Asbestos-related diseases in automobile mechanics.
Jacques Ameille, Nicole Rosenberg, Mireille Matrat, Alexis Descatha, Dominique Mompoint, Lounis Hamzi, Catherine Atassi, Manuela Vasile, Robert Garnier, Jean-Claude Pairon

To cite this version:
Asbestos-related diseases in automobile mechanics

Jacques Ameille, MD¹, Nicole Rosenberg, MD², Mireille Matrat, MD³,⁴, Alexis Descatha, PhD¹, Dominique Mompoint, MD⁵, Lounis Hamzi, MD⁶, Catherine Atassi, MD⁷, Manuela Vasile, MD⁷, Robert Garnier, MD², Jean-Claude Pairon, PhD³,⁴.

¹ AP-HP, Unité de pathologie professionnelle, Hôpital Raymond Poincaré, Garches, France
² AP-HP, Consultation de pathologie professionnelle, Hôpital Fernand Widal, Paris, France
³ Centre Hospitalier Intercommunal, Unité de pathologie professionnelle, Créteil, France
⁴ INSERM, Unité 955, Créteil, France
⁵ AP-HP ; Service de radiologie, Hôpital Raymond Poincaré, Garches, France
⁶ AP-HP, Service de radiologie, Hôpital Fernand Widal, Paris, France
⁷ Centre Hospitalier Intercommunal, Service de radiologie, Créteil, France

Running head
Asbestos-related diseases in automobile mechanics

Corresponding author
Jacques Ameille, MD, Unité de pathologie professionnelle, Hôpital Raymond Poincaré. 104 Boulevard Raymond Poincaré, 92380 Garches, France.
E.mail: jacques.ameille@rpc.aphp.fr
Fax number: 33147107768
Phone number: 33147107754

Word count (text): 2210

Key words
Asbestos, pleural plaques, automobile mechanics, HRCT
Abstract (231 words)

Purpose
Automobile mechanics have been exposed to asbestos in the past, mainly due to the presence of chrysotile asbestos in brakes and clutches. Despite the large number of automobile mechanics, little is known about the non-malignant respiratory diseases observed in this population. The aim of this retrospective multicenter study was to analyze the frequency of pleural and parenchymal abnormalities on HRCT in a population of automobile mechanics.

Methods
The study population consisted of 103 automobile mechanics with no other source of occupational exposure to asbestos, referred to three occupational health departments in the Paris area for systematic screening of asbestos–related diseases. All subjects were examined by HRCT and all images were reviewed separately by two independent readers, with further consensus in the case of disagreement. Multiple logistic regression models were constructed to investigate factors associated with pleural plaques.

Results
Pleural plaques were observed in 5 cases (4.9%) and interstitial abnormalities consistent with asbestosis were observed in 1 case. After adjustment for age, smoking status, and a history of non-asbestos-related respiratory diseases, multiple logistic regression models showed a significant association between the duration of exposure to asbestos and pleural plaques.

Conclusions
The asbestos exposure experienced by automobile mechanics may lead to pleural plaques. The low prevalence of non-malignant asbestos-related diseases, using a very sensitive diagnostic tool, is in favor of a low cumulative exposure to asbestos in this population of workers.
**Introduction**

The presence in the past of chrysotile asbestos in automobile brakes or clutches has given rise to concerns about increased risks of asbestos-related diseases among automobile mechanics (Lemen 2004, Paustenbach et al. 2004, Welsh 2007). A large number of automobile mechanics have been potentially exposed to asbestos. According to the data of the 1999 census, the estimated population of automobile mechanics, aged 16 to 60 years, in France was 242,360 (Imbernon et al. 2005)).


Cases of malignant pleural mesothelioma have been reported in automobile mechanics (Paur et al. 1985, Butnor et al. 2003, Rodelsperger et al. 1986). However, published meta-analysis of the epidemiological literature that examined the risk of pleural mesothelioma (Wong 2001, Goodman 2004) or lung cancer (Goodman 2004) did not show any elevated meta-relative risks.

