Tobacco smoking and hand eczema – is there an association?

Jennifer A. Sørensen¹, Kim K. Clemmensen¹, Rosemary L. Nixon², Thomas L. Diepgen³ and Tove Agner¹

¹Department of Dermatology, University of Copenhagen, Bispebjerg Hospital, 2400 Copenhagen NV, Denmark, ²Occupational Dermatology Research and Education Centre, Skin and Cancer Foundation Inc., 3053 Melbourne, Australia, and ³Department of Social Medicine, Occupational and Environmental Dermatology, University Hospital, D-69115, Heidelberg, Germany

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Summary

Background. Numerous risk factors have been suggested for hand eczema. This systematic review evaluates the association between tobacco smoking and hand eczema.

Objective. To review the literature systematically on the association between smoking and hand eczema.

Methods. The PubMed and EMBASE databases were searched up to 27 January 2015 for articles on the association between tobacco smoking and hand eczema, including human studies in English and German only. Experimental studies, studies on tobacco allergy, case reports, reviews and studies on second-hand smoking were excluded.

Results. Twenty articles were included. Among studies in occupational settings, three of seven found a statistically significant positive association between tobacco smoking and hand eczema prevalence rate, as did four of eight population-based studies. The association was stronger for studies in occupational settings than for population-based studies. No studies reported tobacco to be a clear protective factor for hand eczema. Two of five studies regarding severity found a positive association between smoking and hand eczema severity.

Conclusion. Overall, the data indicate that smoking may cause an increased frequency of hand eczema, particularly in high-risk occupations. However, data from studies controlling for other risk factors are conflicting, and few prospective studies are available. Studies controlling for other risk factors are needed, and information regarding the diagnosis of subclasses of hand eczema, as well as severity, may be important.

Key words: hand eczema; review; tobacco smoking.

Hand eczema (HE) is a frequent disease in the general population (1, 2), and is even more frequent in people in high-risk occupational settings (3, 4), such as hairdressers, bakers, and dental technicians. Not only is HE a common disease, but it also often takes a chronic course (4, 5). HE is a multifactorial disease, for which both environmental factors and individual endogenous factors play a role in the pathogenesis (6). It is well known that a history of atopic dermatitis (1, 2, 7) and wet work (1) are significant risk factors for HE, as is contact sensitization (8). The higher rate of HE in females reported in most studies has been shown to be attributable to a difference in skin exposure (more wet work) rather than female sex itself (9). Genetics, other than the predisposition relating to atopic dermatitis, have also been suggested to play a role in the development of HE (10). One study investigated a loss-of-function mutation in the filaggrin gene, and found that the combination of filaggrin gene mutation and atopic dermatitis is a greater risk factor for HE than atopic dermatitis alone (11).
HE is known to be a chronic disease, and inflammation plays a role in this, but the exact mechanisms are yet to be clarified. Smoking increases the levels of proinflammatory agents, such as acute-phase proteins, proinflammatory cytokines, particularly tumour necrosis factor (TNF)-α, TNF-α receptors and interleukin-6 in the body (12). Smoking not only induces inflammation, but also delays wound healing, partly because it decreases cutaneous blood flow and causes chronic damage to the microcirculation, thus compromising tissue oxygenation. In addition, fibroblast migration into lesional skin is inhibited (13, 14). Smoking has been suspected to be a risk factor for skin diseases such as lupus erythematosus (15, 16), palmoplantar pustulosis (17, 18), psoriasis (19-21), hidradenitis suppurativa and palmoplantar pustulosis, the data have indicated that the cessation of smoking may improve the disease (22, 23).

HE may be disabling and has widespread consequences, such as job loss and impaired quality of life (24, 25). It is a multifactorial disease, and it is important for patients with HE to know how lifestyle factors may impact on their disease. A high number of smokers among HE patients was initially reported almost 30 years ago (26), and this stimulated further studies on the topic. The aim of this study was to review the literature on the association between smoking and HE.

