All-cause and cause specific mortality in a cohort of 20 000 construction workers; results from a 10 year follow up

V Arndt, D Rothenbacher, U Daniel, B Zschenderlein, S Schuberth, H Brenner

Background: Construction workers are potentially exposed to many health hazards, including human carcinogens such as asbestos, silica, and other so-called “bystander” exposures from shared work places. The construction industry is also a high risk trade with respect to accidents.

Methods: A total of 19 943 male employees from the German construction industry who underwent occupational health examinations between 1986 and 1992 were followed up until 1999/2000. Results: A total of 818 deaths occurred during the 10 year follow up (SMR 0.71; 95% CI 0.66 to 0.76). Among those were 299 deaths due to cancer (SMR 0.89; 95% CI 0.79 to 1.00) and 312 deaths due to cardiovascular diseases (SMR 0.59; 95% CI 0.51 to 0.68). Increased risk of mortality was found for non-transport accidents (SMR 1.61; 95% CI 1.15 to 2.27), especially due to falls (SMR 1.87; 95% CI 1.18 to 2.92) and being struck by falling objects (SMR 1.90; 95% CI 0.88 to 3.64). Excess mortality due to non-transport accidents was highest among labourers and young and middle-aged workers. Risk of getting killed by falling objects was especially high for foreign workers (SMR 4.28; 95% CI 1.17 to 11.01) and labourers (SMR 6.01; 95% CI 1.63 to 15.29).

Conclusion: Fatal injuries due to falls and being struck by falling objects pose particular health hazards among construction workers. Further efforts are necessary to reduce the number of fatal accidents and should address young and middle-aged, semi-skilled and foreign workers, in particular. The lower than expected cancer mortality deserves careful interpretation and further follow up of the cohort.

Although the number of construction workers has declined in recent years, the construction industry is still one of the largest industries in Germany with approximately 2 million workers employed.\textsuperscript{1} The structural characteristics of the construction industry with large numbers of relatively small companies, multi-employer work sites, and a highly mobile workforce\textsuperscript{1'} pose challenges in the field of occupational safety and occupational health surveillance.

Construction workers are potentially exposed to asbestos, silica, other dusts, organic solvents, and other chemicals, but also to noise and vibrations\textsuperscript{2} and to “bystander exposures”\textsuperscript{2} present in shared work spaces.\textsuperscript{3} In addition, the construction industry is also a high risk trade with respect to accidents, and the construction industry accounts for the largest numbers of fatal occupational injuries in many countries.\textsuperscript{4,5}

Although there is some epidemiological literature in this field, the relations between mortality among construction workers and specific hazards are not well understood.\textsuperscript{6} Several proportionate mortality ratio (PMR) studies indicate an increase in risk of cancer mortality among construction workers.\textsuperscript{7,8,9,10} For example, increased site specific risks of cancer associated with skilled construction trades have been reported for bone cancer, kidney cancer, nasal cancer, scrotal cancer, lung cancer, rectal cancer, and acute myeloid leukaemia.\textsuperscript{11,12} In contrast, standardised mortality ratio (SMR) studies comparing construction workers with the general population have reported a decreased risk of cancer\textsuperscript{11,12} and all-cause mortality.\textsuperscript{5,13-16} Some of the discrepancy between SMR and PMR studies is likely to be due to a healthy worker effect, but differences in exposure or background cancer risk might also be discussed.

In this context, more detailed analyses regarding work related and socioeconomic factors, which have not been included in most pertinent studies, would be of major interest. Such data have been collected in a cohort study initiated by our group a number of years ago. In a previous report from this study,\textsuperscript{13} we examined mortality and morbidity among 5000 construction workers over a five year period; we found a significant increase in risk of permanent work incapacity and a tendency towards an increased risk of all-cause mortality for construction workers in comparison to a white collar control group. During the past few years, we were able to enlarge the cohort up to 20 000 workers and to expand the follow up period to 10 years. In this paper, we present the results on all-cause and cause specific SMR analyses with respect to work related (job title, years in construction industry) and socioeconomic (age, nationality) factors.

MATERIAL AND METHODS

Study population

The baseline study population comprised all construction workers, aged 25–64 years who were working in one of six job groups (plumbers, carpenters, painters, plasterers, bricklayers, and unskilled workers/labourers) and who participated in a health examination by the Institution for Statutory Accident Insurance and Prevention in the building trade in Baden-Württemberg (a state with about 10 million people in the south of Germany) between 1 August 1986 and 31 December 1992. As over 95% of all construction workers in Germany are men,\textsuperscript{14} our study is limited to male workers. The baseline health examination is part of the routine occupational health surveillance and includes occupational and medical history, a physical examination, pulmonary function test, test of visual acuity, audiometry, and blood and serum analysis. The examination is based on legislation on health and safety at work in Germany (“Arbeitssicherheitgesetz”) and is offered to all construction workers. Participation is initiated by our group a number of years ago. In a previous report from this study,\textsuperscript{13} we examined mortality and morbidity among 5000 construction workers over a five year period; we found a significant increase in risk of permanent work incapacity and a tendency towards an increased risk of all-cause mortality for construction workers in comparison to a white collar control group. During the past few years, we were able to enlarge the cohort up to 20 000 workers and to expand the follow up period to 10 years. In this paper, we present the results on all-cause and cause specific SMR analyses with respect to work related (job title, years in construction industry) and socioeconomic (age, nationality) factors.

