EXTENDED REPORT

Smoking and risk of incident psoriatic arthritis in US women

Wenqing Li,1 Jiali Han,1,2,3 Abrar A Qureshi1,2

ABSTRACT
Objectives Psoriatic arthritis (PsA) is an inflammatory arthritis that is associated with psoriasis. Previous studies have found an association between smoking and psoriasis, but the association with PsA is unclear. The authors aimed to evaluate the association between smoking and the risk of incident PsA in a large cohort of women.

Methods 94 874 participants were included from the Nurses’ Health Study II over a 14-year period (1991–2005). Information on smoking was collected biennially during follow-up. The incidence of clinician-diagnosed PsA was ascertained and confirmed by self-reported questionnaires.

Results During 1 303 970 person-years’ follow-up, the authors identified 157 incident PsA cases. Among total participants, smoking was associated with an elevated risk of incident PsA. Compared with never smokers, the RR was 1.54 for past smokers (95% CI 1.06 to 2.24) and 3.13 for current smokers (95% CI 2.08 to 4.71). With increasing smoking duration or pack-years, the risk of PsA increased monotonically (p for trend <0.0001).

The increase in risk was particularly significant for PsA cases with more severe phenotypes. Secondary analysis among participants developing psoriasis during the follow-up replicated the association, demonstrating an increased risk of PsA among psoriasis cases. The risk was significant for those with higher cumulative measures of smoking or PsA cases with more severe phenotypes.

Conclusion In this study smoking was found to be associated with a risk of PsA and cumulative measures of smoking were also associated with a higher risk of PsA among women.

Psoriatic arthritis (PsA) is an inflammatory joint condition affecting an estimated 520 000 individuals in the US population.1 As a pleomorphic clinical entity, PsA occurs in 6–42% of psoriasis cases and the onset of joint symptoms does not usually occur until 8–10 years’ duration of psoriasis skin lesions.1,2

A delayed diagnosis and treatment of PsA may lead to an erosive arthropathy and impair quality of life.1 PsA can augment the risk of incident diabetes and cardiovascular disease beyond the effect of psoriasis (unpublished data). Evidence is conflicting on the severity of skin phenotypes between psoriasis and PsA.1,3–5 Therefore, prevention and early detection is warranted by understanding the risk factors for PsA.

The unfavourable cutaneous and joint effects of smoking have been suggested from past association studies.5,7 However, only sparse evidence regarding the association between smoking and PsA has been reported.8–12 To the best of our knowledge, no prospective data on smoking and PsA have been reported thus far.

In this study, we investigated the association between smoking status, duration, intensity and incidence of PsA in 94 874 participants from the Nurses’ Health Study II (NHS II).

METHODS

Study cohort

NHS II is an ongoing longitudinal cohort of women that was established in 1989 when 116 430 female nurses aged 25–42 years returned a baseline questionnaire on their medical history and lifestyle practices. Biennially, cohort members receive a questionnaire enquiring about diseases and health-related factors. The follow-up rate exceeds 90%.

Assessment of main exposure

In 1989, participants responded to a question on the lifetime history of smoking 20 packs (one pack equals 20 cigarettes) of cigarettes or more and if they were a past smoker, how many years had passed since quitting (<1 or ≥1 year). The questionnaire enquired about the quantities of smoking at different ages (<15, 15–19, 20–24, 25–29, 30–35 and 36–42 years) in six categories (1–4, 5–14, 15–24, 25–34, 35–44 and ≥45). Biennially, participants reported their smoking status. These questionnaires also enquired about the quantity of smoking in current smokers by the number of cigarettes per day in the above-mentioned six categories.

Based on information obtained biennially, smoking duration was obtained by deducting the age at smoking onset from the current age for current smokers, or from the age of cessation for past smokers. The pack-years were evaluated as the number of packs per day multiplied by the number of years of smoking.

We have the detailed information on smoking biennially for 115 770 participants from 1989, including smoking status, intensity, duration, etc. There was no marked difference in the main characteristics between those with complete and those with missing information on smoking.