Methods

A literature search was performed in the PubMed database up to 27 January 2015, with the following search criteria: (((smoking and HE))) OR ((Smoking and contact dermatitis))) OR ((Tobacco and contact dermatitis))) OR ((Tobacco and HE))), (19)). One hundred and seventy-seven studies were identified. Studies in English or German, and human studies only, were included. Studies on tobacco allergy, case studies, reviews and second-hand smoking and laboratory studies were excluded, leaving 19 articles. The EMBASE database was also searched with the above-listed criteria, and one additional article was identified, leaving 20 articles for analysis in this systematic review. Furthermore, the article reference lists were searched for articles not found in the PubMed and EMBASE searches, but this did not result in the addition of any further articles.

Results

Results of studies on HE and tobacco smoking in occupational settings

There were seven articles in this category (two cohort studies, four case-control studies, and one cross-sectional study). Three studies found a statistically significant positive association between tobacco smoking and HE with respect to prevalence (27–29), and one found no clear association, but a slightly increased risk of HE in heavy smokers in one subgroup, along with a potential protective association in another subgroup (30). Three studies found no association between tobacco smoking and HE prevalence (31–33), but, in one of these, smoking was linked to severe eczema (32), and in another non-smoking was linked to more severe eczema (33). For information regarding adjustment for risk factors in each of the articles, see Table 1.

Kütting et al. (27) studied 1355 metalworkers, mostly males, and found statistically significantly higher scores for erythematous skin lesions on both hands in smokers than in non-smokers at baseline (p = 0.016). In a 1-year follow-up, which included 800 male metalworkers, significantly more smokers than non-smokers reported episodes of dyshidrotic HE (p = 0.027). Furthermore, smokers reported more dermatological consultations and disability caused by HE, but this was not statistically significant.

In a cross-sectional case–control study (28) comprising 87 car repair workers (cases) and 76 booksellers (controls), the self-reported HE prevalence among car repair workers was almost three times higher than among booksellers, and a significant positive association was found between the prevalence of dermatitis and smoking in car repair workers [odds ratio (OR): 3.59; 95% confidence interval (CI): 1.0–13.5]. In almost all cases, dermatitis was localized to the hands, but a few cases of eczema elsewhere were also included in the analysis.

In a study including 158 machine operators and 51 assemblers from a large automobile transmission plant, a significant positive association was found between current smoking and dermatitis (29). Smokers were three times as likely (OR: 3.10; 95% CI: 1.34–7.18) to have dermatitis located on any part of the hands, wrist or forearm than ex-smokers or non-smokers.

Berndt et al. (31) performed a prospective cohort study including 201 male metalworking trainees, followed for 2.5 years from the beginning of their apprenticeship. During the observation period, 47 (23%) of the young men developed at least an early stage of HE. No significant association was found between HE and number of cigarettes smoked per day.

A case–control study (32) included 230 medical laboratory technologists, 122 who had a skin reaction to latex gloves (cases), and 108 who had no problems with gloves (controls). In this study, there was no significant correlation between tobacco smoking and the presence or absence of dermatitis, but, in the ‘case’ group, smoking...
### Table 1. Articles on tobacco and hand eczema (HE) in occupational settings by publication year

