Lifetime risk of hospital diagnosed chronic obstructive pulmonary disease in remote Aboriginal people: a cohort study

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**Lifetime risk of hospital diagnosed chronic obstructive pulmonary disease in remote Aboriginal people: a cohort study**

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**Abstract**

**Objective:** To estimate the lifetime risk of developing hospital-diagnosed chronic obstructive pulmonary disease (COPD) in Aboriginal people living in remote areas.

**Methods:** A total of 1,374 participants in a remote community were followed up to 20 years. Individuals with hospital-diagnosed COPD were identified through hospital records. The lifetime risk of hospital-diagnosed COPD was estimated using a modified technique of survival analysis.

**Results:** Of the 1,374 participants, 164 were identified as having incident hospital-diagnosed COPD during 21,614 person years of follow-up. After adjusting for the presence of competing risk of death from non-COPD causes, the lifetime risk of COPD was 53% for the overall population, higher in women (61%) than in men (45%). Adjusting for baseline age and smoking status, women had a significantly higher risk of COPD than men with a hazard ratio (HR) = 1.55 (95%CI 1.13–2.14), while men were more likely to die from non-COPD causes than women before being diagnosed as having COPD, HR = 2.30 (95%CI 1.64–3.23).

**Conclusions:** These Aboriginal people have a high lifetime risk of COPD, and one in two have hospital-diagnosed COPD during their lifetime. Our findings warrant further efforts and resources to combat this condition in remote Aboriginal communities.

**Key words:** chronic obstructive pulmonary disease, lifetime risk, Aboriginal health, cohort study

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**Participants and hospital-diagnosed COPD**

Participants were recruited from a remote tribal group living in an isolated island setting in the Northern Territory of Australia from 1992 to 1998. All members of the community were invited to participate in the baseline screening in the local clinic, and only people who self-identified as belonging to the group were included. The baseline measurements and the follow-up of the cohort have been described previously. For the current study, 1,374 participants aged five to 75 years and free from known apparent hospital-diagnosed COPD recorded at baseline, representing more than 80% of those age groups in the community, were included in the final analysis. Those participants were followed until 31 May 2012. During the follow-up period, newly recorded COPD events were identified through hospital records of both primary and secondary diagnoses using codes of the International Classification of Diseases (ICD 9 codes 491, 492, 496, and ICD 10 codes J41–J44), including chronic bronchitis and emphysema. The hospitalisation data were obtained from the Northern Territory Department of Health. Deaths and their causes during the follow-up period were determined through a list of death records from the local clinic maintained by the research team.

By the end of the follow-up, one of three possible endpoints applied to each study participant: 1) had hospital-diagnosed COPD; 2) died without COPD; or 3) survived.
without COPD. Individuals were followed up until they were first diagnosed with COPD in their hospital records. Those who were free from hospital-diagnosed COPD during the follow-up period were monitored until death or 31 May 2012. For those participants who reached a hospital-diagnosed COPD event or who died from non-COPD causes before the end of the follow-up, the follow-up time was from the age of their initial screening visit to the age when they were diagnosed as having COPD or died. Others who survived the whole follow-period were censored at 31 May 2012. The chance of being hospitalised outside the Northern Territory was low for people in this remote, isolated region.

**Statistical analysis**

The data were partitioned into age bands of <20, 20–29, 30–39, 40–49, 50–59 and 60+ years throughout the follow-up. For those whose age fell into two or more age bands during the follow-up period, their total follow-up time was subdivided and allocated into corresponding age bands as described by Clayton and Hills. For calculation of lifetime risk, we used a modified technique of survival analysis and its computational technique has been described elsewhere in detail. This method has been used in a number of previous lifetime risk studies. Briefly, lifetime risk was calculated as in a simple Kaplan Meier analysis, but treating death as a true competing event, and a person’s risk for developing COPD was set to zero when they died. We adjusted for the competing risk of death from non-COPD causes and calculated risk separately for men and women. The differences of baseline characteristics among three endpoint groups were evaluated using analysis of variance or Chi-square test, as appropriate. All analyses were done with Stata 12.0.

This project was approved by the University of Queensland Behavioural & Social Science Ethical Review Committee (#2011001232). The study period, 150 died from non-COPD causes. The baseline characteristics of 849 adult participants who were 18 years or older at baseline with different endpoints are shown in Table 1. Those who were diagnosed as having COPD had similar body mass index as those without COPD. The prevalence of smoking and alcohol drinking at baseline was high in all male participants regardless of their endpoints. For women, the prevalence of smoking at baseline was significantly higher in those who were later diagnosed as having COPD than those who were not.