Assessment of main outcome (PsA)


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We confirmed self-reported psoriasis by using the psoriasis screening tool (PST) questionnaire, which enquired about the type of clinicians making the diagnosis and the phenotypes.\textsuperscript{13} We developed scoring algorithms based on multiple a priori hypotheses to assign a diagnosis according to the response. The PST reached 99% sensitivity and 94% specificity for psoriasis screening.\textsuperscript{13} This questionnaire was mailed to 1886 participants who self-reported psoriasis and responded to the 2007 main questionnaire; 1637 (87%) responded and 1511 (92%) were confirmed.

The diagnosis of PsA was confirmed by using the psoriatic arthritis screening and evaluation (PASE) questionnaire, which includes a symptom scale with seven items and a function scale with eight items.\textsuperscript{14} Details of the instrument design and pilot studies have been described previously.\textsuperscript{14, 15} For each item, participants chose one of five categories relating to agreement (strongly agree to strongly disagree). A total score of 47 or greater has been shown in our pilot study to identify PsA with a high sensitivity and specificity.\textsuperscript{14, 15} PASE scores were positively associated with the 28-joint disease activity score (unpublished data). PASE has good test–retest reliability and is sensitive to change with an individual’s response to therapy.\textsuperscript{15} Furthermore, it can distinguish between symptoms of PsA and osteoarthritis.\textsuperscript{14}

### Table 1  Age-standardised baseline characteristics of study participants by smoking status, NHS II\textsuperscript{*}

<table>
<thead>
<tr>
<th>Smoking status</th>
<th>Never</th>
<th>Past</th>
<th>Current</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), years</td>
<td>35.8 (4.7)</td>
<td>37.0 (4.5)</td>
<td>36.6 (4.6)</td>
</tr>
<tr>
<td>Body mass index, kg/m(^2), mean (SD)</td>
<td>24.6 (5.3)</td>
<td>24.6 (5.2)</td>
<td>24.5 (5.1)</td>
</tr>
<tr>
<td>Alcohol intake, g/day, mean (SD)</td>
<td>2.4 (1.9)</td>
<td>2.4 (1.7)</td>
<td>2.2 (1.6)</td>
</tr>
<tr>
<td>Vigorous physical activity, metabolic equivalent h/week, mean (SD)</td>
<td>13.3 (21.9)</td>
<td>15.2 (24.8)</td>
<td>12.9 (22.2)</td>
</tr>
<tr>
<td>Personal history of chronic diseases (yes, %)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>0.6</td>
<td>0.5</td>
<td>0.4</td>
</tr>
<tr>
<td>Cancer</td>
<td>1.2</td>
<td>1.7</td>
<td>1.5</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>0.4</td>
<td>0.4</td>
<td>0.5</td>
</tr>
<tr>
<td>Hypertension</td>
<td>3.4</td>
<td>3.1</td>
<td>3.0</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>9.0</td>
<td>8.8</td>
<td>10.1</td>
</tr>
</tbody>
</table>

\textsuperscript{*} Characteristics of participants at the beginning of follow-up (return date of the 1991 questionnaire). Values are means (SD) or percentages and are standardised to the age distribution of the study population.

\textsuperscript{†} Values are not age-adjusted.

NHS II, Nurses’ Health Study II.

### Table 2  Age and multivariate-adjusted RR for the association of smoking status with the risk of PsA among all participants\textsuperscript{*}

<table>
<thead>
<tr>
<th>Cases</th>
<th>Person-years</th>
<th>Age-adjusted RR (95% CI)</th>
<th>Multivariate-adjusted RR† (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reference: never smokers</td>
<td>76</td>
<td>865 189</td>
<td>1.00</td>
</tr>
<tr>
<td>Smoking status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Past smokers</td>
<td>46</td>
<td>309 749</td>
<td>1.55 (1.07 to 2.23)</td>
</tr>
<tr>
<td>Current smokers</td>
<td>35</td>
<td>129 032</td>
<td>3.06 (2.06 to 4.61)</td>
</tr>
<tr>
<td>=1 cigarettes/day</td>
<td>12</td>
<td>62 479</td>
<td>2.19 (1.19 to 4.02)</td>
</tr>
<tr>
<td>≥15 cigarettes/day</td>
<td>23</td>
<td>66 553</td>
<td>3.92 (2.45 to 6.27)</td>
</tr>
<tr>
<td>Duration, years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;25</td>
<td>55</td>
<td>366 471</td>
<td>1.68 (1.19 to 2.38)</td>
</tr>
<tr>
<td>≥25</td>
<td>26</td>
<td>72 310</td>
<td>3.20 (2.02 to 5.07)</td>
</tr>
<tr>
<td>Pack-years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>44</td>
<td>342 974</td>
<td>1.43 (0.99 to 2.08)</td>
</tr>
<tr>
<td>20–44</td>
<td>34</td>
<td>91 129</td>
<td>3.67 (2.42 to 5.56)</td>
</tr>
<tr>
<td>≥45</td>
<td>3</td>
<td>4678</td>
<td>4.53 (1.41 to 15.46)</td>
</tr>
</tbody>
</table>