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>Study type</th>
<th>n</th>
<th>Smoking habits</th>
<th>HE diagnosis</th>
<th>Association between HE prevalence and smoking</th>
<th>Adjusted for risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salkie (32)</td>
<td>1993</td>
<td>Canada</td>
<td>Case–control</td>
<td>230</td>
<td>Yes/no</td>
<td>SR</td>
<td>No</td>
<td>Univariate analysis</td>
</tr>
<tr>
<td>Sprince et al. (29)</td>
<td>1996</td>
<td>USA</td>
<td>Cross-sectional</td>
<td>209</td>
<td>Yes/no + amount</td>
<td>SR and DE</td>
<td>Yes OR: 3.10; 95% CI: 1.34–7.18</td>
<td>Multivariate analysis</td>
</tr>
<tr>
<td>Berndt et al. (31)</td>
<td>2000</td>
<td>Switzerland and Germany</td>
<td>Cohort</td>
<td>201</td>
<td>Yes/no + amount</td>
<td>OH</td>
<td>No</td>
<td>Multivariate analysis</td>
</tr>
<tr>
<td>Attwa and El-Laithy (28)</td>
<td>2008</td>
<td>Egypt</td>
<td>Case–control</td>
<td>163</td>
<td>Yes/no</td>
<td>DE</td>
<td>Yes OR: 3.59; 95% CI: 1.0–13.5</td>
<td>Univariate analysis</td>
</tr>
<tr>
<td>Meding et al. (30)</td>
<td>2009</td>
<td>Sweden</td>
<td>Case–control</td>
<td>13452</td>
<td>Yes/no + amount</td>
<td>SR</td>
<td>Overall: no (p = 0.51) Heavy smoking in hairdressers: yes (p &lt; 0.01) Protective effect in bakers: (p &lt; 0.05)</td>
<td>Multivariate analysis</td>
</tr>
<tr>
<td>Kütting et al. (27)</td>
<td>2011</td>
<td>Germany</td>
<td>Cohort</td>
<td>1355</td>
<td>Yes/no</td>
<td>DE</td>
<td>Yes Significantly higher score for erythematous lesions and vesicles (p = 0.027)</td>
<td>Univariate analysis</td>
</tr>
<tr>
<td>Patruno et al. (33)</td>
<td>2014</td>
<td>Italy</td>
<td>Case–control</td>
<td>516</td>
<td>Yes/no + amount + duration</td>
<td>DE</td>
<td>No association between HE prevalence and smoking. (p = 0.859) Increased severity in non-smoking housewives (p &lt; 0.05)</td>
<td>Univariate analysis</td>
</tr>
</tbody>
</table>

CI, confidence interval; DE, dermatologist examination; OH, other healthcare personal examination; OR, odds ratio; SR, self-reported HE.
was statistically significantly linked to more severe dermatitis ($p < 0.005$).

Patruno et al. (33) conducted an observational study analysing 516 housewives, 41.5% of whom were affected by chronic HE (cases), and 58.5% of whom were not affected (controls). In the HE group, 33.6% were smokers; in the control group, 35.1% were smokers ($p = 0.589$). HE was found to be significantly more severe in the non-smoking group than in the smoking group ($p < 0.05$).

In a study by Meding et al. (30), data was gathered from three cross-sectional questionnaire studies on bakers, dental technicians, and hairdressers. A total of 13 452 individuals were included. Of 3493 smokers, 437 (12.5%) reported HE in the previous year, as compared with 1294 of 9959 non-smokers (13.0%) ($p = 0.51$), so this study found no association between the 1-year prevalence of HE and smoking. One of the substudies (on hairdressers) included information about the number of cigarettes smoked. Of the hairdressers (cases) smoking >10 cigarettes per day, 22.6% reported HE, as compared with 17.4% of those smoking 0–10 cigarettes per day ($p = 0.01$). Corresponding figures for the controls were 14.5% and 11.7%, respectively ($p = 0.06$). The data suggested that heavy smoking, >10 cigarettes per day, might influence HE prevalence. However, data from another of the included subgroups (the bakers) showed a protective association between smoking and HE [prevalence proportion ratio (PPR): 0.67 (95%CI: 0.49–0.92)] ($p < 0.05$).

Results of studies on HE and tobacco smoking in population-based surveys

There were eight articles in this category (one cohort study; seven cross-sectional studies). Four studies found a positive association, or at least a trend for an association, between tobacco smoking and increased HE prevalence (34–37), whereas another four studies found no association between tobacco smoking and HE (9, 38–40). For information regarding adjustment for other risk factors, see Table 2.

The results from a cross-sectional public health questionnaire study including 27 793 individuals (34) showed a slightly increased HE prevalence among smokers [PPR: 1.025 (95%CI: 1.006–1.044)]. Interestingly, HE was found to be significantly more frequent in individuals who reported stress and obesity, and it was concluded that regular physical exercise may possibly have a protective effect against HE.