The incidence rate of developing new onset COPD increased with age (Figure 1), from less than one per 1,000 person-years for men and women under 30 years of age to 70 (95%CI 46–108) per 1,000 person-years for 60+ year old men and 48 (95%CI 32–72) per 1,000 person-years for 60+ year old women.

After adjusting for the presence of competing risk of death from non-COPD causes, the lifetime risk of COPD was 53% (95%CI 46–58) for the overall study population. The lifetime risk was higher in women (61% and 95%CI 52–69) than in men (45% and 95%CI 36–54), see Figure 2. Further adjusting for baseline age and smoking status, women had a significantly higher risk of COPD than men with a hazard ratio of 1.55 (95%CI 1.13–2.14). On the other hand, men were 2.30 (95%CI 1.64–3.23) times more likely to die from non-COPD causes than women before being diagnosed as having COPD (Figure 3). While adjusting for baseline age and gender, those who were identified as smokers at baseline had a significantly higher risk for COPD than non-smoking adults with a hazard ratio of 2.07 (95%CI 1.35–3.17).

### Table 1: Baseline characteristics of participants by COPD outcomes.

<table>
<thead>
<tr>
<th></th>
<th>Non-COPD</th>
<th>COPD</th>
<th>Died of non-COPD causes</th>
<th>p valueb</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number</td>
<td>276</td>
<td>61</td>
<td>86</td>
<td></td>
</tr>
<tr>
<td>Age range, years, n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;30</td>
<td>157 (56.9)</td>
<td>11 (18.0)</td>
<td>34 (39.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>30-49.9</td>
<td>113 (40.9)</td>
<td>28 (45.9)</td>
<td>37 (43.0)</td>
<td></td>
</tr>
<tr>
<td>≥50</td>
<td>6 (2.2)</td>
<td>22 (36.1)</td>
<td>15 (17.4)</td>
<td></td>
</tr>
<tr>
<td>Age, years, mean (SD)</td>
<td>30.1 (8.4)</td>
<td>43.1 (13.2)</td>
<td>36.5 (13.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI, kg/m², mean (SD)</td>
<td>23.3 (4.5)</td>
<td>23.8 (4.7)</td>
<td>23.8 (4.9)</td>
<td>0.54</td>
</tr>
<tr>
<td>Smoking, n (%)</td>
<td>224 (81.2)</td>
<td>49 (80.3)</td>
<td>67 (77.9)</td>
<td>0.80</td>
</tr>
<tr>
<td>Drinking, n (%)</td>
<td>232 (84.1)</td>
<td>49 (80.3)</td>
<td>76 (88.4)</td>
<td>0.40</td>
</tr>
</tbody>
</table>

| **Women**            |          |      |                         |          |
| Number               | 280      | 101  | 45                      |          |
| Age range, years, n (%) |         |      |                         |          |
| <30                  | 141 (50.4) | 13 (12.9) | 9 (20.0) | <0.001   |
| 30-49.9              | 124 (44.3) | 51 (50.5) | 23 (51.1) |          |
| ≥50                  | 15 (5.4) | 37 (36.6) | 13 (28.9) |          |
| Age, years, mean (SD) | 32.1 (10.2) | 44.7 (13.2) | 43.0 (14.6) | <0.001   |
| BMI, kg/m², mean (SD) | 24.4 (5.7) | 24.9 (6.4) | 24.5 (6.6) | 0.78     |
| Smoking, n (%)       | 168 (60.0) | 87 (86.1) | 33 (73.3) | <0.001   |
| Drinking, n (%)      | 81 (28.9) | 42 (41.6) | 16 (35.6) | 0.06     |

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a Only those who were 18 years or older at baseline were included for the purpose of comparing their characteristics and 525 younger than 18 years at baseline were not included.

b p values were calculated using analysis of variance for continuous variables or Chi-square test for categorical variables.
Discussion