Abbreviations: PMR, proportionate mortality ratio; SMR, standardised mortality ratio
Main messages

- All-cause mortality of construction workers was lower than unity compared with the general population (SMR 0.71).
- Excess mortality was observed for pneumoconiosis (SMR 2.30) and for non-transport accidents such as falls (SMR 1.58) and being struck by falling objects (SMR 1.90).
- Relative risk of fatal non-transport accidents was highest among young and middle-aged workers, workers of foreign nationality, and labourers.

Policy implications

- The construction industry is a high risk trade with respect to non-transport accidents.
- Variations in fatal injury rate across different age, occupational, and ethnic groups indicate the potential for improving occupational safety and health.
- Further efforts to reduce the number of fatal accidents are necessary and should in particular include younger, semi-skilled, and foreign workers.

Follow up

In the absence of a national death index in Germany, residents' registration offices were contacted to obtain most recent information on vital status of all cohort members between October 1998 and February 2000. If necessary (for example, in the case of study participants who have moved within Germany during the follow up), subsequent residents' registration offices were contacted until the latest information on vital status was obtained. Workers who moved to a foreign country (n = 719, 3.6%) or with unknown place of residence (n = 159, 0.8%) were censored with date of last known residence. No follow up information was available for 116 members of the baseline population (0.6%), yielding a final study population of 19 827 men. In case of deceased study participants, death certificates were obtained from the regional public health departments, and cause of death was abstracted from each death certificate by two trained physicians (VA, UD) and classified according to the 9th revision of the International Classification of Diseases (ICD-9). Discrepant indexing was resolved by a professional nosologist.

Statistical methods

Standardised mortality ratios (SMR) for all-cause and cause specific mortality were calculated with the SAS statistical software package using age (25–29, 30–34… 70–79), sex, and calendar year specific mortality data from the Baden-Württemberg general population (comprising citizens of German and non-German nationality) as reference. Exact 95% confidence limits were calculated with the SISA software. In case of >15 observed deaths, the Poisson process approximation was employed.


Information on cause of death could be obtained for 84.8% of all deceased. Missing death certificates arise from the fact that death certificates have to be stored at the regional state’s health offices for five years only in Baden-Württemberg and may be destroyed thereafter. Since this missing information on cause specific deaths affects only the cause specific mortality of our cohort members and not the official mortality rates from the general population, we employed the method described by Rittgen and Becker to adjust for missing death certificates. Under the (quite plausible) assumption that the availability of the cause of death certificate is not related to the exposure under consideration

<table>
<thead>
<tr>
<th>Occupation</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plumbers</td>
<td>2843</td>
<td>14.3</td>
</tr>
<tr>
<td>Carpenters</td>
<td>2667</td>
<td>13.5</td>
</tr>
<tr>
<td>Painters</td>
<td>2999</td>
<td>15.1</td>
</tr>
<tr>
<td>Plasterers</td>
<td>2040</td>
<td>10.3</td>
</tr>
<tr>
<td>Bricklayers</td>
<td>6330</td>
<td>31.9</td>
</tr>
<tr>
<td>Labourers</td>
<td>2948</td>
<td>14.9</td>
</tr>
</tbody>
</table>

Table 1 Characteristics of the study population at baseline examination
Table 2  SMR of all-cause and cause specific mortality within total cohort