Very few data concerning non-malignant asbestos-related diseases or impairment of lung function in automobile mechanics have been published (Plato et al. 1995, Marcus et al. 1987, Boilat and Lob 1973, Dahlqvist et al. 1992). Only one study, using full-size chest radiographs, investigated the prevalence of pleural plaques (Marcus et al. 1987). To our knowledge, no study using chest CT-scan for the detection of asbestos-related diseases in automobile mechanics, has been published.

The aim of the present retrospective multicenter study was to analyze the frequency of pleural and parenchymal abnormalities on chest High Resolution Computed Tomography (HRCT), in a population of automobile mechanics, referred by occupational physicians to occupational health departments in the Paris area, for systematic screening of asbestos-related diseases.
Study population and methods

Study population

The eligible population consisted of all automobile mechanics, referred to occupational health departments in university hospitals of Garches, Créteil, and Paris–Fernand Widal, between 1st January 2002 and 30th June 2009, for HRCT detection of asbestos-related diseases. These subjects were systematically referred by occupational physicians because of known exposure to asbestos. As social welfare benefits, such as early retirement allowance or pension, are provided in France for workers with asbestos-related diseases, including pleural plaques, when confirmed by chest CT-scan examinations, a consensus conference, held in Paris in January 1999, recommended the use of chest CT-scan, after a sufficient latency period, for the surveillance of workers occupationally exposed to asbestos (Conférence de consensus, 1999). Subjects with sources of occupational exposure to asbestos other than their work as automobile mechanics were excluded from the analysis.

Methods

For each subject, information was collected on gender, age, previous chest diseases, and smoking status. Subjects were classified into three categories according to tobacco consumption: smokers, ex-smokers and non-smokers. Ex-smokers were defined as those who had quit smoking for at least one year.

For each subject, the year of starting work as an automobile mechanic was recorded, together with the type of work (light vehicles, trucks, mixed activity), duration of exposure, and the time interval between start of asbestos exposure and the HRCT (latency). For calculation of the duration of exposure to asbestos, only work prior to 1997 (year in which asbestos was banned in France), was taken into account. Information on the use of compressed air during brake cleaning was also collected.

HRCT

Spiral HRCT acquisition parameters were defined in accordance with the French Thoracic Imaging Society guidelines: full inspiration, slice thickness of 1.5 to 5 mm, pitch of 1.5 to 2.0, 120 KV, 60 to 150 mAs. All CT scans were performed without intravenous contrast agent.

Localized acquisition in the prone position was required in the case of suspected gravitational lung abnormalities.
All images were reviewed separately by two independent readers in each occupational health department (a radiologist and an occupational diseases specialist), with further consensus reading in the case of disagreement between the two readers concerning the presence of pleural or parenchymal abnormalities consistent with an asbestos-related disease.

A structured form, elaborated from a previous study (Ameille et al. 2007), was filled out, describing the features of pulmonary abnormalities consistent with asbestosis (thickened interstitial short lines: septal lines, non-septal lines, honeycombing, curvilinear subpleural lines, ground glass opacities), and pleural abnormalities. When associated with rounded atelectasis or parenchymal bands, pleural thickenings were considered to correspond to diffuse pleural thickening (fibrosis of visceral pleura) (Gevenois et al. 1998, Beigelman-Aubry et al. 2007). Pleural plaques were considered to be present in the case of circumscribed quadrangular pleural elevations, with sharp borders and tissue density, sometimes calcified, or in the case of multiple, bilateral, less typical images, with a typical topography (Beigelman-Aubry et al. 2007). Pleural abnormalities not meeting the criteria for pleural plaques or diffuse pleural thickening were considered to be nonspecific pleural thickening.

