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BRIEF REPORT

Smokeless Tobacco (Moist Snuff) Use and the Risk of Developing Rheumatoid Arthritis: Results From a Case–Control Study

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Objective. **To investigate the association between snuff use (smokeless tobacco containing nicotine) and the risk of anti–citrullinated protein/peptide antibody (ACPA)–positive and ACPA-negative rheumatoid arthritis (RA).**

Methods. **Data from the Swedish Epidemiological Investigation of Rheumatoid Arthritis, a population-based case–control study including 1,998 incident cases and 2,252 randomly selected controls (matched on age, sex, and residential area) ages 18–70 years, were analyzed. Ever, current, and past moist snuff users were compared with never users. We calculated odds ratios (ORs) with 95% confidence intervals (95% CIs) by means of unconditional logistic regression models. All analyses were adjusted for cigarette smoking, alcohol consumption, and the matching variables.**

Results. **In total, 254 (13%) cases were ever moist snuff users compared with 290 (13%) controls, resulting in an OR of 1.0 (95% CI 0.8–1.2) of RA overall. When exposure to moist snuff was analyzed in relation to ACPA-positive and ACPA-negative disease, no associations were observed. Neither current nor past moist snuff use was related to the risk of any of the 2 RA subgroups. Analyses restricted to never smokers provided similar results.**

Conclusion. **The use of moist snuff was not associated with the risk of either ACPA-positive or ACPA-negative RA. The increased risk of RA associated with smoking is most probably not due to nicotine.**

Introduction

For the chronic autoimmune inflammatory disease rheumatoid arthritis (RA), smoking is so far the most consistently demonstrated environmental risk factor associated with disease development (1–3). Notably, this increased risk is mainly seen in anti–citrullinated protein/peptide antibody (ACPA)–positive RA (4). A possible biologic explanation involving smoking, citrullination of proteins in the lungs, and HLA–DRB1 shared epitope has been proposed (5).

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Cigarette smoke is a complex mixture of chemical compounds, including nicotine, char, and other adjuvants that seem to act fairly differently to the immune system (6). Some constituents in smoke, such as char, may act as adjuvants and enhance both innate and adaptive immunity by activation of antigen-presenting cells (7), while nicotine may suppress inflammation by relatively well investigated mechanisms (8). Therefore, it is of interest to investigate which substances in inhaled smoke are related to increased RA risk and also to specifically investigate whether exposure to nicotine in smokeless tobacco is able to reduce risk for RA. Both these questions are of considerable clinical, as well as scientific, interest.

Swedish moist snuff (a smokeless tobacco) contains nicotine and is often used as an alternative to smoking. It delivers nicotine into venous circulation through passive absorption across the oral mucosa. Use of moist snuff leads to similar or even higher doses of nicotine compared with smoking (9). Since snuff and tobacco smoke mainly have nicotine in common, snuff use might not lead to exposure of the lung to the other substances in tobacco smoke (10,11). The role of smokeless tobacco in the etiology of RA has to date only been reported from a Swedish study based on male construction workers (12) where no association was observed. However, whether smokeless tobacco is related to RA in the general population, including females, and whether there are different effects of nicotine in

ACPA-positive versus ACPA-negative RA remains to be elucidated.

Therefore, in this report we aimed at investigating the association between moist snuff use and the risk of ACPA-positive and ACPA-negative RA, among males and females separately, by using a population-based case–control study.

