not smoke but are exposed to intensive cigarette smoke in the living or working environment: 11 subjects, 91% men, aged 43.8 ± 12.9 y			% Participation: not reported % Complete data: not reported	
Mohammad et al[20]/2008 Study design: cross-sectional study Funding: not reported	Setting and period: Syria, first semester of 1994-1995 Participants' characteristics: 254 subjects, 100% women, no comorbidity, no respiratory symptoms related to factors other than smoking, and no exposure to other risk factors in her daily life; none of the subjects smoked both water pipe and cigarettes Water-pipe smokers: 77 subjects, aged 40.99 ± 12.54 y Cigarette smokers: 77 subjects, aged 44.84 ± 10.55 y Nonsmokers: 100 subjects, aged 39.13 ± 12.898 y	Type: water pipe, cigarette Measurement: self-developed pilot-tested tool, no further validation reported Water-pipe cumulative consumption calculated using: $Q = S \times q\ (g) \times T\ (d)/1{,}000$ Cigarette cumulative consumption calculated using: $Q = N\ (g) \times T\ (d)/1{,}000$ Exposure levels of participants: Water-pipe smokers: 58 ± 3.8 kg, 60 ± 3.81 y Cigarette smokers: 40 ± 3.8 kg, 38 ± 3.81 y	Pulmonary function: Measurement tool: automatic calibrated spirometer; percent predicted values calculated automatically according to the Communité Europeenne du Carbon e de l'Acie method Blinding of outcome adjudicator: not reported FEV_1: considered abnormal of < 80% FEV_1/FVC: considered to reflect obstruction if < 70%	Selection: subjects recruited by field survey Information collection: objective outcome evaluation, nonstandardized exposure assessment tool Confounding handling: no matching or adjustment in the analysis reported % Participation: 88 % Complete data: 96	FEV_1, FVC, and FEV_1/FVC significantly lower in water-pipe smokers than nonsmokers FEV_1, FVC, and FEV_1/FVC significantly lower in cigarette smokers than water-pipe smokers There was a negative correlation for cumulative quantity of both cigarette and WPS with FEV_1, FVC, and FEV_1/FVC.
Köseoğlu et al[21]/2006 Study design: cross-sectional study Funding: not reported	Setting and period: local coffee shops in Izmir City (Turkey), period not reported Participants' characteristics: 58 subjects, all subjects who had history of systemic or pulmonary disease, COPD, lung cancer, previous lung surgery, common cold or viral respiratory disease, allergy history, history of active pulmonary infection, or taking medications that increase or decrease mucociliary clearance were excluded	Type: water pipe, cigarette Measurement: self-developed tool, no standardization reported; personal interview Objective measurement tool (urinary cotinine level) Exposure levels of participants: Water-pipe smokers: Average amount of tobacco = 35.5 ± 22.8/y Urinary cotinine = 838.9 ± 762.5 ng/mL Cigarette smokers:	Pulmonary function: Measurement tool: spirometer (Sensor Medics V-Max) according to the American Thoracic Society; percentage predicted values calculated according to Knudson and Hankinson methods Blinding of outcome adjudicator: not reported	Selection: subjects selected from local coffee shops Information collection: objective outcome evaluation, nonstandardized exposure assessment tool Confounding handling: matching not reported, no significant difference in ages, BMI, and pulmonary function test parameters % Participation: not reported % Complete data: not reported	FEV_1, FVC, and FEV_1/FVC nonsignificantly lower in water-pipe smokers than nonsmokers FEV_1, FVC, and FEV_1/FVC nonsignificantly lower in cigarette smokers than water-pipe smokers

(Continued)

Table 1—(Continued)

Study/Year	Population	Exposure	Outcome	Methodologic Characteristics	Results
	Water-pipe smokers: 20 subjects, aged 56.1 ± 8.4 y Cigarette smokers: 23 subjects, aged 52.0 ± 5.7 y Passive smokers: 15 subjects, aged 54.5 ± 19 y	Average amount of tobacco = 36.2 ± 23.1 pack-years Urinary cotinine = 1,576 ± 974.9 ng/mL			

Data are presented as mean ± SD unless otherwise indicated. Jurak = type of tobacco paste that contains various spices and dried fruits; N = number of cigarettes/d; Q = cumulative quantity smoked, kg; q = quantity smoked per session, g; S = number of sessions/d; T = duration of smoking, d; WPS = water-pipe smoking.

