

Example EBC II Course Project

Submitted by:
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Step 1

Hypothesis 1:

Does exposure to asbestos and smoking lead to a greater incidence of asbestosis?

Patient- Worker
Intervention- Exposure to asbestos and smoking
Comparison- Including non-smokers and people not exposed to asbestos
Outcome- Higher incidence of asbestosis

Hypothesis 2:

Does occupational exposure to asbestos create respiratory impairment?

Patient- Exposed worker
Intervention- Exposure to asbestos
Comparison- None
Outcomes- Increased respiratory impairment

Step 2

Searched PubMed and CINAHL databases

Abstracts included

Hypothesis 1	PubMed	CINAHL
Asbestos	8623	197
Asbestosis	3694	34
Asbestosis AND smoking	512	7

Hypothesis 2	PubMed	CINAHL
Asbestos AND exposure	4466	105
Asbestos AND(occupational exposure)	1575	83
Asbestos AND respiratory impairment AND (o.e)	13	1

Step 3

Citation of Article Reviewed

Brodkin CA, Bamhart S, Checkoway H, Balmes J, Omenn GS, Rosenstock L.
Longitudinal pattern of reported respiratory symptoms and accelerated ventilatory loss
in asbestos-exposed workers. Chest 1996; 109(1): 120-6

Bullet List

Merits

- Identifies need for prospective study and not a cross sectional study
- Full coverage of information about subject accounting, why they were removed from study
- Adequate number of subjects. Several hundred participants good considering this is a major industrial safety concern
- Clearly lists what outcomes were measured ie: FVC BDR etc...
- How they measured the outcomes was listed ie: spirometry, chest x-rays
- Statistics information on t-test and ANOVA comparison explained

What it Lacks

- Hypothesis is not clearly stated
- Subjects were not assigned randomly. Mostly based on their symptom levels
- Did not explain how they measures subjects to determine which of the 4 groups they were included in Would be tough to reproduce
- Groups were not similar at the start. They all had different levels of exposure to the asbestos

Randomized Clinical Trial Question List found in text book is submitted

Step 4

How the article could be applied to patients?

Respiratory problems are a common occurrence in the waiting room of a medical office. With so many industries still using asbestos in manufacturing, it is not unreasonable to think that some of these cases could be more serious than the seasonal flu. With common symptoms and signs such as wheezing, coughing, phlegm and dyspnea it would not be unreasonable for a doctor to dismiss it as nothing more than the cold.

With the information provided in the article reviewed it is possible to see how a Doctor can change their role and maybe help detect ventilatory loss early. First it is important to note that the authors of this article recognized a need for a longitudinal study versus the cross studies currently available. The current cross sectional research is limited and this sort of undertaking will require years of data collection to make appropriate assumptions. As a doctor you need to change your approach to identify decreased ventilation factors in asbestos exposed workers.

Eliciting a good history and occupation will most be essential. The symptoms of wheezing, and coughing must persist for many years before the flags of suspicion are raised. Find out if your patient works in an asbestos related industry, and begin with a questionnaire like the ATS-DLD (American Thoracic Society Division of Lung Disease) to standardize whether they are asymptomatic, in the developmental stage or the persistent stage. Also take a chest x-ray to identify any abnormalities from the lungs.

From here keep you check-up visits more frequent and observe by questionnaire response how the symptoms you ran a baseline on the first visit are progressing. Studies show that persistence to coughing and phlegm with a development of abnormalities on x-ray are key indicators of future respiratory problems for workers in asbestos related industries. With baseline information on hand you are more likely to be suspicious of that same cough a year later on the patients annual physical.

So how can the doctor help? Well by acting differently and with research at arms length they could lobby against asbestos using industries to change production, as evident by the workers symptom development. For existing patients they may be able to warn against the joining of industry that uses asbestos, and finally for patients already in their practice they will have a new method of differentiating common cold like symptoms from serious ventilatory function loss. With earlier detection, better options may be given to patients to improve and prolong their lives.

Step5

How would I improve efficiency and effectiveness?

If I were to approach this project again I feel there are many things that could have been done to make it more efficient and effective. First I would most likely read in general information sources about the topic in question. Find out if society considers it an epidemic or serious issue to deal with. This would most likely allow me to better focus my hypotheses on what is significant in the topic and most likely find out what the most current and state of the art research is.

Secondly a research effort in the legal action against industry would assist. Asbestosis and other lung related diseases have received a lot of coverage, ever since the connection between asbestos and mesothelioma was made. Law annuls may not have all the scientific data, but they will certainly cover epidemiology and general information about related diseases.