Subjects and methods

This study is based on the Swedish Epidemiological Investigation of Rheumatoid Arthritis (EIRA) project (see Appendix A for list of EIRA Study Group members), a population-based, case–control study comprising incident cases and controls ages 18–70 years living in south/central regions of Sweden between 1996–2006. Cases were patients who received an RA diagnosis (American College of Rheumatology [ACR] 1987 criteria [13]) by rheumatologists in the participating area. Controls were randomly selected, matched by age, sex, and residential area, from the national population register. One control was selected per case. If an invited control declined to participate, another control was invited. At the start of the EIRA, cases that did not fulfill the 1987 ACR criteria were included. These cases were later excluded from the study, but their controls were kept for analyses. Details of the study design have been reported elsewhere (3). In total, 2,097 cases and 2,770 controls were invited, of which 1,998 cases (95%) and 2,252 controls (81%) participated by filling out a self-administrated questionnaire. Cases and controls were also asked to provide a blood sample for serologic analyses. All the participants consented to contribute to the study and ethical approval was obtained from relevant ethical committees.

Antibody assays. ACPA in EIRA blood samples was determined by measurement of anti–cyclic citrullinated peptide antibodies with a second-generation enzyme-linked immunosorbent assay (Immunoscan RA Mark2, Euro-Diagnostica). The cutoff for positivity was 25 AU/ml.

Exposures. The questionnaire comprised 2 questions on snuff use: 1) are you currently using snuff? (yes/no), and 2) if not, have you previously used snuff? (yes/no). Ever snuff users were defined as individuals who reported they had ever used moist snuff, including both current and former snuff users, while never snuff users reported they had never used snuff.

Statistical analysis. Odds ratios (ORs) and 95% confidence intervals (95% CIs) for the development of RA overall, as well as ACPA-positive and ACPA-negative RA associated with snuff use were calculated by means of unconditional logistic regression models. Ever, current, and former snuff users were compared with never users. First, we performed an analysis of the association between snuff use and RA risk based on all cases and controls, with adjustment for the design variables (age, sex, and residency) and for cigarette smoking (pack-years; pack-years of smoking were calculated with 1 pack-year equivalent to smoking 20 cigarettes per day for 1 year, and they were categorized as never smokers [0 pack-years], light smokers [<10 pack-years], medium smokers [10–20 pack-years], and heavy smokers [≥20 pack-years]), as well as for alcohol consumption (drinks/week; total alcohol consumption was measured in drinks per week [1 drink = 16 gm alcohol] and was categorized as never drinkers, light drinkers [<3 drinks/week], and medium/heavy drinkers [≥3 drinks/week]). We further performed the primary analysis in a matched manner by using conditional logistic regression models. The ORs from the matched analysis were consistent with the unmatched results; but since we lost information from 246 controls, we present the results from the unmatched analyses. Additional adjustments for education (<12 years, ≥12 years), silica exposure (yes/no), body mass index (modeled as both continuous and categorical), the primary genetic risk factor shared epitope (carriers of any copies of shared epitope versus noncarriers), and the second most important risk gene, the protein tyrosine phosphatase gene (carriers of any risk alleles of *PTPN22* rs2476601 versus noncarriers) did not substantially change the ORs and were therefore not retained in the final analyses. Since cigarette smoking is the major known environmental risk factor for RA, and there is a risk for residual confounding from smoking in the analysis based on the whole data set, we also performed analyses among never and ever smokers separately. We further performed the abovementioned analyses among men and women separately. All analyses were implemented through SAS, version 9.3.

Results

In total, data from 1,998 cases and 2,252 controls were available for the current study. Among the cases, 63% were ACPA-positive and the median time from symptom onset to diagnosis was 10 months. Thirty-four patients lacked information on ACPA status, while 7 individuals (2 cases and 5 controls) lacked information on snuff use, resulting in 1,962 cases and 2,247 controls available for the analyses. More ACPA-positive cases were current smokers and had a higher number of pack-years of smoking, while more controls were ever drinkers and had higher alcohol consumption, in accordance with previously reported observations (see Supplementary Table1, available in the online version of this article at http://onlinelibrary.wiley.com/doi/10.1002/acr.22325/abstract).