<table>
<thead>
<tr>
<th>Cause of death (ICD-9)</th>
<th>Observed</th>
<th>Expected*</th>
<th>SMR (95% CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer (ICD 140–208)</td>
<td>299</td>
<td>335</td>
<td>0.89 (0.79 to 1.00)</td>
</tr>
<tr>
<td>Cancer of the oral cavity and pharynx (ICD 140–149)</td>
<td>23</td>
<td>26.8</td>
<td>0.86 (0.54 to 1.29)</td>
</tr>
<tr>
<td>Cancer of the digestive system (ICD 150–159)</td>
<td>99</td>
<td>112</td>
<td>0.89 (0.72 to 1.08)</td>
</tr>
<tr>
<td>Cancer of the respiratory system (ICD 160–165)</td>
<td>100</td>
<td>99</td>
<td>1.01 (0.82 to 1.22)</td>
</tr>
<tr>
<td>Circulatory system (ICD 390–459)</td>
<td>185</td>
<td>312</td>
<td>0.59 (0.51 to 0.68)</td>
</tr>
<tr>
<td>Ischaemic heart disease (ICD 410–414)</td>
<td>109</td>
<td>178</td>
<td>0.61 (0.50 to 0.74)</td>
</tr>
<tr>
<td>Heart failure, other heart disease (ICD 420–429)</td>
<td>29</td>
<td>52</td>
<td>0.55 (0.37 to 0.80)</td>
</tr>
<tr>
<td>Cerebrovascular disease (ICD 430–438)</td>
<td>22</td>
<td>49</td>
<td>0.45 (0.28 to 0.68)</td>
</tr>
<tr>
<td>Diseases of arteries, arterioles, and capillaries (ICD 440–448)</td>
<td>10</td>
<td>11</td>
<td>0.88 (0.44 to 1.67)</td>
</tr>
<tr>
<td>Respiratory system (ICD 460–519)</td>
<td>23</td>
<td>38</td>
<td>0.60 (0.38 to 0.90)</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease and allied conditions (ICD 490–496)</td>
<td>14</td>
<td>25</td>
<td>0.55 (0.31 to 0.94)</td>
</tr>
<tr>
<td>Pneumoconiosis and other lung diseases related to external agents (ICD 500–508)</td>
<td>3</td>
<td>1.3</td>
<td>2.30 (0.48 to 6.74)</td>
</tr>
<tr>
<td>Digestive system (ICD 520–579)</td>
<td>63</td>
<td>90</td>
<td>0.70 (0.54 to 0.90)</td>
</tr>
<tr>
<td>Injury and poisoning (ICD 800–999)</td>
<td>86</td>
<td>95</td>
<td>0.90 (0.72 to 1.12)</td>
</tr>
<tr>
<td>Accidents (E800–E899)</td>
<td>52</td>
<td>44</td>
<td>1.18 (0.88 to 1.54)</td>
</tr>
<tr>
<td>Transport accidents (E800–E848)</td>
<td>16</td>
<td>22</td>
<td>0.73 (0.42 to 1.18)</td>
</tr>
<tr>
<td>Non-transport accidents (E850–E869, E880–E929)</td>
<td>36</td>
<td>22</td>
<td>1.61 (1.15 to 2.27)</td>
</tr>
<tr>
<td>Falls (E880–E888)</td>
<td>21</td>
<td>11</td>
<td>1.87 (1.18 to 2.92)</td>
</tr>
<tr>
<td>Struck by falling objects (E916–E928)</td>
<td>9</td>
<td>4.7</td>
<td>1.90 (0.88 to 3.44)</td>
</tr>
<tr>
<td>Suicide (E950–E959)</td>
<td>27</td>
<td>46</td>
<td>0.59 (0.39 to 0.85)</td>
</tr>
<tr>
<td>All deaths (ICD 001–999)</td>
<td>818</td>
<td>1150</td>
<td>0.71 (0.66 to 0.76)</td>
</tr>
</tbody>
</table>

*With correction for missing death certificates according to Rittgen and Becker.

RESULTS

Table 1 shows characteristics of the study population at baseline examination. Bricklayers represented the largest professional group, with almost one third of the total study population. Mean age of the study population at baseline was 42.6 years, with highest numbers in the age groups 25–34 years and 45–54 years. Almost 75% of the cohort members were of German nationality, followed by migrants (or their descendants) from former Yugoslavia, Italy, and Turkey. On average, cohort members have worked over 20 years in the building trade. Exposure to asbestos and to silica was very small (three cases).

During the follow-up period 818 men died. Death certificates were available for 694 (84.8%) of the deceased. Among these, cancer (43.1%) and cardiovascular disease (26.7%) were the most common causes of death. The third leading cause of death was injuries and poisoning (12.4%). Injuries and poisoning were the leading causes of death among those construction workers who died aged 25–44 years, and accounted for almost one third of all fatalities in this age group. Digestive diseases (63 cases) accounted for 9.1% of all deaths across all ages and were mainly related to liver cirrhosis (58 cases; data not shown).

Table 2 shows standardised all-cause and cause specific mortality ratios for the total cohort. With respect to all-cause mortality, this cohort of construction workers experienced a 29% lower mortality than males from the general population (SMR 0.71). Lower than expected mortality was observed for most causes of death, in particular, mortality due to cardiovascular diseases (SMR 0.59) and cancer (SMR 0.89). Cancers of the gastrointestinal system and the respiratory system accounted for one third of cancer deaths each. For cancer of the respiratory system, the number of observed was very close to the number of expected cases (SMR 1.01) despite a high smoking prevalence in the cohort and the potential for exposure to asbestos and silica dust. No excess cancer mortality was observed for other major tumour sites.