In a cross-sectional population study including 3471 individuals (44% of invited participants), a significant positive association between smoking and HE was reported ($p = 0.005$) (35). The study showed that current light smokers had a higher HE prevalence than current heavy smokers and previous smokers.

A questionnaire study including 9316 individuals (36) (response rate of 78.1%) used multiple logistic regression analysis, and found that smoking was an independent risk factor for an increased 1-year prevalence of HE [OR 1.35 (95%CI: 1.04–1.75)].

Meding et al. (37) studied self-reported HE and smoking in a cross-sectional questionnaire study of 25 428 individuals (response rate of 59%). The PPR for HE among individuals smoking >15 cigarettes a day was 1.40 (95%CI: 1.15–1.71), with never smokers as a reference in the multivariate analysis. Furthermore, a positive dose–response relationship between level of smoking and the 1-year prevalence of HE was observed, indicating a significantly increased prevalence of HE among individuals with higher consumption of tobacco.

In a cross-sectional questionnaire study (38) including 7543 upper secondary school children, no significant association between smoking and the 1-year prevalence of HE was found ($p = 1.00$). The response rate was 81%, and of these responders, 4376 were willing to participate and were included in the analysis.

Bo et al. (39) conducted another cross-sectional questionnaire-based study including 18 747 adults (response rate of 45.9%). The study showed no association between smoking and HE. The study did, however, show a positive association between smoking and psoriasis.

A prospective follow-up study of a population-based twin cohort determined the incidence rate of HE, and also investigated the role of genetic factors and other potential risk factors (9). The study found no significant association between HE and smoking ($p = 0.660$).

In a cross-sectional study including 771 adults, from an adolescent cohort followed for atopic dermatitis (40), no association between HE and smoking was reported [OR: 1.4 (95%CI: 0.9–2.1)].

Results of studies on the effect of tobacco smoking on HE severity

There were four articles in this category (two cohort and two case–control studies; Table 3). Two studies found a positive association between severity and smoking (32, 41); one of these was performed in an occupational setting, and the results are reported above (32). One study found no association between tobacco smoking and HE severity (42), and one study found that HE was more severe in non-smoking housewives (33). Two of these studies concerning severity were from occupational settings, and are therefore listed in Table 1 as well as in
Table 2. Population-based studies on tobacco smoking and hand eczema (HE) prevalence by publication year