Moist snuff use and the risk of ACPA-positive/-negative RA. As shown in Table 1, 254 cases (12.9%) were ever moist snuff users compared with 290 controls (12.9%), resulting in an OR of 1.0 (95% CI 0.8–1.2) for RA overall. No association was observed when moist snuff use was analyzed in relation to incidence of ACPA-positive RA (OR 1.0, 95% CI 0.8–1.3) or ACPA-negative RA (OR 0.9, 95% CI 0.7–1.2). Furthermore, neither current nor past moist snuff use was related to the risk of any of the 2 RA subgroups.

Moist snuff use and the risk of ACPA-positive/-negative RA by smoking status. Among never smokers, no significant differences for ORs between snuff use and RA risk were seen for ACPA-positive RA (OR 1.0, 95% CI 0.5–1.9

Table 1. Association of moist snuff use and the development of RA overall, ACPA-positive RA, and ACPA-negative RA in the EIRA (Sweden, 1996–2006)*

Subjects	Moist snuff use		OR†	95% CI
	No, no. (%)	Yes, no. (%)		
Ever user				
Controls	1,957 (87.1)	290 (12.9)	1.0	Ref.
RA overall	1,708 (87.1)	254 (12.9)	1.0	0.8–1.2
ACPA-positive cases	1,066 (86.1)	172 (13.9)	1.0	0.8–1.3
ACPA-negative cases	642 (88.7)	82 (11.3)	0.9	0.7–1.2
Current user				
Controls	1,957 (91.6)	180 (8.4)	1.0	Ref.
RA overall	1,708 (91.2)	164 (8.8)	1.1	0.8–1.4
ACPA-positive cases	1,066 (90.7)	109 (9.3)	1.0	0.8–1.4
ACPA-negative cases	642 (92.1)	55 (7.9)	1.0	0.7–1.4
Former user				
Controls	1,957 (94.7)	110 (5.3)	1.0	Ref.
RA overall	1,708 (95.0)	90 (5.0)	0.9	0.6–1.2
ACPA-positive cases	1,066 (94.4)	63 (5.6)	1.0	0.7–1.4
ACPA-negative cases	642 (96.0)	27 (4.0)	0.8	0.5–1.2

* RA = rheumatoid arthritis; ACPA = anti–citrullinated protein/peptide antibody; EIRA = Epidemiological Investigation of Rheumatoid Arthritis; OR = odds ratio; 95% CI = 95% confidence interval; Ref. = reference.
† OR adjusted for age, sex, residential area, pack-years of smoking, and alcohol consumption.

for ever snuff users; OR 1.0, 95% CI 0.5–2.2 for current snuff users) as compared with ACPA-negative RA (OR 1.0, 95% CI 0.5–2.0 for ever snuff users; OR 1.5, 95% CI 0.7–3.3 for current snuff users) (Table 2). Notably, the numbers of observations were low, in particular in the ACPA-negative group. Similarly, among ever smokers, neither current nor former moist snuff use was related to the risk of ACPA-positive or ACPA-negative RA.

Since moist snuff use among never smokers was low (5.1%), we decided to increase our sample size by incorporating non–regular smokers and former smokers who had quit smoking a long time ago (women >12 years, men >32 years) (3) into the never smoker stratum. We considered it logical to merge these groups since we have not found any associations between non–regular smoking or former smoking with a long period of smoking cessation

Table 2. Association of moist snuff use and the development of RA overall, ACPA-positive RA, and ACPA-negative RA, stratified by smoking status in the EIRA (Sweden, 1996–2006)*