There was no excess mortality with respect to diseases of the respiratory system or to diseases of the digestive system. However, there was some (non-significant) excess mortality due to pneumoconiosis (SMR 2.30), but the number of cases was very small (three cases).

Injury and poisoning were responsible for 86 deaths. Among those were 16 transport accidents, 36 non-transport accidents, and 27 suicides. Excess mortality was limited to non-transport accidents (SMR 1.61) and mainly due to falls (21 cases; SMR 1.87) and being struck by falling objects (9 cases; SMR 1.90). Increase in risk was not observed for transport accidents (16 cases; SMR 0.73) or for suicides (27 cases; SMR 0.59).

Further in-depth analysis with stratifying the cohort by age, nationality, occupation, and duration of employment did not reveal any increased risk with respect to total mortality and cancer, diseases of the circulatory, respiratory, or the digestive system as cause of death (table 3) with the exception of an increased SMR of 1.51 (95% CI 1.00 to 2.30) for cancer mortality among the 25–44 year olds. When we stratified the sample into workers of German and non-German nationality, lower than expected mortality was observed for both groups, but workers of non-German nationality showed a statistically significant lower all-cause mortality than German worker (p = 0.01). Similar differences by nationality were found within the cause specific analysis. Stratification by profession revealed that mortality was highest among labourers for all-cause mortality and for mortality from most causes specifically addressed in this...
### Table 3  All-cause and cause specific SMR by age, nationality, occupation, and duration of employment