**Statistical analysis**

Values for continuous variables were expressed as the mean (standard deviation, SD). Bivariate analysis was performed to study factors associated with pleural plaques, using Fisher’s exact test for qualitative variables, or Wilcoxon’s test for continuous variables. A p-value less than 0.05 was considered statistically significant. Multiple logistic regression models were constructed, including age, previous respiratory diseases, smoking status, latency, and duration of exposure to asbestos. Due to the strong collinearity between latency and duration of exposure, separate models were constructed. A p-value less than 0.05 was considered statistically significant.

Statistical analysis was performed using SAS software version 9.1 (SAS Institute Inc, Cary, NC, USA).

Informed consent was obtained from all subjects. The study was approved by the French national committee for data protection (Commission Nationale de l’Informatique et des Libertés: CNIL).
Independent ethics committee approval was not necessary, as chest CT scans are recommended in France for screening purposes in asbestos-exposed workers (Conférence de consensus, 1999).

**Results**

During the study period, 242 automobile mechanics were referred to the occupational health departments participating in the study. Eighty-nine automobile mechanics with other possible sources of asbestos exposure, and 50 automobile mechanics with no available HRCT were excluded. The study population therefore consisted of 103 subjects.

General characteristics of the study population are given in Table 1. All subjects were male with a mean age of 54.4 (7.2) years; 69 (68%) were smokers or ex-smokers. Mean latency was 36.8 (7.0) years (range: 19-57 years). Mean duration of asbestos exposure was 22.6 (8.1) years (range: 4-45 years). Most of the study population (83.5%) used compressed air for brake cleaning.

Interstitial abnormalities consistent with asbestosis were observed in one subject. This subject had worked for 24 years as an automobile mechanic, ensuring brake repairs on trucks for more than 8 hours a week. Interstitial abnormalities consisted of bilateral honeycombing, ground-glass opacities and non-septal lines in the postero-inferior zones of the lungs. Five automobile mechanics had pleural plaques, 4 had diffuse pleural thickening, and 9 had nonspecific pleural thickening. No case of lung cancer or pleural mesothelioma was detected. Pleural plaques were diagnosed in each occupational health department participating in the study (one in two departments, two in one department). The 5 subjects with pleural plaques had no previous chest diseases likely to explain pleural thickening.

Characteristics of the subjects according to HRCT abnormalities are presented in Table 2. Subjects with nonspecific pleural thickening had similar asbestos exposure to that of subjects without HRCT abnormalities, in terms of latency or duration of exposure. Subjects with pleural plaques and subjects with diffuse pleural thickening had longer latency and duration of asbestos exposure than subjects without HRCT abnormalities (Table 2).

In comparison with subjects without pleural plaques, subjects with pleural plaques had significantly longer latency and duration of asbestos exposure (Table 3).
After adjustment for age, smoking status, and history of previous asbestos-unrelated respiratory diseases, multiple logistic regression models showed a statistically significant association between the duration of asbestos exposure and the presence of pleural plaques (Table 4).

Discussion
In this population of automobile mechanics referred for systematic detection of asbestos-related diseases by HRCT, only one case of interstitial abnormalities consistent with asbestosis, 5 cases of pleural plaques (4.9%) and 4 cases (3.9%) of diffuse pleural thickening were observed. The 5 subjects with pleural plaques had no known history of chest disease likely to explain pleural abnormalities. The prevalence of pleural plaques in this study is similar to that observed in the only previous published study that investigated the frequency of pleural plaques in car mechanics (Marcus et al. 1987). In that study, based on chest radiographs, pleural plaques, defined by pleural thickening at least 5 mm thick on the chest wall or 3 mm thick on the diaphragm, were observed in 41 out of 925 car mechanics (4.4%). However, our results must be compared cautiously with those reported by Marcus et al. due to selection factors and the use of different tools for the radiological detection of asbestos-related abnormalities.

The prevalence of pleural plaques observed in this study was similar to that observed on HRCT examinations in subjects exposed to indoor asbestos pollution (De Raeve et al. 2001) or in urban transportation workers with low cumulative exposure to asbestos (Ameille et al. 2007).