\textsuperscript{*} Psoriasis cases with only skin phenotypes were excluded during the follow-up.

\textsuperscript{†} Adjusted for age (continuous variable), body mass index (<18.5, 18.5–24.9, 25–29.9, 30–34.9 or ≥35 kg/m\(^2\)), alcohol drinking (no, <4.9, 5.0–9.9, 10–14.9, 15–29.9 or ≥30.0 g/day), vigorous physical activity (metabolic equivalent h/week, in quintiles).

PsA, psoriatic arthritis.

### Statistical analysis

We performed two sets of association analyses. One was analysed among the total participants. The other set was performed by only including individuals with psoriasis to evaluate the risk of PsA associated with smoking among those with psoriasis. We excluded self-reported psoriasis cases that were not validated for the main analysis. Simultaneously, we performed a sensitivity analysis by using all self-reports. Of the 97 476 responders, for both the two sets, we excluded participants with the psoriasis item passing through (N=58), PsA or psoriasis that occurred before 1991 (N=1376), self-reported psoriasis cases who were not confirmed (N=97) or did not respond to the PST or PASE questionnaire (N=467), PsA cases with a lack of diagnosis date (N=2), and participants with missing information on smoking (N=506). For the first set, we excluded incident psoriatics without joint phenotypes in the first follow-up period (N=96); therefore 94 874 participants remained. For the second set, we...
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Table 3  Age and multivariate-adjusted RR for the association between smoking and the risk of PsA with different severity scores*

<table>
<thead>
<tr>
<th>Person-years</th>
<th>Scores 47–57</th>
<th>Multivariate-adjusted RR† (95% CI)</th>
<th>Scores ≥57</th>
<th>Multivariate-adjusted RR† (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reference: never smokers</td>
<td>865 189</td>
<td>54</td>
<td>1.00</td>
<td>22</td>
</tr>
<tr>
<td>Smoking status</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Past smokers</td>
<td>309 749</td>
<td>35</td>
<td>1.61 (1.04 to 2.48)</td>
<td>11</td>
</tr>
<tr>
<td>Current smokers</td>
<td>129 032</td>
<td>15</td>
<td>2.30 (1.35 to 3.91)</td>
<td>16</td>
</tr>
<tr>
<td>1–14 cigarettes/day</td>
<td>62 479</td>
<td>7</td>
<td>1.88 (0.85 to 4.16)</td>
<td>5</td>
</tr>
<tr>
<td>≥15 cigarettes/day</td>
<td>66 553</td>
<td>12</td>
<td>2.64 (1.40 to 4.99)</td>
<td>11</td>
</tr>
<tr>
<td>p for trend</td>
<td></td>
<td></td>
<td>0.002</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Duration, years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;25</td>
<td>366 471</td>
<td>42</td>
<td>1.80 (1.19 to 2.72)</td>
<td>13</td>
</tr>
<tr>
<td>≥25</td>
<td>72 310</td>
<td>12</td>
<td>1.80 (0.95 to 3.43)</td>
<td>14</td>
</tr>
<tr>
<td>p for trend</td>
<td></td>
<td></td>
<td>0.002</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Pack-years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>342 974</td>
<td>33</td>
<td>1.54 (0.99 to 2.40)</td>
<td>11</td>
</tr>
<tr>
<td>≥20</td>
<td>95 807</td>
<td>21</td>
<td>2.47 (1.47 to 4.17)</td>
<td>16</td>
</tr>
<tr>
<td>p for trend</td>
<td></td>
<td></td>
<td>0.001</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