Subjects	Never smokers				Ever smokers			
	No moist snuff use, no. (%)	Moist snuff use, no. (%)	OR†	95% CI	No moist snuff use, no. (%)	Moist snuff use, no. (%)	OR‡	95% CI
Ever user								
Controls	802 (94.9)	43 (5.1)	1.0	Ref.	1,141 (82.4)	244 (17.6)	1.0	Ref.
RA overall	577 (95.5)	27 (4.5)	1.0	0.6–1.7	1,127 (83.2)	227 (16.8)	1.0	0.8–1.3
ACPA-positive cases	317 (95.2)	16 (4.8)	1.0	0.5–1.9	748 (82.7)	156 (17.3)	1.0	0.8–1.4
ACPA-negative cases	260 (95.9)	11 (4.1)	1.0	0.5–2.0	379 (84.2)	71 (15.8)	0.9	0.6–1.2
Current user								
Controls	802 (96.6)	28 (3.4)	1.0	Ref.	1,141 (88.5)	149 (11.5)	1.0	Ref.
RA overall	577 (96.3)	22 (3.7)	1.2	0.7–2.2	1,127 (88.8)	142 (11.2)	1.0	0.8–1.4
ACPA-positive cases	317 (96.7)	11 (3.4)	1.0	0.5–2.2	748 (88.4)	98 (11.6)	1.1	0.8–1.5
ACPA-negative cases	260 (95.9)	11 (4.1)	1.5	0.7–3.3	379 (89.6)	44 (10.4)	0.9	0.6–1.3
Former user								
Controls	802 (98.2)	15 (1.8)	1.0	Ref.	1,141 (92.3)	95 (7.7)	1.0	Ref.
RA overall	577 (99.1)	5 (0.9)	0.5	0.2–1.5	1,127 (93.0)	85 (7.0)	1.0	0.7–1.4
ACPA-positive cases	317 (98.5)	5 (1.5)	1.0	0.3–2.8	748 (92.8)	58 (7.2)	1.0	0.7–1.4
ACPA-negative cases	260 (100)	0 (0)	NA	NA	379 (93.4)	27 (6.6)	0.9	0.5–1.5

* Smoking information was available for 1,958 cases and 2,230 controls of the 1,962 cases and 2,247 controls. RA = rheumatoid arthritis; ACPA = anti–citrullinated protein/peptide antibody; EIRA = Epidemiological Investigation of Rheumatoid Arthritis; OR = odds ratio; 95% CI = 95% confidence interval; Ref. = reference; NA = not applicable.
† OR adjusted for age, sex, residential area, and alcohol consumption.
‡ OR adjusted for age, sex, residential area, pack-years of smoking, and alcohol consumption.

Table 3. Association of moist snuff use and the development of RA overall, ACPA-positive RA, and ACPA-negative RA among never smokers together with non–regular smokers and former smokers with a long period of smoking cessation in the EIRA (Sweden, 1996–2006)*

Subjects	Moist snuff use		OR†	95% CI
	No, no. (%)	Yes, no. (%)		
Ever user				
Controls	1,223 (91.1)	119 (8.9)	1.0	Ref.
RA overall	887 (92.6)	71 (7.4)	0.9	0.7–1.3
ACPA-positive cases	503 (92.6)	40 (7.4)	0.9	0.6–1.4
ACPA-negative cases	384 (92.5)	31 (7.5)	1.0	0.7–1.6
Current user				
Controls	1,223 (94.2)	76 (5.8)	1.0	Ref.
RA overall	887 (94.8)	49 (5.2)	1.0	0.7–1.5
ACPA-positive cases	503 (95.3)	25 (4.7)	0.9	0.5–1.4
ACPA-negative cases	384 (94.1)	24 (5.9)	1.3	0.8–2.1
Former user				
Controls	1,223 (96.6)	43 (3.4)	1.0	Ref.
RA overall	887 (97.6)	22 (2.4)	0.8	0.5–1.4
ACPA-positive cases	503 (97.1)	15 (2.9)	1.0	0.5–1.8
ACPA-negative cases	384 (98.2)	7 (1.8)	0.6	0.3–1.4

* RA = rheumatoid arthritis; ACPA = anti–citrullinated protein/peptide antibody; EIRA = Epidemiological Investigation of Rheumatoid Arthritis; OR = odds ratio; 95% CI = 95% confidence interval; Ref. = reference.
† OR adjusted for age, sex, residential area, and alcohol consumption.

and the risk of RA (3). Again, we observed no significant association between current or former snuff use and RA overall for either ACPA-positive or for ACPA-negative RA subsets (Table 3).