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>Study type</th>
<th>n</th>
<th>Smoking habits</th>
<th>HE diagnosis</th>
<th>Association between HE and smoking</th>
<th>Adjusted for risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Montnemery et al. (36)</td>
<td>2005</td>
<td>Sweden</td>
<td>Cross-sectional</td>
<td>9316</td>
<td>&gt; five cigarettes daily or no</td>
<td>SR</td>
<td>Yes OR: 1.35; 95% CI: 1.04–1.75 (p = 0.022)</td>
<td>Multivariate analysis including age, sex, asthma, nasal symptoms, and occupation</td>
</tr>
<tr>
<td>Lerbaek et al. (9)</td>
<td>2007</td>
<td>Denmark</td>
<td>Cohort</td>
<td>4128</td>
<td>Yes/no + previous + amount ever smoked</td>
<td>SR</td>
<td>No (p = 0.66)</td>
<td>Multivariate analysis including sex, age, atopic dermatitis, contact allergy, and wet work</td>
</tr>
<tr>
<td>Bø et al. (39)</td>
<td>2008</td>
<td>Norway</td>
<td>Cross-sectional</td>
<td>18747</td>
<td>Yes/no + previous</td>
<td>SR</td>
<td>No statistically significant association</td>
<td>Multivariate analysis including mental distress, income, education, physical activity, alcohol, and BMI</td>
</tr>
<tr>
<td>Röhrli and Stenberg (38)</td>
<td>2010</td>
<td>Sweden</td>
<td>Cross-sectional</td>
<td>4376</td>
<td>Yes/no</td>
<td>SR</td>
<td>No (p = 1.0)</td>
<td>Multivariate analysis including atopic dermatitis, nickel allergy, and sex</td>
</tr>
<tr>
<td>Thysen et al. (35)</td>
<td>2010</td>
<td>Denmark</td>
<td>Cross-sectional</td>
<td>3471</td>
<td>Yes/no + amount + occasional, previous</td>
<td>SR</td>
<td>Yes Light smoking: OR: 1.51; 95% CI: 1.14–2.02 Heavy smoking: OR: 1.38; 95% CI: 0.99–1.9 Previous smoking: OR: 1.13; 95% CI: 0.90–1.40</td>
<td>Multivariate analysis including sex, age, atopic dermatitis, contact allergy, alcohol, and educational level</td>
</tr>
<tr>
<td>Meding et al. (37)</td>
<td>2010</td>
<td>Sweden</td>
<td>Cross-sectional</td>
<td>25428</td>
<td>Yes/no + amount + occasional, previous</td>
<td>SR</td>
<td>Yes Heavy smoking: PPR: 1.40; 95% CI: 1.15–1.71 Dose–response relationship between level of smoking and 1-year prevalence of HE (p&lt;0.001)</td>
<td>Multivariate analysis including age, history of atopy, sex, and wet work</td>
</tr>
<tr>
<td>Anveden Berglind et al. (34)</td>
<td>2011</td>
<td>Sweden</td>
<td>Cross-sectional</td>
<td>27793</td>
<td>Yes/no</td>
<td>SR</td>
<td>Yes PPR: 1.025 95% CI: 1.006–1.044</td>
<td>Multivariate analysis including physical exercise, alcohol, stress, obesity, depression, and wet work</td>
</tr>
<tr>
<td>Mortz et al. (40)</td>
<td>2014</td>
<td>Denmark</td>
<td>Cross-sectional within a cohort</td>
<td>771</td>
<td>Yes/no</td>
<td>SR and DE</td>
<td>No OR: 1.4; 95% CI: 0.9–2.1 (p = 0.13)</td>
<td>Multivariate analysis including atopic dermatitis, nickel sensitization, wet work, taking care of children, sick leave/disability pension/rehabilitation, smoking, education level, and sex</td>
</tr>
</tbody>
</table>

BMI, body mass index; CI, confidence interval; DE, dermatologist examination; OR, odds ratio; PPR, prevalence proportion ratio; SR, self-reported HE.
Brans et al. (41) recently presented data from a prospective cohort study on 1608 patients with occupational HE, followed up after 3, 6 and 10 weeks, respectively. HE severity, as measured with the Osnabrück Hand Eczema Severity index, was statistically significantly greater in smokers than in non-smokers at all times.

In a Danish study from private dermatology practice (42), HE severity was assessed in 522 consecutive patients by use of a severity scoring system ranging from 0 to 3, and including parameters such as pruritus, erythema, scaling, vesicles, fissures, and area. Two hundred and twenty-four of the included patients were smokers. No statistically significant association was found between smoking and HE severity, between palmar or dorsal location of dermatitis, or between smoking and long-standing eczema.

Results of other studies

In Table 4, three additional studies are presented: two cross-sectional in design, and one case–control study. The three studies were performed on patch tested patients (43), on patients with chronic HE (44), and on patients with HE (45). For information regarding the correction for other risk factors, see Table 4.

Edman (43) performed a study based on 425 consecutively patch tested patients. The study showed a positive association between the prevalence of vesicular HE and smoking in males. Of 45 smoking males 46.7% had vesicular HE, and of 63 non-smoking males, 27% had vesicular HE ($p = 0.029$).

Molin et al. (44) performed a study on 153 patients with chronic HE. This showed that significantly more smokers than non-smokers suffered from combined allergic and irritant HE ($p < 0.05$). Furthermore, there was a tendency for smokers to have nickel sensitization and palmar hyperhidrosis more frequently than non-smokers, although the differences were not statistically significant.