*Psoriasis cases with only skin phenotypes were excluded during the follow-up. PsA cases were classified into two groups by the cutoff of scores at 57, picked to include those with the top third of the scores into one category.
†Adjusted for age (continuous variable), body mass index (<18.5, 18.5–24.9, 25–29.9, 30–34.9 or ≥35 kg/m²), alcohol drinking (no, <4.9, 5.0–9.9, 10–14.9, 15–29.9 or ≥30.0 g/day), vigorous physical activity (metabolic equivalent h/week, in quintiles), and alcohol intake (0, <4.9, 5.0–9.9, 10–14.9, 15–29.9 or ≥30.0 g/day). As a sensitivity analysis, postmenopausal hormone use (premenopausal, never, or ever users), personal history of cancer, diabetes, cardiovascular disease, hypertension and hypercholesterolaemia (yes or no) were concomitantly adjusted for. For multycategorical measures of smoking, trend tests were carried out by using the median in different categories. All analyses were updated because the variables are all time varying.

We performed a sensitivity analysis in a case–control design to include all PsA and confirmed psoriasis cases before and after 1991.

We performed association analyses between smoking and the risk of PsA with different severity scores (47–57 or ≥57). We set the cutoff value as 57 because approximately a third of the PsA cases in our study had a score higher than 57.

Statistical analysis system software (SAS, version 9.2) was used to conduct statistical analyses. All tests were two-tailed, and the significance level was set at p<0.05.

excluded participants who did not develop psoriasis (N=94 389); 581 with confirmed psoriasis remained.

Person-years of follow-up were calculated by deducting the return date of the 1991 questionnaire from the date of PsA diagnosis, or June 2005, whichever came first.

Information on smoking was updated during the follow-up. Smoking status was categorised as never, past and current. The quantities of current smokers were classified to 1–14 or 15 or more cigarettes per day. Other variables included smoking duration (<25 or ≥25 years) and pack-years (<20, 20–44 or ≥45 pack-years).

Stratified by age and follow-up interval, we performed Cox proportional hazards analysis to calculate the age and multivariate-adjusted RR and 95% CI for the association between smoking and incident PsA. Multivariate-adjusted analysis was performed after adjusting for age (continuous variable), body mass index (<18.5, 18.5–24.9, 25–29.9, 30–34.9 or ≥35 kg/m²), vigorous physical activity (metabolic equivalent h/week, in quintiles) and alcohol intake (0, <4.9, 5.0–9.9, 10–14.9, 15–29.9 or ≥30.0 g/day). As a sensitivity analysis, postmenopausal hormone use (premenopausal, never, or ever users), personal history of cancer, diabetes, cardiovascular disease, hypertension and hypercholesterolaemia (yes or no) were concomitantly adjusted for. For multycategorical measures of smoking, trend tests were carried out by using the median in different categories. All analyses were updated because the variables are all time varying.

We performed a sensitivity analysis in a case–control design to include all PsA and confirmed psoriasis cases before and after 1991.

We performed association analyses between smoking and the risk of PsA with different severity scores (47–57 or ≥57). We set the cutoff value as 57 because approximately a third of the PsA cases in our study had a score higher than 57.

Statistical analysis system software (SAS, version 9.2) was used to conduct statistical analyses. All tests were two-tailed, and the significance level was set at p<0.05.

Table 4  Age and multivariate-adjusted RR for the association between smoking and the risk of PsA among participants with psoriasis