When searching I would not limit my search to indexed journals. I would look in a more detective like fashion, starting with a very general term and through use of Boolean operators narrow the search to a few hundred articles. A quick scan may not reveal desired papers, but looking at where the papers are published may limit my search even more. If this topic covers respiratory impairment then I could limit the search to indexed journals such as Chest and Journal of Respiratory Medicine where those topics are covered in more detail.

Ultimately refining my hypothesis to investigate a focused region of asbestos would have assisted. Perhaps a look at something like development of benign pleural adhesions because of work related asbestos would have helped. Or a topic where I focus on only one respiratory component such as Tidal Volume of Vital Capacity would have produce d a more limited search with results that would have a more direct impact than surveys and early detection of symptoms, which commonly present in many conditions.

Abstracts

Brodkin CA, Barnhart S, Checkoway H, Balmes J, Omenn GS, Rosenstock L. Longitudinal pattern of reported respiratory symptoms and accelerated ventilatory loss in asbestos-exposed workers. *Chest*. 1996 Jan;109(1): 120-6.

Studies investigating the relation between respiratory symptoms and change ventilatory function have been limited by use of reported symptoms at a single point in time To assess the relation between the longitudinal pattern of reported cough, phlegm, wheeze, and dyspnea and ventilatory loss. we prospectively investigated changes in FVC and FEV1 associated with development, resolution, or persistence of these symptoms over a 3- to 5-year period in 446 asbestos-exposed workers. Longitudinally reported symptoms changed frequently, with 52 to 61 of subjects reporting a specific symptom noting resolution or development of that symptom during follow-up. Initially reported symptoms were not predictive of accelerated loss of FVC or FEV1. In contrast development of any new respiratory symptom, and to a lesser extent persistence of symptoms during follow-up, were associated with significantly greater ventilatory losses compared with asymptomatic individuals, ranging from 28 mL/yr in FEV1 for newly developed dyspnea, to 67 mL/yr in FVC for developed wheeze ($p < 0.01$). We conclude that development or persistence of respiratory symptoms over time, rather than the presence of symptoms per se, is predictive of future ventilatory loss Recognition of interval changes in symptom reporting during surveillance of asbestos-exposed workers may effectively identify groups at risk for progressive ventilatory impairment.

Ohar J, Sterling DA, Bleecker E, Donohue J. Changing patterns in asbestos-induced lung disease. *Chest*. 2004 Feb; 125(2); 744-53.

STUDY OBJECTIVES: To determine patterns in asbestos-induced lung diseases found in older, less exposed workers. **DESIGN:** Review of a database evaluating lung function, smoking status, form of asbestos-induced lung disease, and radiograph abnormalities. **SETTING:** Outpatient clinic. **PARTICIPANTS:** A total of 3383 asbestos-exposed workers referred for independent medical evaluation, including control subjects who lacked asbestos-specific radiograph abnormalities ($n = 243$), subjects with low International Labor Organization (ILO) scores ($n = 2,685$), high ILO scores ($n = 312$), bronchogenic cancer ($n = 63$), and mesothelioma ($n = 80$). Of these, 3,327 workers have specific smoking status information and 3,312 workers have lung volume measures. **INTERVENTIONS:** Chest radiographs were interpreted by a certified B-reader, and abnormalities were quantified according to the ILO scoring system. Spirometry and lung volume measurement were performed. Subjects completed a self-administered questionnaire that was reviewed at the time of examination. Control subjects were screened on two separate occasions at least 10 years apart to exclude

subclinical or slowly progressive asbestos-induced lung disease

MEASUREMENTS AND RESULTS: The mean age of the population was 65.1 +/- 9.9 years, and the latency was 41.4 +/- 10.1 years (+/- SD). Most subjects (41.8) had normal pulmonary function. Obstruction was the most common pulmonary function abnormality (25.4), followed by restriction (19.3) and a mixed pattern (6.0). Most subjects (79.4) had low ILO scores. Benign pleural abnormalities were the only findings in 54 of subjects with low ILO score. Subjects with high ILO scores were older, smoked more and had a longer latency than subjects with low ILO scores and control subjects. Smokers were younger, had a shorter latency, and had paradoxically greater ELO scores than nonsmokers. Subjects with bronchogenic cancer and mesothelioma had longer latencies than control subjects and subjects with benign asbestos-induced lung disease. **CONCLUSIONS:** Asbestos-induced lung disease today is characterized by low ILO scores, long latencies, greater disease magnitude in smokers, and a normal or obstructive pattern of pulmonary function abnormality. Spirometric evaluation in the absence of lung volume measurements caused misclassification that resulted in overestimation of the presence of a restrictive pattern of pulmonary function.