In a case–control study by Weigl and Wildner (45), data on smoking were obtained from 124 HE patients. A slight trend towards a positive association between smoking and vesicular HE as compared with other subtypes of HE was found; however, the difference was not statistically significant [OR: 1.14 (95%CI: 0.43–3.02)] ($p = 0.797$).

Discussion

The results from studies on HE and tobacco smoking are inconsistent, as approximately half of the included studies show an increased prevalence and/or severity of HE.
Table 4. Articles on other topics, such as the association between tobacco and sub-classification of hand eczema (HE), by publication year

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>Study type</th>
<th>n</th>
<th>HE diagnosis</th>
<th>Multivariate or univariate analysis adjusted for risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edman (43)</td>
<td>1988</td>
<td>Sweden</td>
<td>Cross-sectional</td>
<td>425 patch test patients</td>
<td>Yes/no</td>
<td>Positive association with palmar eczema: OR: 2.4 (p = 0.029)</td>
</tr>
<tr>
<td>Weigl and Wildner (45)</td>
<td>2011</td>
<td>Germany</td>
<td>Case–control</td>
<td>124 patients with HE</td>
<td>Yes/no</td>
<td>OR: 1.14 (95% CI: 0.43–3.02) (p = 0.797)</td>
</tr>
<tr>
<td>Molin et al. (44)</td>
<td>2014</td>
<td>Germany</td>
<td>Cross-sectional</td>
<td>153 patients with chronic HE</td>
<td>Current, previously, or never</td>
<td>Positive association with combined allergic and irritant hand eczema (p &lt; 0.05)</td>
</tr>
</tbody>
</table>

CI, confidence interval; DE, dermatologist examination; OR, odds ratio; SR, self-reported hand eczema.

In smokers, whereas the other half report no association. However, a protective effect of tobacco on HE prevalence was reported in only one study and in a subpopulation of patients only (Table 1). Of the 15 studies examining HE prevalence in relation to smoking, in either occupational settings or in a population-based context (Tables 1 and 2), seven studies found a positive association (27–29, 34–37), whereas five studies found no association (30, 31). The relationship between hand eczema severity and smoking was evaluated in five studies only (28, 34–36, 41), of which one found a positive association between smoking and smoking was found to be more severe in smokers than in non-smokers (32, 41). One study (30) showed conflicting data: no significant association was found between smoking and the 1-year prevalence of HE in the total group, but in one subgroup (hairdressers), heavy smoking was a risk factor for HE, whereas a protective association of smoking was found for HE in another subgroup (bakers). Among the studies that found no association (42–45), one reported a protective association of smoking with erythematous lesions and vesicles (27).
rates varied from 44% to 84%. The studies that included the two largest populations, 25,428 individuals (37) and 27,793 individuals (34), found a positive association between smoking and HE prevalence, and two studies (27, 36) with a high response rate (~78%) showed significantly positive associations between smoking and HE prevalence. Often, people from the lower social strata are overly represented in the non-responding group (46). Within this social group, we know that there are more smokers and more blue-collar workers, so if there is an association between smoking and HE, it could be underestimated, as lack of smoking in the (smaller) studies with low response rates could lead to a lack of power to detect an association between smoking and HE.

Ten studies were cross-sectional studies (29, 34–40, 43, 44), in which it is not possible to draw conclusions on causality, for which prospective cohort studies are required (9, 27, 31, 41, 42). The follow-up period in the cohort studies ranged from 1 to 9 years, and loss to follow-up, which can cause significant bias, varied in the studies from 0% to 51%.

An interesting observation is that the occupational studies that reported a positive association between HE prevalence and smoking had much higher ORs than population studies [OR > 3 in occupational studies and PPR/OR < 2 in population-based studies, respectively (Tables 1 and 2)]. This raises the question of whether smoking in high-risk occupations is a greater risk factor for the development of HE than smoking in low-risk occupations. Smoking causing a delay in the restoration of the broken skin barrier in individuals with HE could possibly explain this.

Only one study included information about the time of year when the study was conducted (29). HE is usually worse during the winter, and it would be interesting to evaluate whether winter combined with smoking is a greater risk factor for HE than smoking during the other seasons.