<table>
<thead>
<tr>
<th>Cases</th>
<th>Person-years</th>
<th>Age-adjusted RR (95% CI)</th>
<th>Multivariate-adjusted RR* (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reference: never smokers</td>
<td>76</td>
<td>2356</td>
<td>1.00</td>
</tr>
<tr>
<td>Smoking status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Past smokers</td>
<td>46</td>
<td>1191</td>
<td>1.17 (0.77 to 1.78)</td>
</tr>
<tr>
<td>Current smokers</td>
<td>35</td>
<td>805</td>
<td>1.56 (0.98 to 2.47)</td>
</tr>
<tr>
<td>1–14 cigarettes/day</td>
<td>12</td>
<td>328</td>
<td>1.12 (0.55 to 2.29)</td>
</tr>
<tr>
<td>≥15 cigarettes/day</td>
<td>23</td>
<td>477</td>
<td>1.91 (1.11 to 3.27)</td>
</tr>
<tr>
<td>p for trend</td>
<td></td>
<td></td>
<td>0.004</td>
</tr>
<tr>
<td>Duration, years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;25</td>
<td>55</td>
<td>1680</td>
<td>1.18 (0.81 to 1.74)</td>
</tr>
<tr>
<td>≥25</td>
<td>26</td>
<td>316</td>
<td>1.85 (1.08 to 3.17)</td>
</tr>
<tr>
<td>p for trend</td>
<td></td>
<td></td>
<td>0.024</td>
</tr>
<tr>
<td>Pack-years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>44</td>
<td>1453</td>
<td>1.08 (0.71 to 1.63)</td>
</tr>
<tr>
<td>≥20</td>
<td>37</td>
<td>543</td>
<td>1.87 (1.17 to 3.01)</td>
</tr>
<tr>
<td>p for trend</td>
<td></td>
<td></td>
<td>0.084</td>
</tr>
</tbody>
</table>

*PsA, psoriatic arthritis.

Adjusted for age (continuous variable), body mass index (<18.5, 18.5–24.9, 25–29.9, 30–34.9 or ≥35 kg/m²), alcohol drinking (no, <4.9, 5.0–9.9, 10–14.9, 15–29.9 or ≥30.0 g/day), vigorous physical activity (metabolic equivalent h/week, in quintiles).
The participants’ completion and return of the self-administered questionnaires was accepted as informed consent for the present study.

RESULTS

The age-adjusted baseline characteristics of the participants by smoking status are reported in table 1. Alcohol intake tended to increase from the never smokers to the current smokers.

During 1 303 970 person-years of follow-up, 157 were identified as PsA. Compared with never smokers, we observed an increased risk of incident PsA among past (multivariate-adjusted RR 1.54, 95% CI 1.06 to 2.24) or current smokers (RR 3.12, 95% CI 2.07 to 4.69). The risk of PsA was monotonically associated with increasing smoking quantities among current smokers (p for trend <0.0001; table 2). There was a graded association between duration or pack-years of smoking and the risk of PsA (p for trend <0.0001). The RR was 1.70 for duration less than 25 years and 3.12 for 25 years or over; 1.48 for less than 20 pack-years, 3.33 for 20–44 pack-years and 3.91 for 45 or more pack-years.

We evaluated the association between smoking and PsA with different severity scores. The association appears much stronger for PsA with scores of 57 or greater (table 3).

We repeated the analyses after excluding all participants without developing psoriasis to evaluate the risk of PsA among psoriasis cases. As is shown in table 4, the risk of PsA among confirmed psoriasis cases was monotonically elevated with smoking intensity and duration, but only significantly increased among current smokers with 15 or more cigarettes per day (RR 1.93, 95% CI 1.09 to 3.40), those with duration of 25 years or more (RR 1.90, 95% CI 1.09 to 3.33), or 20 or more pack-years (RR 2.02, 95% CI 1.24 to 3.29). We also evaluated the association by using all self-reported individuals with psoriasis and observed similar results (see supplementary table S1, available online only).

Among individuals with psoriasis, the risk of PsA with more severe phenotypes (PASE score ≥57) was remarkably elevated with the increasing smoking intensity or duration (p for trend <0.05; table 5). However, we did not observe a significant association for PsA with PASE scores of 47–57.

Secondary analyses were performed to adjust for postmenopausal hormone use and personal history of chronic diseases. The association was only slightly attenuated. Secondary analyses were also performed by excluding baseline chronic diseases, and no material change was observed (data not shown). We conducted a case–control analysis to include all psoriasis and PsA cases before and after 1991 and the association remains robust (see supplementary tables S2 and 3, available online only).

DISCUSSION

In this study, we observed that smokers were at an independently increased risk of developing PsA. The risk was positively correlated with smoking intensity and duration.

