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Key words: Cochrane Library, Cochrane Skin Group, e-publication, systematic reviews

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Supplementary material

The following supplementary material is available online for this article:
Appendix S1 List of journals of which the editors were mailed questionnaires
Appendix S2 Editor questionnaire

Appendix S3 Cochrane skin systematic review lead author questionnaire

This material is available as part of the online article from: http://www.blackwell-synergy.com/doi/abs/10.1111/j.1365-2133.2008.08552.x.

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The revolutionary consequences of skin disease

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Sir, Professor Shuster has suggested that Karl Marx (1818–1883) may have suffered from hidradenitis suppurativa.1 It is an interesting suggestion based on a plausible interpretation of the available data.

The recognition of dermatological diseases in the great and the famous may add insight into their personalities but may also add personal histories to the diseases. This occurs in the Arts where the knowledge that a given artist was suffering from a skin disease may provide for a better understanding of his or her works, but may also give insight into coping mechanisms of the creative patient.2 The stringently controlled linear design and use of subdued colours in Paul Klee’s (progressive systemic scleroderma) paintings may easily be interpreted to reflect the nature of his disease by the artistically uneducated amateur.

The occurrence of skin diseases in people involved with political power may be an even more interesting topic. Skin diseases have strong psychosocial aspects. Dermatological diseases not only affect the patient through itching or pain, but also affect the interaction between the patient and his or her peers through the emotional reactions they evoke in healthy people.3,4 Skin disease may therefore significantly contribute to the patients’ perception of social relations, and, in consequence, of society.

Professor Shuster’s essay has therefore brought attention to an interesting feature of dermatological disease. Off hand, it would appear that skin diseases have been a prominent feature in the lives of some of the most famous revolutionary figures of history. The eczema herpeticum of the Jacobin Jean-Paul Marat (1743–1793) may thus be speculated to have affected the radical policies of revolutionary France.5,6 The ideas of the French revolution in turn prepared the way for Karl Marx’s philosophical approach to the theme of societal development. Provokingly, it may further be speculated that the perceived stigmatization of Josef Stalin’s (1879–1953) psoriasis may subsequently have affected the execution, literal in all senses of the word, of Karl Marx’s communist ideas.7

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It is highly likely that a chronic recurrent disease such as hidradenitis suppurativa may have affected the life of Karl Marx profoundly and thereby significantly contributed to shaping his view of the world. The disease, which affects the hair follicles of inverse areas of the body surface, has a significant impact on the lives of patients: not only through suppuration and pain, but also through increased use of medical services, sick-days and a generally lowered quality of life.5–11 In Karl Marx’s case it may have affected the quality of life of millions of people, making it a unique disease in more ways than one.

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Are filaggrin mutations associated with hand eczema or contact allergy? – we do not know

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Sir, We were interested to read the recent report by Lerbaek et al. describing their investigation of the association between filaggrin loss-of-function mutations and hand eczema or contact dermatitis. The study addresses an important question: filaggrin is known to play a significant role in epidermal barrier function and filaggrin null mutations result in deficiency or absence of filaggrin in the skin,2 so do these mutations also predispose to hand eczema and allergic sensitization? However, unfortunately the study design – specifically the choice of controls and lack of statistical power – means that this study is unable to give an answer to the question. These limitations are acknowledged by the authors but are not reflected in the title or abstract of their paper.

Genetic association studies require careful phenotype definition and the matching of cases with an appropriate control population.3 This is particularly important for the investigation of a complex trait,4 where multiple genetic factors must be identified on a background of multiple environmental factors which all contribute to the pathogenesis of disease.

The large cohort of Danish twins with data on hand eczema is a valuable resource which could be well used to address the question under investigation. However, the authors’ choices of control populations are significantly flawed. The twins genotyped for this study had previously been selected on the basis that one or both of the twin pair had self-reported hand eczema, hence they cannot be presumed to represent an unaffected control group even if they do not have hand eczema at the current time. Lerbaek et al.1 go on to choose a second suboptimal control group: a selected cohort of children born to mothers with asthma. While established eczema cases were excluded, this still represents a rather unusual group in which ‘atopy genes’ (as well as environmental influences) are likely to be over-represented. Even the filaggrin null allele frequencies are higher than reported in some other population control groups.5,6

This study design contributes to a reduction in the statistical power because the controls include individuals with an over-representation of genetic factors associated with atopic eczema and hand eczema. Hence even the association of filaggrin null mutations with atopic eczema is not observed when comparing twins with and without atopic eczema, although this strong association has been demonstrated and replicated in all studies published to date.7 Similarly, as atopic eczema is one of the main risk factors for hand eczema8 the children of mothers with asthma are not an ideal control group, despite exclusion of the children who have so far developed eczema. Comparison of twins having contact allergy with this control group may be less inappropriate, as allergic contact dermatitis does not appear to share a common genetic predisposition with atopy or eczema. However, the number in this small subgroup (45 individuals) may be insufficient to demonstrate an association unless there was a particularly powerful genetic effect, which has not been predicted from previous studies.9,10

While acknowledging this lack of statistical power, as well as the suboptimal control groups, Lerbaek et al. proceed to conclude that ‘there is no association between the variant alleles and hand eczema or contact allergy’.1 This conclusion is misleading and is not supported by the presented data.

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