WPS Compared With Nonsmoking

The pooled SMD for FEV_1 was −0.43 (95% CI, −0.58 to −0.29; $I^2 = 24\%$) equivalent to a 4.04% lower $FEV_1\%$ value in the water-pipe group (Fig 3). In the sensitivity analysis excluding studies in which the nonsmokers were described as passive smokers, the pooled SMD remained statistically significant at −0.46 (95% CI, −0.60 to −0.31; $I^2 = 21\%$). The SMD for FVC was −0.15 (95% CI, −0.34 to 0.04; $I^2 = 0\%$), equivalent to a 1.38% reduction in FVC% in the water-pipe group (Fig 4). In the sensitivity analysis, the pooled SMD was −0.19 (95% CI, −0.40 to 0.01; $I^2 = 0\%$). The SMD for FEV_1/FVC was −0.46 (95% CI, −0.93 to 0.01; $I^2 = 92\%$), suggesting a lower percent predicted value in the water-pipe group, by 3.08% (Fig 5). In the sensitivity analysis, the pooled mean difference was −0.51 (95% CI, −1.06 to 0.05; $I^2 = 94\%$). The GRADE overall quality of evidence for FEV_1 was moderate; it was downgraded secondary to study limitations.

WPS Compared With Cigarette Smoking

The pooled SMD for FEV_1 between the two groups was 0.20 (95% CI. −0.15 to 0.55; $I^2 = 87\%$), which translates into a nonsignificantly lower $FEV_1\%$ by 1.88% in the cigarette smoking group (Fig 6). The SMD for FVC between the two groups was 0.27 (95% CI, 0.09-0.44; $I^2 = 83\%$), which translates into a significantly lower percentage predicted value in the cigarette smoking group, by 2.48% (Fig 7). The SMD in FEV_1/FVC between the two groups was 0.22 (95% CI, −0.29 to 0.73; $I^2 = 94\%$), suggesting a nonsignificantly lower percentage predicted value (1.47%) in the water-pipe group (Fig 8). The GRADE overall quality of evidence for FEV_1 was low; it was downgraded secondary to study limitations and imprecision.

Association Between the Duration of WPS and Lung Function

Four studies reporting on the association between the duration of WPS and lung function had mixed results. Two studies reported no correlation between the duration of WPS and the decline in FEV_1.[16,17] The other two studies reported an association; one found a marked decline in FEV_1 and FEV_1/FVC when comparing heavy smokers (> 2 water pipe/d) with light smokers (1-2 water pipe/d), whereas the other reported a negative correlation for cumulative quantity of WPS with FEV_1, FVC, and FEV_1/FVC.

Discussion

We systematically reviewed the scientific literature for the effects of WPS on lung function. Compared

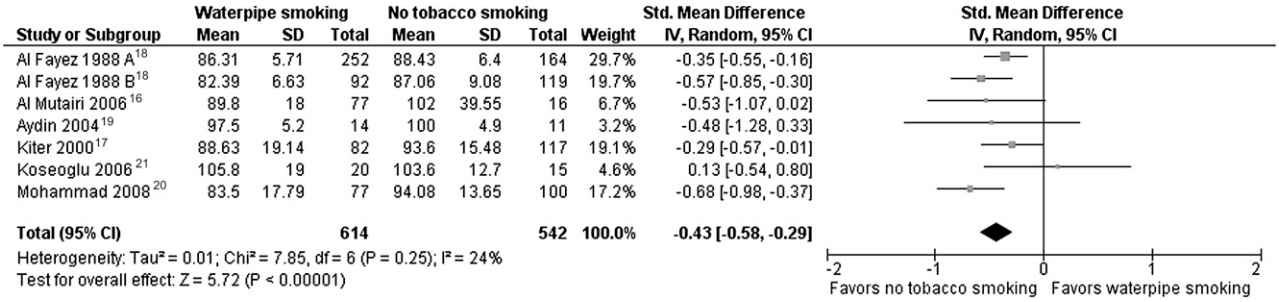

FIGURE 3. Comparison of FEV_1 in water-pipe smokers and nonsmokers. Al Fayez 1988 A includes only the male participants; Al Fayez 1988 B includes only the female participants. IV = inverse variance; Std = standard.