The main limitations of this study are the relatively small number of subjects, the absence of an unexposed control group, and the lack of objective assessment of the level of occupational exposure to asbestos. The representativity of the study sample is also questionable. However, inclusion criteria had nothing to do with symptoms or abnormalities on chest radiographs.

One of the strengths of this study is the use of HRCT for detection of asbestos-related diseases. HRCT has been shown to be much more sensitive and specific than chest radiographs for the detection of early pleural and parenchymal changes.

Another strength is the standardized reading, with systematic double interpretation followed by consensus reading in the case of disagreement. Furthermore, only automobile mechanics with no other known occupational exposure to asbestos and with a sufficiently long latency (> 20 years in 99% of cases, > 30 years in 89% of cases) to allow evaluation of asbestos-related diseases, especially pleural plaques, were included in the study (Schwartz 1991, Paris et al. 2009).

In the absence of a control group, demonstration of a significant relationship between pleural plaques and duration of exposure (used as a surrogate of cumulative exposure to asbestos) is an argument in favor of the causal role of asbestos (Paris et al. 2009).

The low prevalence of pleural plaques and interstitial abnormalities in this study is an indirect argument in favor of low cumulative exposure to asbestos for most automobile mechanics. These data are consistent with the meta-analysis of epidemiologic studies that investigated the incidence of lung cancer and pleural mesothelioma in automobile mechanics and failed to demonstrate increased meta-relative risks (Wong 2001, Goodman 2004). Our data are also consistent with the results of a recently published population-based case-control study (Rolland et al. 2010) that reported a slight but not significant increased risk of pleural mesothelioma in male car mechanics (OR=1.50; 95% CI: 0.76 - 2.95).

Many measurements in car repair workshops have shown that occupational exposure of car mechanics to asbestos is generally fairly low. Rohl et al (1976) reported airborne chrysotile concentrations of up to 30 f/ml. However, these were peak exposure concentrations measured during brake cleaning with the use of compressed air. According to Paustenbach et al (2003), analysis of the available published data indicates that 90% of 8 hours time weighted average (TWA) concentrations, experienced by mechanics servicing brakes in the 1970s, was less than 0.1 f/ml (mean 0.04 f/ml). In Sweden, Plato et al (1995), using a model based on data from the international literature and quantitative asbestos measurements performed in 1976-1978 in Swedish car repair workshops, estimated the mean cumulative exposure to be 2.6 f/ml x years, in a group of 103 car and bus mechanics,
with more than 15 years of asbestos exposure. Car mechanics had a TWA exposure range of 0.11-0.41 f/ml (mean 0.21 f/ml) in 1965, and 0.003-0.08 f/ml in 1985 (mean 0.021 f/ml). In Germany, based on dust measurements in 76 service stations, and the occupational histories of 210 vehicle mechanics with an average duration of employment of 21±10 years, a mean cumulative exposure to asbestos was estimated to be 0.54 ±1.1 f/ml x years for car mechanics (Rodelsperger et al. 1986). Finley et al (2007) recently developed estimates of cumulative chrysotile exposures experienced by automobile mechanics working with brakes and manual clutches. They reported that the 95th percentile and 99th percentile cumulative exposures for vehicles mechanics in the 1970s, were 2.0 and 5.7 f/ml years, respectively. Another explanation for the low prevalence of asbestos-related diseases in automobile mechanics might be that decomposition of asbestos occurs during normal use of brakes due to thermal decomposition into forsterite, a non-fibrous material (Lemen 2004).

Conclusion
The asbestos exposure experienced by French car mechanics may lead to pleural plaques. The low prevalence of non-malignant asbestos-related diseases, despite the use of HRCT which is a very sensitive diagnostic tool, is in favor of low cumulative exposure to asbestos in the study population.