Contact sensitization is a well-established risk factor for HE (8), and an association between contact sensitization and smoking has also been suggested (47–49). One study reported that smoking was a risk factor for contact sensitization in women (47), another study found that smoking was a risk factor for nickel sensitization (48), and one study reported an association between >15 pack years and the prevalence rates of contact allergy and allergic nickel contact dermatitis, and also showed a dose–response relationship, with a higher OR in the group with the highest amount of pack years (49). Although these data are rather convincing, more studies are needed to allow firm conclusions on smoking and contact sensitization to be drawn. It is also possible that smoking provides systemic exposure to some allergens (50).

Factors such as female sex, atopy and wet work are well known risk factors for HE. However, only seven studies included all of the above listed risk factors (9, 31, 35, 37, 38, 40, 45) and performed a multivariate analysis controlling for these possible confounders. In a further three studies (28, 41, 44), risk factors were controlled for in other ways. Among these controlled studies, a positive association between smoking and HE prevalence was reported in one (28), whereas five (9, 28, 38, 40, 45) found no significant association. A higher positive association with light than with heavy smoking was reported in one controlled study (35), whereas one study reported a positive association with heavy smoking only (37). With respect to smoking and HE severity, a positive association was confirmed in one controlled study (41).

In all of the included studies, data on smoking were self-reported, as this is the only feasible way to gather this information. A possible bias related to self-reported smoking could be a tendency for occasional smokers to state that they are non-smokers. Preferably, questions concerning smoking should involve the statements ‘occasionally’ and ‘previously’, and the amount that a person smokes per day is also important, in order to explore a possible dose–response relationship. Some studies only asked whether the participants smoked or not (27, 28, 30, 32, 34, 36, 38, 40, 42, 43, 45), some added the term occasionally and/or previously (9, 29, 35, 37, 39, 44), and some added a question on the amount of smoking (9, 29–31, 33, 35, 37, 41). Only one study took pack years into consideration (9). To fully understand smoking habits, information on pack years is important, because smoking patterns in the individual can change but might still have an influence on the risk of HE.

Self-reported HE was usually obtained by an answer to the question ‘have you had HE on any occasion during the past 12 months?’ or a question with similar wording (30). This question has proven to have high specificity (96–99%), but lower sensitivity (53–59%) (51). This would lead to an underestimation of the true HE prevalence in the studies based on self-reporting. Among the included studies, HE was self-reported in nine (9, 30, 32, 34–39), whereas in the other 11, HE was diagnosed by a dermatologist or another healthcare professional (27–29, 31, 33, 40–45).

Among the studies in which HE was self-reported, four of nine showed a positive association (34–37), and among the studies in which HE was diagnosed by a dermatologist or another healthcare professional, 6 of 11 showed a positive association (27–29, 41, 43, 44).
is a tendency for studies including a clinical examination to more often show a positive association than studies in which HE was self-diagnosed. This could be partly attributable to the possible underestimation of the prevalence, because of the low sensitivity of the question asked, as this could lead to a weakening of the association.

This review has some limitations, as only English-language and German-language articles were included. However, the articles included were searched for further references, and none were found.

Future studies exploring this association should be designed as prospective cohort studies, on which a possible effect of smoking on the prevalence and severity of HE could be better assessed. Subclassification and determination of severity of HE, including dermatologist examination rather than self-reporting, should be performed, a detailed smoking history should be obtained, and information on possible confounding factors and effect modifiers should be included to ensure the validity of the results.

Conclusion

Most studies reported either an increased HE prevalence in smokers or no significant association, and a protective effect of smoking on HE was reported in a subgroup of patients in one study only. This indicates a positive association, and differences between population-based studies and studies in occupational settings point to a possible association between HE and smoking particularly in occupations with a high risk for HE. However, data from studies controlling for other risk factors for HE are conflicting: there are few prospective studies available, and studies regarding the associations of different subclasses of HE and severity of HE are few. Future studies including these important parameters are needed.

References

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