<table>
<thead>
<tr>
<th>Age (during follow up)</th>
<th>Obs</th>
<th>SMR [95% CI]</th>
<th>SMR [95% CI]</th>
<th>Obs</th>
<th>SMR [95% CI]</th>
<th>Obs</th>
<th>SMR [95% CI]</th>
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<th>SMR [95% CI]</th>
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<th>SMR [95% CI]</th>
<th>Obs</th>
<th>SMR [95% CI]</th>
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</thead>
<tbody>
<tr>
<td>25–44</td>
<td>98</td>
<td>0.88 (0.71 to 1.07)</td>
<td>12</td>
<td>0.80 (0.41 to 1.40)</td>
<td>2</td>
<td>1.24 (0.15 to 4.52)</td>
<td>9</td>
<td>1.12 (0.51 to 2.14)</td>
<td>25</td>
<td>0.80 (0.52 to 1.19)</td>
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<tr>
<td>45–54</td>
<td>191</td>
<td>0.78 (0.67 to 0.90)</td>
<td>33</td>
<td>0.61 (0.42 to 0.86)</td>
<td>2</td>
<td>0.37 (0.05 to 1.34)</td>
<td>15</td>
<td>0.69 (0.38 to 1.13)</td>
<td>24</td>
<td>1.00 (0.64 to 1.49)</td>
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<tr>
<td>55–64</td>
<td>417</td>
<td>0.67 (0.61 to 0.74)</td>
<td>107</td>
<td>0.38 (0.47 to 0.70)</td>
<td>11</td>
<td>0.49 (0.24 to 0.86)</td>
<td>34</td>
<td>0.67 (0.46 to 0.93)</td>
<td>34</td>
<td>1.05 (0.74 to 1.48)</td>
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<tr>
<td>&gt;65</td>
<td>112</td>
<td>0.65 (0.54 to 0.78)</td>
<td>33</td>
<td>0.60 (0.41 to 0.84)</td>
<td>8</td>
<td>0.96 (0.42 to 1.90)</td>
<td>5</td>
<td>0.61 (0.20 to 1.42)</td>
<td>3</td>
<td>0.63 (0.13 to 1.83)</td>
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<td>Nationality</td>
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<tr>
<td>German</td>
<td>663</td>
<td>0.75 (0.70 to 0.81)</td>
<td>156</td>
<td>0.62 (0.53 to 0.73)</td>
<td>19</td>
<td>0.61 (0.37 to 0.96)</td>
<td>53</td>
<td>0.75 (0.56 to 0.98)</td>
<td>71</td>
<td>0.94 (0.74 to 1.19)</td>
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<tr>
<td>Other</td>
<td>153</td>
<td>0.57 (0.49 to 0.67)</td>
<td>29</td>
<td>0.50 (0.34 to 0.72)</td>
<td>4</td>
<td>0.59 (0.16 to 1.51)</td>
<td>10</td>
<td>0.55 (0.27 to 1.02)</td>
<td>15</td>
<td>0.81 (0.44 to 1.30)</td>
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<td>Occupation</td>
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<tr>
<td>Plumbers</td>
<td>66</td>
<td>0.57 (0.44 to 0.72)</td>
<td>33</td>
<td>1.04 (0.71 to 1.45)</td>
<td>8</td>
<td>0.28 (0.12 to 0.54)</td>
<td>2</td>
<td>0.59 (0.07 to 2.13)</td>
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<td>0.44 (0.12 to 1.11)</td>
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<td>0.40 (0.14 to 0.97)</td>
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<tr>
<td>Carpenters</td>
<td>83</td>
<td>0.54 (0.44 to 0.68)</td>
<td>30</td>
<td>0.68 (0.46 to 0.97)</td>
<td>22</td>
<td>0.52 (0.33 to 0.80)</td>
<td>5</td>
<td>0.94 (0.31 to 2.20)</td>
<td>7</td>
<td>0.60 (0.26 to 1.31)</td>
<td>7</td>
<td>0.54 (0.23 to 1.11)</td>
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<tr>
<td>Painters</td>
<td>120</td>
<td>0.74 (0.61 to 0.88)</td>
<td>45</td>
<td>0.91 (0.66 to 1.20)</td>
<td>33</td>
<td>0.73 (0.50 to 1.03)</td>
<td>3</td>
<td>0.55 (0.11 to 1.60)</td>
<td>4</td>
<td>0.29 (0.08 to 0.73)</td>
<td>17</td>
<td>1.13 (0.66 to 1.82)</td>
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<tr>
<td>Bricklayers</td>
<td>95</td>
<td>0.73 (0.60 to 0.89)</td>
<td>35</td>
<td>0.89 (0.61 to 1.22)</td>
<td>22</td>
<td>0.39 (0.17 to 0.90)</td>
<td>3</td>
<td>0.66 (0.14 to 1.61)</td>
<td>9</td>
<td>0.86 (0.37 to 1.55)</td>
<td>8</td>
<td>0.76 (0.31 to 1.61)</td>
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<tr>
<td>Labourers</td>
<td>295</td>
<td>0.72 (0.64 to 0.81)</td>
<td>104</td>
<td>0.86 (0.70 to 1.04)</td>
<td>72</td>
<td>0.64 (0.50 to 0.80)</td>
<td>5</td>
<td>0.36 (0.12 to 0.83)</td>
<td>26</td>
<td>0.81 (0.53 to 1.19)</td>
<td>28</td>
<td>0.89 (0.60 to 1.31)</td>
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<tr>
<td>Duration of employment (at time of baseline examination)</td>
<td></td>
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<tr>
<td>&lt;15 years</td>
<td>119</td>
<td>0.74 (0.62 to 0.89)</td>
<td>29</td>
<td>0.84 (0.56 to 1.19)</td>
<td>20</td>
<td>0.63 (0.38 to 0.97)</td>
<td>3</td>
<td>0.79 (0.16 to 2.31)</td>
<td>8</td>
<td>0.72 (0.31 to 1.43)</td>
<td>24</td>
<td>0.94 (0.62 to 1.42)</td>
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<tr>
<td>15–29 years</td>
<td>222</td>
<td>0.64 (0.56 to 0.73)</td>
<td>77</td>
<td>0.78 (0.61 to 0.97)</td>
<td>51</td>
<td>0.57 (0.42 to 0.75)</td>
<td>6</td>
<td>0.57 (0.20 to 1.19)</td>
<td>22</td>
<td>0.77 (0.48 to 1.15)</td>
<td>24</td>
<td>0.81 (0.51 to 1.19)</td>
<td></td>
</tr>
<tr>
<td>&gt;30 years</td>
<td>371</td>
<td>0.72 (0.65 to 0.80)</td>
<td>153</td>
<td>0.93 (0.79 to 1.09)</td>
<td>94</td>
<td>0.60 (0.49 to 0.74)</td>
<td>10</td>
<td>0.50 (0.24 to 0.92)</td>
<td>29</td>
<td>0.72 (0.49 to 1.04)</td>
<td>26</td>
<td>0.93 (0.61 to 1.36)</td>
<td></td>
</tr>
</tbody>
</table>

*With correction for missing death certificates according to Rittgen and Becker.*
analysis. In particular, deaths due to external causes showed an SMR of 1.60 (95% CI 1.00 to 2.47). Finally, there was no clear association between mortality and duration of employment, either for all-cause mortality or for any specific cause. Furthermore, analysis by latency (with 20, 30, and 40 years lag time) revealed no evidence for an increased risk either for cancer or for all-cause mortality (data not shown).

Table 4 depicts some details of non-transport accidents which represent an area of concern within this cohort. Relative risk of non-transport accidents was highest for workers below age 55 and labourers. Risk of fatal falls was increased for all professions in this cohort compared with general population norms, whereas being struck by falling objects seemed to represent a particular hazard for labourers (SMR 6.01) and non-German workers (SMR 4.28).