Acknowledgments
This work was supported by a grant from the Caisse nationale d’assurance maladie des travailleurs salariés (Département des risques professionnels), and the Caisse régionale d’assurance maladie d’Ile de France.

The authors declare that they have no conflict of interest
References


Aberle DR, Gamsu G, Ray CS, Feuerstein IM (1988, b) Asbestos-related pleural and parenchymal fibrosis: detection with high-resolution CT. Radiology; 166: 729-734


Boillat MA, Lob M (1973) [Risk of asbestosis in workers employed in replacing automobile brake linings]. Schweiz Med Wschr; 103: 1354-1359


Neri S, Antonelli A, Falashi F, Boraschi P, Baschieri L (1994) Findings from high-resolution computed tomography of the lung and pleura of symptom free workers exposed to amosite who had normal chest radiographs and pulmonary function tests. Occup Environ Med; 51: 239-243


Table 1. Characteristics of the study population (n=103)

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>%</th>
<th>Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gender: male</strong></td>
<td>103</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td></td>
<td></td>
<td>54.4 (7.2)</td>
</tr>
<tr>
<td>&lt; 50</td>
<td>23</td>
<td>22.3</td>
<td></td>
</tr>
<tr>
<td>50-59</td>
<td>62</td>
<td>60.2</td>
<td></td>
</tr>
<tr>
<td>&gt; 60</td>
<td>18</td>
<td>17.5</td>
<td></td>
</tr>
<tr>
<td><strong>Tobacco consumption</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-smokers</td>
<td>32</td>
<td>31.7</td>
<td></td>
</tr>
<tr>
<td>Ex-smokers</td>
<td>41</td>
<td>40.6</td>
<td></td>
</tr>
<tr>
<td>Smokers</td>
<td>28</td>
<td>27.7</td>
<td></td>
</tr>
<tr>
<td><strong>Latency(^1) (years)</strong></td>
<td></td>
<td></td>
<td>36.8 (7.0)</td>
</tr>
<tr>
<td>&lt; 20</td>
<td>1</td>
<td>1.0</td>
<td></td>
</tr>
<tr>
<td>20-30</td>
<td>10</td>
<td>9.7</td>
<td></td>
</tr>
<tr>
<td>&gt; 30</td>
<td>92</td>
<td>89.3</td>
<td></td>
</tr>
<tr>
<td><strong>Duration of exposure(^2) (years)</strong></td>
<td></td>
<td></td>
<td>22.3 (8.1)</td>
</tr>
<tr>
<td>&lt; 20</td>
<td>31</td>
<td>30.1</td>
<td></td>
</tr>
<tr>
<td>20-30</td>
<td>50</td>
<td>48.5</td>
<td></td>
</tr>
<tr>
<td>&gt;30</td>
<td>22</td>
<td>21.4</td>
<td></td>
</tr>
<tr>
<td><strong>Brake repair of trucks</strong></td>
<td>43</td>
<td>41.8</td>
<td></td>
</tr>
<tr>
<td><strong>Use of compressed air</strong></td>
<td>86</td>
<td>83.5</td>
<td></td>
</tr>
</tbody>
</table>

\(^1\) Time interval between start of asbestos exposure and HRCT

\(^2\) Duration of asbestos exposure before 1997
Table 2. Characteristics of the subjects according to HRCT abnormalities