with no smoking, WPS was associated with a statistically significant reduction of FEV_1 and a trend toward lower FVC and FEV_1/FVC. The quality of evidence is moderate (ie, further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate).[15] There were no statistically significant differences in FEV_1 and FEV_1/FVC between water-pipe smokers and cigarette smokers. The quality of evidence was low (ie, further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate).[15]

Our study has a number of strengths. First, we used the comprehensive approach of Cochrane Collaboration for conducting systematic reviews, including a very sensitive search strategy, a duplicate and independent selection and data abstraction processes, and a rigorous evaluation of study methodological quality. Also, by pooling results across studies we were able to obtain relatively precise estimates of the outcomes of interest. Last, this is the first meta-analysis, to our knowledge, that assesses the association of WPS with lung function.

The study also has a number of limitations. Although the primary objective of this study was to assess the effects of WPS on lung function, the available data from cross-sectional studies provide evidence for an association but does not establish causality. Our confidence in the results of the meta-analysis is reduced by the methodologic limitations of the included individual studies. Indeed, none of the studies used a standardized tool to measure the degree of exposures of interest (eg, in terms of smoking patterns, frequency and lengths of smoking sessions, the type and quality of tobacco used for both WPS and cigarette smoking). This is particularly problematic given the potential variability in exposure. Finally, all but two studies failed to distinguish passive smokers from nonsmokers for exposure to WPS or cigarette smoking. However, the results of sensitivity analyses excluding these two studies were consistent with the results of the main analyses.

The association between WPS and reduction in FEV_1 is not only statistically significant but also of potential clinical relevance. We can assess the clinical relevance comparing the effect size to the minimal important difference, defined as "the smallest difference in score in the outcome of interest that informed patients or informed proxies perceive as important, either beneficial or harmful, and which would lead the patient or clinician to consider a change in management." It has been suggested that the minimal important difference for FEV_1 is in the range of 100 to 140 mL.[22] The mean difference in our study was estimated to be around 4%, which approximates to 173 mL for a 40-year-old white man of 180 cm height. Therefore, the reduction of FEV_1 associated with WPS is clinically relevant.

The association of WPS with a significant reduction in FEV_1 suggests its implication as a risk factor for obstructive disease. This is consistent with the finding of a trend toward reduction in FVC and FEV_1/FVC among water-pipe smokers. The lack of statistical significance for these reductions is likely due to the lack of statistical power. Taken together,

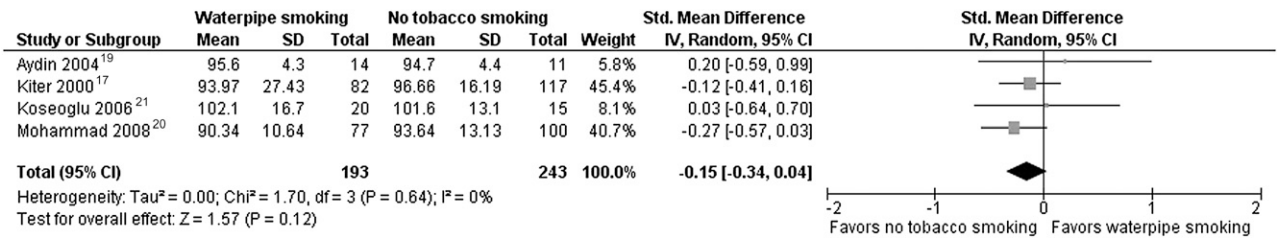

FIGURE 4. Comparison of FVC in water-pipe smokers and nonsmokers. See Figure 3 legend for expansion of abbreviations.