**DISCUSSION**

Overall, construction workers in the German building trade experience higher risk of dying due to non-transport accidents and pneumoconiosis, but seem to experience lower risk of dying due to non-transport accidents, falls, and being struck by falling objects compared with males from the general population. This lower than expected mortality might be surprising given the working conditions including potential exposure to carcinogens, physically demanding jobs, climatic conditions, heavy lifting, noise, vibration, and lifestyle factors such as cigarette smoking and heavy alcohol consumption and deserves careful discussion.

Excess mortality due to accidents seems to be limited to non-transport accidents in this cohort and does not apply to transport accidents as reported by Ore and Fosbroke, who observed that construction workers were twice as likely to be killed by a motor vehicle as the average worker. This difference might be explained by the fact that our cohort was recruited within the building trade and not from road construction where transportation accidents pose a major problem.

Almost 20% of all work related injuries in Germany and over 23% of all work related fatal injuries occur in the construction industry. The annual injury rate (non-fatal and fatal accidents) of 82 per 1000 construction workers is about 2.5 times the average rate of 34.5 per 1000 in all trade industries. Similar figures have been reported from the USA and the UK. We observed a statistically significant excess mortality in this cohort for non-transport accidents, such as falls and being struck by falling objects. Other studies have not established whether older or younger workers are at higher risk of injuries. In several studies older workers were more likely to suffer work related injuries, whereas other reports have suggested that younger workers are at increased risk. In our study, young and middle-aged men, labourers, and workers of foreign nationality seemed to represent high risk groups for fatal accidents.

The dangers of construction sites are well known and high rates of traumatic fatalities have been previously documented, specifically falls from ladders or scaffolds, falls from or out of buildings or structures, and electrocutions. Carpenters experienced the lowest risk for fatal falls in this cohort although they work predominantly with a risk of falling. This apparent discrepancy might be explained by their experience and awareness of the risks and safety guidelines while working on roofs, ladders, or scaffolds/platforms. In contrast, non-awareness of safety guidelines might be a reason for the high fatality rates due to being struck by falling objects among foreign workers and labourers. However, the numbers of fatal falls within each job category were very small and the findings deserve confirmation from larger samples.

The low mortality with respect to all-cause mortality, cancer, and cardiovascular disease is likely to reflect a ‘healthy worker survivor effect’, a continuing selection process in that those who remain employed tend to be healthier than those who leave employment. Previous studies among construction workers using SMR techniques revealed similar findings regarding all-cause and cancer specific mortality. For example Engholm and Englund reported an SMR of 0.74 for all-cause mortality and of 0.88 for cancer mortality among construction workers in Sweden when compared with the general population. Both figures are very close to the results derived from our cohort.

Although our study subjects had been working in the construction industry for over 20 years on average (at baseline) and followed over an 8–14 years period, the current...
In our study, migrants from other countries experienced a 25% lower mortality compared to German construction workers. This phenomenon may be due to either a healthy migrant effect caused by a selection process in the 1960s when workers from Southern European countries with good physical health were hired to work in Germany, or an “unhealthy re-migration effect”.4,11

Our data indicate that, among construction workers, labourers experience the highest mortality with respect to several causes of death. Similarly, a Finnish study12 reported that semiskilled construction workers had the highest mortality rate almost independent of the cause of death, but differences to other professional groups were highest with respect to accidents. A recent study from the USA also indicates that labourers experience the highest rates of fatal occupational injuries.25

A major limitation inherent in studies which rely on voluntary participation is the potential for bias due to non-representative self selection of the study members. Results from the Swedish construction industry preventive health programme indicated that non-participants of occupational safety and health screening programmes had mortality rates 72% higher than those of participants.16 The differences seemed to be especially noteworthy for features associated with poor health behaviour such as alcoholism and liver cirrhosis. In this study, we were not able to obtain further information from non-participants. Therefore we cannot rule out that the observed mortality of study participants is somewhat underestimating the true mortality of the source population, although over 75% of all eligible employees participated in the baseline examination. Similarly, we are also concerned about those who left the construction industry for health reasons before the baseline examination took place. Another limitation inherent in mortality studies is the fact that hazards that cause substantial morbidity (but impose a low risk of mortality) such as hearing loss, and musculoskeletal and skin disorders cannot be studied but nevertheless pose major challenges for occupational health and also deserve close attention. So far, we have only been able to look at mortality data, which are often less accurate than morbidity data. Information of cause of death was able to look at mortality data, which are often less accurate than morbidity data. Information of cause of death was adjusted by Rittgen and Becker works well for the more frequent causes of death, estimates for rare causes of deaths (such as site specific cancers) might be less accurate. Further follow up with respect to mortality and morbidity should help to resolve some of the open issues. Despite its limitations, our study indicates that the construction industry is still a high risk trade with respect to non-transport accidents with a large number of injuries, which are in general primarily preventable. The observed variations in fatal injury rates with respect to age, nationality, and occupation indicate the potential for further targeting of occupational safety and health measures. Further efforts to reduce the number of fatal accidents are necessary and should address young and middle-aged, semi-skilled, and foreign workers, in particular.