<table>
<thead>
<tr>
<th></th>
<th>No abnormalities n = 84</th>
<th>Nonspecific pleural thickening n = 9</th>
<th>Pleural plaques n = 5</th>
<th>Diffuse pleural thickening n = 4</th>
<th>Asbestosis n = 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age: m in years (SD)</td>
<td>53.9 (7.4)</td>
<td>54.1 (4.0)</td>
<td>59.0 (6.9)</td>
<td>59.5 (7.1)</td>
<td>63</td>
</tr>
<tr>
<td>Latency²: m in years (SD)</td>
<td>36.2 (7.1)</td>
<td>36.0 (4.8)</td>
<td>42.2 (5.6)</td>
<td>42.5 (5.9)</td>
<td>47</td>
</tr>
<tr>
<td>Duration of exposure³: m in years (SD)</td>
<td>21.8 (8.0)</td>
<td>23.1 (5.8)</td>
<td>31.0 (3.9)</td>
<td>27.8 (12.9)</td>
<td>24</td>
</tr>
<tr>
<td>Brake repair of trucks: n (%)</td>
<td>34 (40.5)</td>
<td>5 (55.6)</td>
<td>3 (60.0)</td>
<td>0 (0)</td>
<td>1 (100)</td>
</tr>
<tr>
<td>Use of compressed air: n (%)</td>
<td>68 (81.0)</td>
<td>8 (88.9)</td>
<td>5 (10.0)</td>
<td>4 (100)</td>
<td>1 (100)</td>
</tr>
<tr>
<td>Previous respiratory diseases: n (%)</td>
<td>10 (11.9)</td>
<td>3 (33.3)</td>
<td>1 (20.0)</td>
<td>1 (25.0)</td>
<td>0</td>
</tr>
</tbody>
</table>

¹ Interstitial abnormalities consistent with asbestosis

² Time interval between start of asbestos exposure and HRCT

³ Duration of asbestos exposure before 1997
Table 3. Characteristics of the subjects according to the presence of pleural plaques

<table>
<thead>
<tr>
<th>Pleural plaques</th>
<th>Yes: n = 5</th>
<th>No: n = 98</th>
<th>p^1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age: m (SD)</td>
<td>59.0 (6.9)</td>
<td>54.2 (7.1)</td>
<td>0.12</td>
</tr>
<tr>
<td>Latency^2: m (SD)</td>
<td>42.2 (5.6)</td>
<td>36.6 (7.0)</td>
<td>0.047</td>
</tr>
<tr>
<td>Duration of exposure^3: m (SD)</td>
<td>31.0 (3.9)</td>
<td>22.2 (8.0)</td>
<td>0.01</td>
</tr>
<tr>
<td>Brake repair of trucks: n (%)</td>
<td>3 (60.0)</td>
<td>40 (40.6)</td>
<td></td>
</tr>
<tr>
<td>Use of compressed air: n (%)</td>
<td>5 (100.0)</td>
<td>81 (81.8)</td>
<td></td>
</tr>
</tbody>
</table>

^1 Wilcoxon test
^2 Time interval between start of asbestos exposure and HRCT
^3 Duration of asbestos exposure before 1997
Table 4. Pleural plaques: study of determinants by multivariate logistic regression analysis

<table>
<thead>
<tr>
<th></th>
<th>aOR(^1) [95% CI]</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>1.0 [0.8-1.2]</td>
<td>NS(^2)</td>
</tr>
<tr>
<td>Previous respiratory diseases (yes vs no)</td>
<td>1.2 [0.1-13.7]</td>
<td>NS</td>
</tr>
<tr>
<td>Smokers or ex-smokers (yes vs no)</td>
<td>2.0 [0.2-20.7]</td>
<td>NS</td>
</tr>
<tr>
<td>Latency(^3) (years)</td>
<td>1.1 [0.9-1.4]</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Model 2</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>1.0 [0.9-1.2]</td>
<td>NS</td>
</tr>
<tr>
<td>Previous respiratory diseases (yes vs no)</td>
<td>1.1 [0.1-17.8]</td>
<td>NS</td>
</tr>
<tr>
<td>Smokers or ex-smokers (yes vs no)</td>
<td>3.7 [0.3-46.3]</td>
<td>NS</td>
</tr>
<tr>
<td>Duration of exposure(^4) (years)</td>
<td>1.2 [1.0-1.4]</td>
<td>0.048</td>
</tr>
</tbody>
</table>

\(^1\)Adjusted odds ratio

\(^2\)NS

\(^3\)Latency

\(^4\)Duration of exposure