In this cohort study, we found that one in two individuals in this remote community had hospital-diagnosed COPD during their lifetime. The lifetime risk in this Aboriginal group is much higher than those previously reported in non-Indigenous populations. In Ontario, Canada, Gershon et al. reported about one in four people in the general population will develop physician-diagnosed COPD in their lifetime. In a study of about 185,000 subjects in the Netherlands, Afonso et al. reported even lower lifetime risks of COPD for women and 24% for men. In another study in the Netherlands, Van Durme et al. reported 16% lifetime risk of developing COPD for women and 12.7% for men. In a recent study in Denmark by Lykkegaard et al., the lifetime risk of hospital-diagnosed COPD was 12.0% for women and 10.9% for men. Compared to those reported lifetime risks, the lifetime risk in Aboriginal people in this study is much higher, as shown in Figure 4. It is possible that the heterogeneity in COPD lifetime risk estimates among different populations could be due to the differences in COPD diagnosis, sampling procedures and risk factor levels. Nevertheless, the lifetime risk estimates in our study are considerably higher than those reported in the Canadian study using similar diagnostic criteria according to ICD 9 and ICD 10 codes.

In a cross-sectional study in Sweden, Lundback et al. suggested that the prevalence of COPD among elderly smokers is as high as 50%. A cohort study in Finland reported that the lifetime risk of chronic bronchitis was as high as 42% in smokers and 12% in never-smokers. The prevalence of COPD in Aboriginal people 50 years or older in the Northern Territory was 30%. Tobacco smoking among our study participants was common in both men and women – about 80% reported smoking at baseline. It is likely that a proportion of non-smokers at baseline could have become smokers during the follow-up period. Since smoking might have some effects on all members of the community, we were unable to accurately assess the effects of smoking on the lifetime risk of COPD in this study. The observed association between smoking and COPD is likely to be an underestimation. Other factors might also have contributed to the high COPD lifetime risk in this population. It has been reported that education, occupation exposure, childhood respiratory diseases and body mass index are associated with COPD in never smokers. There is a higher prevalence of asthma among Aboriginal Australians than other Australians. Aboriginal Australians are more likely to live in overcrowded housing and be exposed to pollutants and allergens such as dust and pollens. The rates of respiratory infections in NT Aboriginal infants are excessive. They also are more likely to have low birthweight, which has been suggested to be associated with pulmonary deaths. In this study, we found that women had a higher lifetime risk of hospital diagnosed COPD than men. In studies in Canada and Netherlands, men had a slightly higher lifetime risk than women while, in a Denmark study, women had slightly higher lifetime risk of COPD (Figure 4). These differences can be partly explained by sex differences in the prevalence of smoking among different populations. However, since men had a higher prevalence of smoking in our study population, the higher lifetime risk in women cannot be fully explained by higher cigarette smoking alone. It is possible that men with COPD may be less likely to seek care and may not have access to appropriate care. Another possible explanation is that Aboriginal men had a significantly higher risk of dying from competing non-COPD causes at younger ages before being diagnosed as having COPD in comparison with women. Nevertheless, further understanding the underlying causes in Aboriginal women is important for COPD prevention and management.

The lifetime risk of COPD in our study was higher than the lifetime risk of coronary heart disease and end-stage renal disease and similar to that of diabetes previously reported in the same population. Our findings draw attention to the huge burden of COPD in this remote Aboriginal group. Those estimates can be useful for promoting public interest in controlling COPD, particularly in Aboriginal women.

There are limitations in our study. First, the follow-up data were obtained from one tribal group in a remote region. It is possible that COPD risks are heterogeneous among different regions. Whether our findings are generalisable to the broader Aboriginal population in Australia should be further verified. Second, COPD events were determined based on routinely documented diagnosis information in hospital records during the follow-up period according to ICD codes. It is possible that asthma could have been misclassified as COPD and vice versa. However, we believe it is more likely that some true COPD cases were underdiagnosed through hospital records. Furthermore, the community is on an island about 80 km north of Darwin, where the nearest hospital is located. Therefore, it is relatively difficult
for the community members to visit hospital. Patients with minor COPD symptoms are more likely to visit the local clinics and those cases may not be captured in the hospital records. Therefore, lifetime risks in this study might have underestimated the true lifetime risk in the study population.

In summary, Aboriginal people in the study community have a high lifetime risk of COPD, and one in two will have hospital-diagnosed COPD during their lifetime. Our findings provide evidence for improving public awareness of COPD in this population, and warrant further efforts and resources to combat this condition. Our data also stress the importance of smoking cessation programs and the importance of implementing the COPD management guidelines in remote Aboriginal communities.

Acknowledgements

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References