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Known or suggested risk factors for osteoarthritis of the hip (coxarthrosis) include physical workload, sporting activities, and obesity. Farmers and farm workers may be at particularly high risk and, within farming, tractor driving (especially in a twisted position), working with dairy herds or pigs, milking cows, and working on small farms have all been regarded as possible risk factors. Now a Danish study of four successive cohorts of male workers has provided fresh data about time trends and risks in different socioeconomic and occupational groups.

All employed Danish men aged 20–59 years in January 1981, 1986, 1991, and 1994 were grouped according to main occupation one year previously and followed up for two to five years for hospital admission with hip arthritis. The highest rates of first admission for this cause during 1994–99 were among self employed men and unskilled workers (standardised hospitalisation ratios (SHRs) 140 and 121) and unemployed men (SHR 156). Managers, salaried staff, and skilled workers had relatively low SHRs (56–96). Between 1981 and 1999 the SHR decreased slightly among self employed men and increased slightly among leading salaried staff and skilled and unskilled workers. Between 1981–85 and 1994–97 the SHR remained steady and high (281–286) among self employed farmers and was lower but rising (114–189) among farm employees. Men in agricultural tractor pools had an SHR of 210 in 1981–85 and 183 in 1994–99. Men who had much lower SHRs in 1994–99 included electronics mechanics and servicers (SHR 12), teachers (37), doctors (46) and armed forces (57). High risk occupations included bakers and pastry cooks (SHR 204), waiters and bartenders (204) and machine operators (207).

In recent decades the number of farms and farmers has fallen in Denmark but the number of farm workers has remained the same. Increased SHRs among farm workers might be due to their having taken over tasks previously done by farmers. Specific factors probably associated with increased risk of hip osteoarthritis include heavy lifting, prolonged standing, body vibration, tractor driving, and walking on rough ground.

All-cause and cause specific mortality in a cohort of 20,000 construction workers; results from a 10 year follow up

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Can δ-aminolevulinic acid dehydratase 2 allele exert certain protective measures against the neurotoxic effects of lead?

Recently, there has been a lot of interest regarding δ-aminolevulinic acid dehydratase (ALAD) polymorphism and health effects of inorganic lead. Most of these reports focused on renal effects.1-3 There have been reports on the effects of lead on neurobehavioural functions of exposed workers.4-6 As far as we know, only one paper has reported the effects of ALAD polymorphism and neurobehavioural testing. Bellinger et al studied 72 adolescents with high (>24 μg/dl) and low (<12 μg/dl) blood lead levels. The results suggested that the body burden and effects of lead on neurobehavioural functions were worse among ALAD1 homozygotes.7 We have carried out a cross-sectional study in 106 male workers exposed to low or moderate levels of inorganic lead in order to investigate the association between ALAD1 and ALAD2 genotypes and neurobehavioural functions. Blood and urine were collected for each worker to determine the ALAD genotype, blood lead levels, ALAD, and urinary δ-aminolevulinic acid (ALAU). ALAD1-1 was the predominant genotype for all three ethnic groups (Chinese, Malays, and Indians) while ALAD2-2 was the rarest. The distribution of ALAD1-2 was higher among the Malays (17.5%) and Indians (19.2%) compared to the Chinese (8.4%). A battery of tests from the World Health Organisation Neuro-behavioural Core Test Battery and the Grooved Peg Board (GPP) test (an additional test for motor dexterity) were used to assess the neurobehavioural functions.8 Workers with ALAD1-1 genotype had significantly higher mean ALAU (0.86 mg/g creatinine) compared to workers with ALAD1-2/2-2 genotypes (0.61 mg/g creatinine) even after correcting for possible confounders. No significant differences were noted for mean blood lead and haemoglobin levels for both the groups. ALAD1-2/2-2 genotype workers had significantly better results compared to ALAD1 genotypes in the mean GPP preferred hand (55.5 seconds vs 62.6 seconds; p < 0.01), GPP non-preferred hand (60.3 seconds vs 67.7 seconds; p < 0.05), and mean GPP scores for preferred and non-preferred hands (57.9 seconds vs 65.4 seconds; p < 0.001) tests.

These two groups of workers had similar lead exposure as measured by their blood lead levels (ALAD1-1 v ALAD1-2/2-2: 21.3 μg/dl v 22.7 μg/dl, respectively). Although there were no significant differences between the mean blood lead levels for the two groups, workers with ALAD1-1 genotypes had significantly higher ALAU compared to those with ALAD1-2/2-2 genotypes. It could be that, given the same amount of lead exposure, ALAD2 alleles are more resilient to the effects of lead as reflected in a lower concentration of ALAU.

