The idea that this devastating skin disease may have influenced to some degree his revolutionary thoughts is rather intriguing. We want to draw attention to another aspect of this historical issue. Apparently, the skin disorder of the well-known founding father of the communist doctrine can be taken as an instructive example of a serious consequence of smoking, this particular sequel being neither of vascular nor neoplastic nature.

Marx was an excessive smoker of cigars from his student time until his death. Franz Mehring, a German publicist and politician, wrote in 1918 in a biography on Marx: 'Nor was he a great drinker, although he did not abstain completely, and as a true son of the Rhineland he appreciated a good drop of wine. On the other hand, he was a passionate smoker and a demon for matches. He was accustomed to say jokingly that his ‘Capital’ would not bring him in sufficient to pay for the cigars he had smoked whilst writing it. During the long years of poverty he undoubtedly had to put up with many inferior brands and, as a result, his passion for smoking certainly did his health no good, in fact, his doctor prohibited smoking on a number of occasions’. As a severe bronchitis was the reason for the repeated and fruitless orders to stop smoking.

In which way is HS statistically related to smoking? Breitkopf et al. found in a cohort of 55 men and 94 women with HS that 85% of men were smokers compared with an expected prevalence of 36% from regional statistics, and 84% of women were smokers compared with an expected prevalence of 23%. Similarly, we found in a questionnaire study including 63 patients with HS that 89% were active smokers vs. 46% in a matched-pair control group (P = 0.001), and we concluded that smoking is a major triggering factor of HS.

In search of a mechanism explaining this relationship, Hana et al. studied the effect of nicotine on an organotypically cultured epidermis equivalent. From their in vitro findings they concluded that nicotine may promote infundibular epithelial hyperplasia and thus follicular plugging.

We are aware of the fact that virtually all of the smoking HS patients included in the two questionnaire studies were addicted to cigarettes, whereas Marx smoked cigars. Smoking of cigarettes or cigars, however, harbours a comparable risk of addiction to nicotine, and the health damage is similar in addictive smokers. Hence, we think that Karl Marx’s skin disorder can be taken as a textbook case exemplifying the close relationship between smoking and HS. Needless to say that a genetic predisposition may be a prerequisite and that a small proportion of HS patients is nonsmoking.

So far, however, this concept is not generally accepted. In several more recent questionnaire studies on quality of life, patients with HS were not even asked whether they were smokers. In other articles on HS smoking is either ignored or brushed aside as a little worsening factor among other determinants such as obesity, irritation by deodorants, shaving, depilation, occlusion or maceration, or hidden in an even longer list of risk factors such as ‘history of acne, diabetes, hypercholesterolemia, low metabolic rate, and smoking and being obese, African American, or female’.

A lesson to be learned from Karl Marx: smoking triggers hidradenitis suppurativa

DOE: 10.1111/j.1365-2133.2008.08604.x

Sir, We read with great interest Sam Shuster’s excellent essay on Karl Marx’s chronic cutaneous illness. He convincingly argues that Marx suffered from hidradenitis suppurativa (HS).
Let us hope that Sam Shuster’s discovery of the true nature of Marx’s skin disease will help convince the scientific community that in most cases of HS, smoking is not just a little additive risk factor, but of crucial causative significance.

Department of Dermatology,
Philipp University of Marburg,
35033 Marburg, Germany
E-mail: happle@med.uni-marburg.de

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Key words: hidradenitis suppurativa, Karl Marx, smoking, tobacco

Conflicts of interest: none declared.

A lesson to be learned from Karl Marx: smoking triggers hidradenitis suppurativa: reply from author

DOI: 10.1111/j.1365-2133.2008.08605.x

Sn, Pleased though I am with the interest now being taken in Marx’s hidradenitis suppurativa (HS) and its possible effects on his work,¹ Happle and König go well beyond what is permissible from the evidence. All that has been found is that the percentage of hospital patients with HS who smoke is greater than that reported for the population at large²,³ and than in paired controls where the difference is much less. But this does not allow the presumption of causality: that requires a relationship to onset, offset and dose-response, none of which has been demonstrated (the study of nicotine in tissue culture⁴ is preliminary, unspecific and irrelevant). Of the 63 patients in the study by König et al.,⁵ only 32 started smoking before their HS (taking an average 10 long years to achieve its alleged malign effect); worse still, 10 patients were allowed to bulge the causal statistic, although they started smoking only after the onset of the HS! Surely, then, the reasonable conclusion must be that what ‘triggers’ HS is not so much the smoking, as the possibility that an individual may take to smoking in the future! While a smoking trigger makes an interesting variant on the smoking gun, the notion that smoking fires HS is a totally unsubstantiated allegation.

But if the general case disperses at first puff, it disappears completely in its specific application to Marx. Apart from the lack of evidence for a temporal or dose relationship, there is a failure to understand that the significance of Marx’s smoking must depend on the prevalence of smoking at the time, by people of his age, sex and social circle: if most of his confreres smoked, Marx’s smoking habit would have no statistical significance. From written accounts, Marx made tobacco available to his visitors, with the expectation that most, if not all, smoked; he characterized the meetings of his group of ‘socialist workers’ by their ‘smoking, eating and drinking’; Engels, Marx’s closest collaborator, wallowed in cigar smoke; so did Marx’s fellow journalists who worked in a ‘room concealed in such a thick cloud of tobacco smoke that it was impossible to see a newcomer...’. Nevertheless, despite the many accounts and anecdotes suggesting that most of Marx’s peer group were smokers, we don’t know its true prevalence, and cannot therefore play games of significance with the simple, single statistic of his being a smoker.

Unlike Happle and König I do not complain that the smoking trigger ‘concept is not generally accepted’: it is ignored for good reason, and must stay that way unless something more substantial is revealed behind their smoke screen. But I can only agree that this story is indeed a ‘textbook case’: but not of ‘the close relationship between smoking and HS’, of which there isn’t a whiff of evidence – but of the increasingly common misapplication of associative epidemiological statistics to aetiology. Causal mechanics is something it cannot do, and the frequent, inappropriate attempts serve only to produce the daily horror stories and lists of unsubstantiated associations that clog our journals and newspapers. Use of this bastard epidemiological substitute for science has spread like a cancer; it is doing much damage to genuine research, and has done much harm to the public’s belief in medical science. Old Marx was a tough thinker, very keen on a scientific rationalism, and he would have hated this development. But he also had a wicked sense of humour, and would have laughed it out of court – between bouts of his smoker’s cough.