PsA is a chronic inflammatory arthritis associated with psoriasis.1–2 It has been postulated that smoking may play a role in the development of psoriasis through a variety of mechanisms, which may also be similar in PsA. Smoking induces oxidative stress and reduces antioxidant levels, leading to an imbalance of oxidants and antioxidants.6–9 More importantly, smoking can adversely alter the immunological and inflammatory processes, which could play important roles in the development of PsA.10–13 The association between smoking and psoriasis has been evaluated previously.5,6 Smoking is an established risk factor for rheumatoid arthritis.7 These data suggest a possible role of smoking in the development of PsA. However, the direct evidence regarding the association between smoking and PsA is sparse.8–12 Duffin et al6 reported that smoking may modulate the effect of interleukin-13 polymorphisms on risk of PsA. Several case–control studies including both women and men and psoriasis with different severities did not demonstrate an increased risk of PsA associated with smoking. Two did not point to a link between smoking and PsA in individuals with psoriasis.9,10 One indicated smoking as a protective for PsA.11 Another study showed that smoking may delay the onset of PsA in individuals with psoriasis.12 Given the study design, those results may have been affected by selection and information bias. Moreover, those studies are limited by study design such that they cannot determine the temporal association between smoking and PsA onset. They did not adjust for the main confounders. They also failed to indicate a dose–response relationship to validate their reports. Our longitudinal data provide evidence regarding the association between smoking and the risk of PsA. Given the temporal sequence, our results indicate that smoking is a risk factor for the development of PsA.

Previous studies showed that smoking impacts the occurrence or severity of psoriasis in a dose-dependent fashion.5,20 In the...
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present study, we observed a gradually elevated risk of develop-
ing PsA corresponding with the increasing quantities of cur-
rent smokers, smoking duration and pack-years. Furthermore,
according to the distribution, we categorised the PsA cases and
found that the effect of smoking appeared stronger for those
with higher PsA severity scores.

Our study is the first cohort study on the association between
smoking and the development of PsA. We evaluated multiple mea-
sures of smoking associated with the risk of PsA. We performed
secondary analyses by excluding or adjusting for psoriasis comor-
bidity for their possible association with smoking. We compared
the effect of smoking on the risk of PsA among total participants
as well as only among individuals with psoriasis. These analy-
ses observed a consistently increased risk of PsA associated with
smoking, which supported the robustness of the association.

In our study, physician-diagnosed psoriasis was collected in
2005; therefore survival bias may exist because we cannot obtain
information for individuals with psoriasis who died. However,
this is a younger female cohort of health professionals. We
found that the mean age of responders to the psoriasis question-
was even a little higher than non-responders; therefore, it is less
likely that our results were greatly distorted. We did not ask
the question on PsA in the main questionnaire and ascertained
the diagnosis by using the PASE questionnaire among self-re-
ported individuals with psoriasis. Case ascertainment could be
a concern due to misclassification. However, the psoriasis self-
reports have reached a confirmation rate of 92%.13 Pilot studies
indicated that PASE was a valid and reliable tool to screen for
PsA, especially active PsA among individuals with psoriasis.14 15
As our pilot study on PST and PASE showed high accuracy, we
expect an overall high validity of confirmation among registered
nurses. The prevalence of PsA among confirmed psoriasis cases
from 1991 is 21.2%, which fell within the intervals of previous
reports. There could be concerns about possible misclassification
with other musculoskeletal conditions, such as fibromyalgia and
ostearthritis. However, PASE can distinguish between symp-
toms of PsA and ostearthritis.14 In addition, evidence showed
an inverse or no association between smoking and the risk of
ostearthritis.21 22 There is little concern about fibromyalgia
because this occurs less commonly in psoriasis and the studies
on smoking and fibromyalgia still seem conflicting.23 24

Our study has other limitations. Most of the participants are
Caucasian women younger than 60 years, which limits the gen-
eralisation to men or other ethnic groups. The generalisation to
older women should be made with caution considering there
were no published findings to support: AAQ and JH. Study supervision: WL, AAQ and JH.

Critical revision of the manuscript for important intellectual content: WL, AAQ and JH.

Drafting of the manuscript: WL, AAQ and JH.

Obtained.

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concept and design: AAO, JH and WL. Acquisition of data: AAO and JH. Analysis and
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