Several lines of evidence have suggested that δ-aminolevulinic acid (ALA) is the neuropathological correlate in lead poisoning. In vitro studies have shown the neurotoxicity of ALA. Clinical manifestations of lead poisoning closely resemble those of the acute neurological attacks in the hepatic porphyrias, during which the levels of ALA and porphobilinogen are significantly increased. The role of ALA accumulation in lead poisoning is supported by the report that asymptomatic heterozygotes for the ALAD deficient porphyrins are prone to acute lead poisoning when exposed to low levels of lead. Lead is known to inhibit ALAD which results in the build up of ALA, detectable in the plasma and urine at blood lead levels less than 10 μg/dl. Aminolevulinic acid resembles γ-aminobutyric acid receptors in the nervous system; this is thought to be one of the primary mechanisms of lead induced neurotoxicity.9 Bellinger et al studied 79 subjects (aged 19 or 20 years) using a battery of neuropsychological tests. Sixty seven of the subjects had ALAD1-1 phenotypes, while five had ALAD1-2. On “nearly every endpoint” of the neuropsychological test, the five individuals with the ALAD2 phenotype had better scores compared to 67 subjects with ALAD1, even after adjustment for dentin lead levels.10 Our subjects with ALAD1-2/2-2 genotypes also did significantly better in one of the neurobehavioural tests compared to subjects with ALAD1-1 genotype.

In summary, workers with ALAD1-1 genotypes have significantly higher ALAU and had significantly poorer neurobehavioural scores involving motor dexterity (GPP) compared to workers with ALAD1-2/2-2 genotypes. The ALAD2 allele may exert certain protective measures against the neurotoxic effects of lead as shown by lower ALAU levels among workers with the ALAD2 allele. This hypothesis is preliminary given the small sample size of the group with ALAD1-2/2-2 genotypes. Further study involving a larger cohort of workers with ALAD2 allele would be needed to confirm this.

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BOOK REVIEWS

ABC of occupational and environmental medicine, 2nd edition

What is an ABC? I think it is usually taken to be a simple introductory text, suitable for those contemplating learning more about the subject, and therefore I read this book with the needs of such people in mind. They might be medical students, general practitioners or non-medical occupational health workers, or indeed the sort of people I have taught over the years. How far does it go towards serving this purpose?

No fewer than 21 people have contributed, which must be close to a record for such a short book. This means that most of the chapters are written by people who have extensive practical knowledge of their subjects, and the editors have made a good effort to see that the specialist contributors have covered the occupational medical issues and vice versa. The format is of brief textual descriptions supplemented by many tables, boxes, and illustrations. This does not make for fluent reading since these interrupt each other to fit the size of the page rather than the logic of the argument. The 20 chapters vary in length and detail but are up-to-date and accurate in the facts provided; the tables in particular are a fund of useful information. However, the level of information does vary and some chapters serve the prospective reader very well as an introduction while others will bewilder with detail. In terms of an interesting read (which in my old fashioned way I always hope for in a book), the last four chapters on genetic and environmental matters are the most thought provoking. The lists of recommendations for further reading, including a good number of websites, are useful.

In my view it does not serve as an introduction to the subject, which purpose requires more explanation and less information. I think a tyro reading it would not really get a balanced understanding of what the practice of occupational and environmental medicine is about, and might even find it a daunting text. It does however have plenty in it for a postgraduate revising for examinations (but does not replace more detailed textbooks), and such trainees will certainly find it useful, as will most specialists for a bit of personal CMD. And I shall also find the tables and references useful for the next few years. For the price, it is good value for anyone training in or practising occupational medicine.

A Seanon
The particulate air pollution controversy: a case study and lessons learned


Confused about particles? Read this book!
The past 15 years have seen an explosion in interest in and concerns about the effects of ambient particles on health. Huge sums have been spent on research and journals are dominated by papers ranging from cutting edge epidemiology to molecular biology. Something for everybody—certainly—and no solution yet in sight.

Robert Phalen has tried to bring order to the field by producing a short book setting out what is known, what is unknown, and what are the lessons that should have been learnt. The factual content will be familiar to those in the field though many who comment on the field would do well to read this book closely. More important than the review of what we know are the author’s critiques of ill-founded inferences allegedly based on the evidence: The reader is stimulated by this and should recall: evidence is not proof, and hypotheses are not facts. Because the topic is important and because reducing levels of pollutants is becoming expensive and may involve actions that may affect health negatively, we need to be exacting in our requirements for proof of effects and proof of benefits. But how does all this fit in with the Precautionary Principle? This is not discussed and is a lapse on the part of the author. It may be that the US-centric approach that the author has adopted is responsible. European (including UK!) thinking about the Precautionary Principle is developing rapidly though this cannot be discussed here.

The author asks important questions about low dose affects, hormesis, and the overall costs and benefits of lowering levels of particles. Many will find areas for disagreement; all should be stimulated!