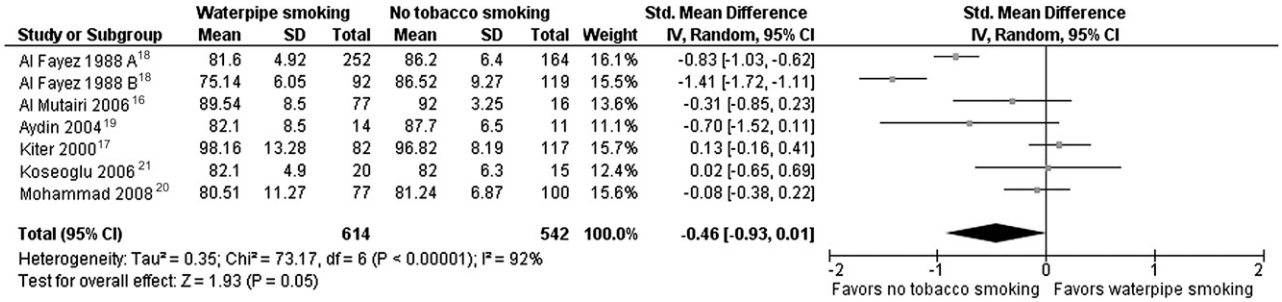

FIGURE 5. Comparison of FEV_1/FVC in water-pipe smokers and nonsmokers. Al Fayez 1988 A includes only the male participants; Al Fayez 1988 B includes only the female participants. See Figure 3 legend for expansion of abbreviations.

however, these findings suggest a possible role of WPS in the development of COPD.

There is additional evidence of the implication of WPS in COPD. Two studies using questionnaires adapted from the Medical Research Council[16] and the European Coal and Steel Community[20] on the symptoms of chronic bronchitis identified statistically significant higher number of positive responses in water-pipe smokers than in cigarette smokers. In the first study, symptoms of chronic bronchitis were identified in 11.75% of water-pipe smokers, as compared with 9.5% of cigarette smokers, and 0% in nonsmokers.[16] In the second study, chronic bronchitis was found to be more prevalent in water-pipe smokers than cigarette smokers for cumulative quantity and duration.[20] Despite the limited available data, these results help add up to the evidence of the risk of development of COPD in water-pipe smokers.

Although the results of FEV_1 and FEV_1/FVC comparing WPS to cigarette smoking show no statistical difference, they suffered from a high level of heterogeneity. The most likely explanation for this heterogeneity is the variation of levels of exposure to the two forms of smoking. Although the effect on lung function is associated with the levels of exposure to cigarette smoking and (likely) WPS, the degree of exposure was not measured. It is also possible that that the lack of observed difference between water-pipe smokers and cigarette smokers may be because of inadequate power.

Some authors have hypothesized a less important effect of WPS compared with cigarette smoking on lung function based on a number of assumptions: the inability of smoke to reach the lower airways because of the smoking pattern and because of the filtration of smoke by the water and a better healing of small airway inflammation because of intermittent nature of smoking.[17,19] Our study found no statistically significant difference in FEV_1 and FEV_1/FVC between the two forms of smoking. Indeed, recent evidence has shown that water does not significantly filter out the nicotinic products produced by WPS.[23] Also, water-pipe smokers have an elevation in the level of parameters of oxidation injury and a decreased total antioxidant activity.[24,25] The resulting oxidative stress is believed to play an important role in the pathogenesis of COPD.[1]

CONCLUSIONS

Implications for Public Health Policy

This study adds to the rapidly growing evidence of the association of WPS with deleterious health outcomes,[11] which has very important implications for both clinical and public health practice. Spirometry performance might give the clinician an opportunity to convince smokers to quit.[26] More importantly, our study supplies the physician with data they might use in counseling patients about the deleterious effect of