Finally, we performed the above mentioned association analyses (Tables 1, 2, and 3) stratified by sex. No notable differences were found for results from separate analyses in males and females, as compared with results from both sexes combined.

Discussion

Our main finding is that snuff use (either current or former) was not significantly associated with risk of developing ACPA-positive or ACPA-negative RA. Furthermore, snuff use was not related to these 2 subgroups of disease, neither among ever smokers or among never smokers.

Our study had several strengths. We used a population-based, case–control study design, enrolling incident RA cases. The high participation proportion among both cases and controls limited the magnitude of potential selection bias. Furthermore, data from more than 4,000 subjects allowed us to perform analysis stratified by smoking status. With this analysis strategy we were able to study snuff use among the "clean" group of never smokers, therefore minimizing the confounding, which might be caused by smoking. In addition, this study is the first to use a population-based approach for effects of moist snuff on risk of RA. However, several limitations need to be acknowledged. First, the lack of information on intensity of snuff use (parcel/day) hampered us from evaluating potential dose-response effects. Furthermore, with retrospective information on exposure, recall bias might be introduced if the cases report their exposures differently from the controls; but since we only ask the participants about current/past use, we regard this potential bias to be of minor magnitude in our study. Finally, with our current sample size and exposure frequency, we can detect an OR for RA overall in the order of 0.76 (or 1.28) with 80% power. The subsequent stratified analysis by smoking and ACPA status further reduced the sample size. Due to a relatively low number of exposed cases, especially among women, we were also hampered in drawing any conclusions regarding potential sex differences. Therefore, even though our study, to the best of our knowledge, is the largest study performed to date (in terms of number of cases) examining snuff use and RA risk, we are not able to rule out a moderate protective (or harmful) effect of snuff on the risk of developing RA.

Among the limited numbers of epidemiologic investigations regarding snuff use and risk of inflammatory diseases available so far, no association between snuff use and the risk of RA, Crohn's disease, or ulcerative colitis (UC) have been reported (12). However, a long duration of snuff use has been observed to be associated with a decreased risk of multiple sclerosis (14). A lower disease activity of RA among snuff users compared with smokers has been reported (15). Nicotine is suggested to have antiinflammatory effects in mouse models of RA (16,17), as well as being an effective treatment in experimental animal models of UC (18) and sepsis (19). A recent study using fibroblast-like synoviocytes from RA patients indicated that nicotine suppressed tumor necrosis factor α–induced production of interleukin-6 (IL-6) and IL-8 in a dose-dependent manner, as well as suppressed the activation of the proinflammatory mediator-associated NF-κB signaling pathway (20). Based on these observations, we consider it theoretically possible that nicotine also has an antiinflammatory effect in human RA. Our study provides the best epidemiologic

evidence so far that such an effect is at most limited, although we are unable to exclude moderate effects, in particular of larger doses of nicotine.

To summarize, we observed no association between snuff use and the risk of ACPA-positive or ACPA-negative RA. We conclude that other inhaled constituents of tobacco smoke than nicotine are more likely to be involved in the pathogenesis of ACPA-positive RA. Studies with larger sample size are warranted to verify our findings.

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AUTHOR CONTRIBUTIONS

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be submitted for publication. Ms Jiang had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study conception and design. Jiang, Alfredsson, Klareskog, Bengtsson.

Acquisition of data. Jiang, Alfredsson, Klareskog.

Analysis and interpretation of data. Jiang, Alfredsson, Klareskog, Bengtsson.

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APPENDIX A: MEMBERS OF THE SWEDISH EPIDEMIOLOGICAL INVESTIGATION OF RHEUMATOID ARTHRITIS STUDY GROUP

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