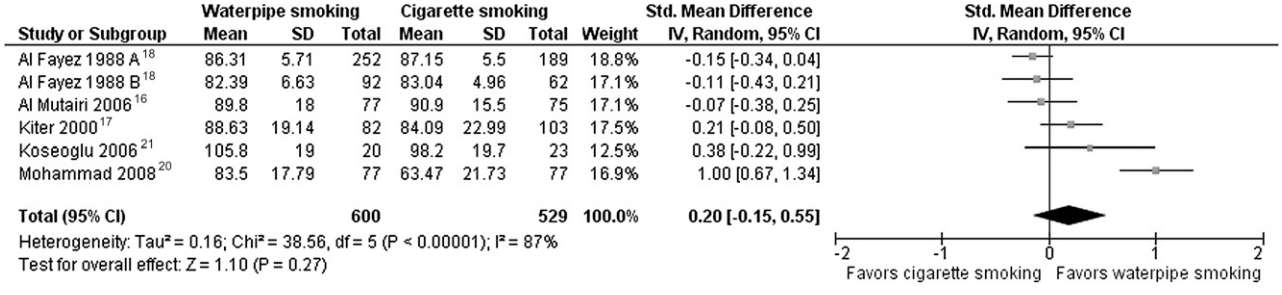

FIGURE 6. Comparison of FEV_1 in water-pipe smokers and cigarette smokers. Al Fayez 1988 A includes only the male participants; Al Fayez 1988 B includes only the female participants. See Figure 3 legend for expansion of abbreviations.

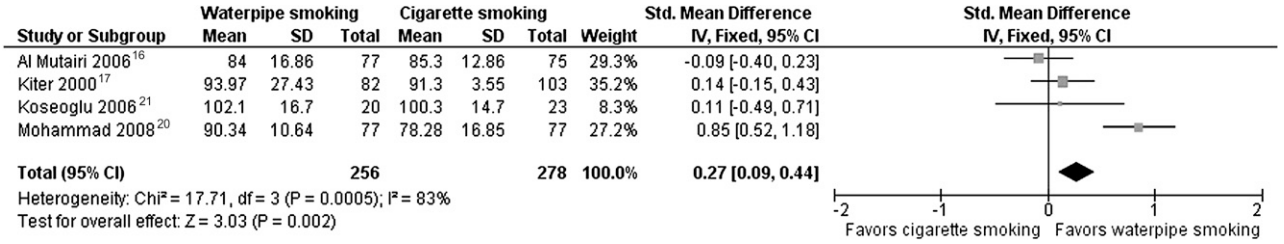

FIGURE 7. Comparison of FVC in water-pipe smokers and cigarette smokers. See Figure 3 legend for expansion of abbreviations.

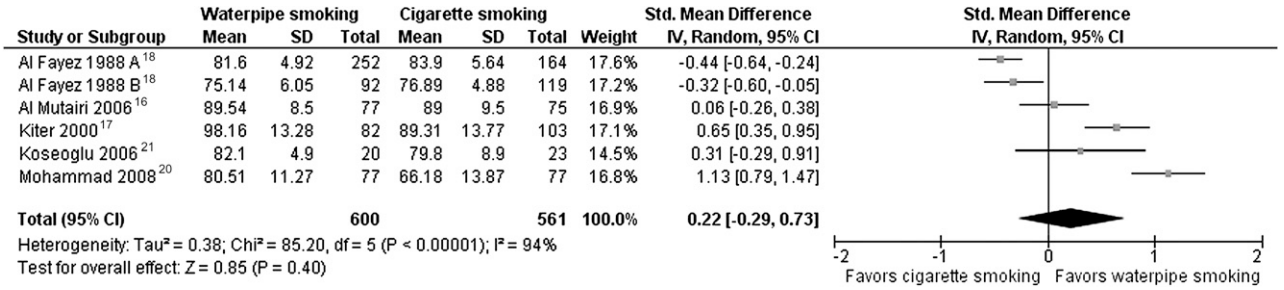

FIGURE 8. Comparison of FEV_1/FVC in water-pipe smokers and cigarette smokers. Al Fayez 1988 A includes only the male participants; Al Fayez 1988 B includes only the female participants. See Figure 3 legend for expansion of abbreviations.

WPS on lung function. As for the public health practice, this study illustrates that WPS may be as harmful as cigarette smoking in terms of lung function. Public health policy makers need to aggressively address the epidemic of WPS to raise awareness and advocate for appropriate policy changes.

Implications for Research

As advised by the World Health Organization, there is a need for more research related to WPS.[27] Specifically, there is a need for higher-quality prospective studies that could more clearly identify the causal relationship between WPS and clinical outcomes. Similarly, there is a need for exploring whether quitting smoking slows down or reverses the deterioration of lung function. Finally, researchers need to focus on standardizing the exposure measurement tools in order to reliably assess for dose-response relationships.[28]

ACKNOWLEDGMENTS

Author contributions: *Dr Raad:* contributed to study selection, data abstraction, data analysis, data interpretation, drafting of the manuscript, revising the article critically for important intellectual content, and approving the final version of this manuscript.
Dr Gaddam: contributed to study data abstraction, revising the article critically for important intellectual content, and approving the final version of this manuscript.
Dr Schunemann: contributed to data interpretation, revising the article critically for important intellectual content, and approving the final version of this manuscript.
Dr Irani: contributed to drafting the protocol and designing the search strategy, revising the article critically for important intellectual content, and approving the final version of this manuscript.
Dr Abou Jaoude: contributed to screening titles, abstracts, and full texts; revising the article critically for important intellectual content; and approving the final version of this manuscript.
Dr Honeine: contributed to screening titles, abstracts, and full texts; revising the article critically for important intellectual content; and approving the final version of this manuscript.
Dr Akl: contributed to drafting the protocol, designing the search strategy, developing the forms, screening, data abstraction, data analysis, data interpretation, drafting of the manuscript, revising the article critically for important intellectual content, and approving the final version of this manuscript.
Financial/nonfinancial disclosures: The authors have reported to *CHEST* that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.
Other contributions: We thank Ann Grifasi for her administrative assistance, Drs Monique Chaaya, Yousser Mohammad, Sana Al Mutairi, and Nalan Koseoglu for their correspondence with results, and Dr Sameer Gunukula for his help in formatting the table.
Additional information: The e-Appendices can be found in the Online Supplement at http://chestjournal.chestpubs.org/content/139/4/764/suppl/DC1.

REFERENCES

1. Global Initiative for Obstructive Lung Disease. 2009. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. Updated 2009. Based on April 1998 NHLBI/WHO workshop. http://www.goldcopd.com/Guidelineitem.asp?l1=2&l2=1&intId=2003. Accessed July 26, 2010.
2. Rabe KF, Hurd S, Anzueto A, et al; Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. *Am J Respir Crit Care Med*. 2007;176(6):532-555.
3. Lopez AD, Shibuya K, Rao C, et al. Chronic obstructive pulmonary disease: current burden and future projections. *Eur Respir J*. 2006;27(2):397-412.
4. World Health Organization. World Health Report. http://www.who.int/whr/2000/en/whr00_annex_en.pdf. Accessed July 26, 2010.

5. Marco M, Minette A. Lung function changes in smokers with normal conventional spirometry. *Am Rev Respir Dis*. 1976; 114(4):723-738.
6. Sherman CB. The health consequences of cigarette smoking. Pulmonary diseases. *Med Clin North Am*. 1992;76(2): 355-375.
7. Milic-Emili J. Measurement of "closing volume" as a simple and sensitive test for early detection of small airway obstruction. *Ann N Y Acad Sci*. 1974;221:115-116.
8. Halbert RJ, Natoli JL, Gano A, Badamgarav E, Buist AS, Mannino DM. Global burden of COPD: systematic review and meta-analysis. *Eur Respir J*. 2006;28(3):523-532.
9. Cobb C, Ward KD, Maziak W, Shihadeh AL, Eissenberg T. Waterpipe tobacco smoking: an emerging health crisis in the United States. *Am J Health Behav*. 2010;34(3):275-285.
10. An emerging deadly trend: waterpipe tobacco use: American Lung Association 2007. http://slati.lungusa.org/reports/Trend%20Alert_Waterpipes.pdf. Accessed July 26, 2010.
11. Akl EA, Gaddam S, Gunukula SK, et al. The effects of waterpipe tobacco smoking on health outcomes: a systematic review. *Int J Epidemiol*. 2010;39(3):834-857.
12. Soriano JB, Visick GT, Muellerova H, Payvandi N, Hansell AL. Patterns of comorbidities in newly diagnosed COPD and asthma in primary care. *Chest*. 2005;128(4):2099-2107.
13. Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. *Am J Respir Crit Care Med*. 1999;159(1):179-187.
14. Higgins JPT, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. *BMJ*. 2003;327(7414): 557-560.
15. Schünemann HJ, Jaeschke R, Cook DJ, et al; ATS Documents Development and Implementation Committee. An official ATS statement: grading the quality of evidence and strength of recommendations in ATS guidelines and recommendations. *Am J Respir Crit Care Med*. 2006;174(5):605-614.
16. Al Mutairi SS, Shihab-Eldeen AA, Mojiminiyi OA, Anwar AA. Comparative analysis of the effects of hubble-bubble (sheesha) and cigarette smoking on respiratory and metabolic parameters in hubble-bubble and cigarette smokers. *Respirology*. 2006;11(4):449-455.
17. Kiter G, Uçan ES, Ceylan E, Kilinç O. Water-pipe smoking and pulmonary functions. *Respir Med*. 2000;94(9):891-894.
18. Al-Fayez SF, Salleh M, Ardawi M, Zahran FM. Effects of sheesha and cigarette smoking on pulmonary function of Saudi males and females. *Trop Geogr Med*. 1988;40(2):115-123.
19. Aydin A, Kiter G, Durak H, Ucan ES, Kaya GC, Ceylan E. Water-pipe smoking effects on pulmonary permeability using technetium-99m DTPA inhalation scintigraphy. *Ann Nucl Med*. 2004;18(4):285-289.
20. Mohammad Y, Kakah M, Mohammad Y. Chronic respiratory effect of narguileh smoking compared with cigarette smoking in women from the East Mediterranean region. *Int J Chron Obstruct Pulmon Dis*. 2008;3(3):405-414.
21. Köseoğlu N, Aydin A, Uçan ES, et al. The effects of waterpipe, cigarette and passive smoking on mucociliary clearance [in Turkish]. *Tuberk Toraks*. 2006;54(3):222-228.
22. Cazzola M, MacNee W, Martinez FJ, et al; American Thoracic Society; European Respiratory Society Task Force on outcomes of COPD. Outcomes for COPD pharmacological trials: from lung function to biomarker. *Eur Respir J*. 2008; 31(2):416-469.
23. Neergaard J, Singh P, Job J, Montgomery S. Waterpipe smoking and nicotine exposure: a review of the current evidence. *Nicotine Tob Res*. 2007;9(10):987-994.
24. Al-Numair K, Barber-Heidal K, At-Assaf A, et al. Water-pipe (shisha) smoking influences total antioxidant capacity and oxidative stress of healthy Saudi males. *Journal of Food, Agriculture, and Environment*. 2007;5(3-4):17-22.
25. Wolfram RM, Chehne F, Oguogho A, Sinzinger H. Narghile (water pipe) smoking influences platelet function and (iso-) eicosanoids. *Life Sci*. 2003;74(1):47-53.
26. Ferguson GT, Enright PL, Buist AS, Higgins MW. Office spirometry for lung health assessment in adults: A consensus statement from the National Lung Health Education Program. *Chest*. 2000;117(4):1146-1161.
27. WHO study group on Tobacco Product Regulation (TobReg). Advisory Note. Waterpipe tobacco smoking: health effects, research needs and recommended actions by regulators. http://www.who.int/tobacco/global_interaction/tobreg/Waterpipe%20recommendation_Final.pdf. Accessed July 26, 2010.
28. Maziak W, Ward KD, Afifi Soweid RA, Eissenberg T. Standardizing questionnaire items for the assessment of waterpipe tobacco use in epidemiological studies. *Public Health*. 2005;119(5):400-404.