produce hepatotoxicity, especially when high doses are used. Moreover, antimalarials cannot be given for a long period of time because of their ocular toxicity especially in older patients. For this reason, low doses of chloroquine (125 mg twice weekly) have been recommended (3). In our patient, even 62.5 mg weekly proved enough to obtain a clinical remission. To the best of our knowledge, so low a dosage has never been reported previously.

REFERENCES

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Could Bacterial Acetaldehyde Production Explain the Deleterious Effect of Alcohol on Skin Diseases?

Sir,

Heavy alcohol consumption is known to be associated with the aggravation of certain skin diseases, like seborrheic and nummular dermatitis, psoriasis, acne and rosacea (1, 2). In contrast to the classic alcohol-associated stigmata skin may be afflicted as an early feature of alcohol misuse (1). So far the pathogenesis of these phenomena is not fully elucidated.

A number of bacteria and yeasts are known to possess alcohol dehydrogenase (ADH) activity (3). In the presence of excess ethanol these microbes produce reactive and toxic acetaldehyde (4, 5). Since ethanol concentrations of sweat are equal to those in blood (6), we wanted to study whether bacteria and fungi associated with pathological dermatological conditions contain ADH and produce acetaldehyde from ethanol.

Propionibacterium acnes, Staphylococcus aureus, Staphylococcus epidermidis, Streptococcus pyogenes and Pityrosporum ovale strains were isolated from outpatients at the Department of Dermatology, Helsinki University Central Hospital. The bacteria were identified with usual microbiological criteria. In addition, the identification was verified by analyzing the cell components gas chromatographically (MIDI, Sherlock, Barksdale, Newkirk, DE).

Cytoplasmic ADH activity was determined spectrophotometrically by measuring, after addition of ethanol, the reduction of nicotinic amide dinucleotide (NAD) to is reduced form (NADH) (5). Bacterial acetaldehyde production was determined by incubating bacterial suspensions in closed vials with 1% ethanol at pH 7.4, 37°C for 2 h. After the incubation, the acetaldehyde produced was determined using head space gas chromatography as described earlier (7).

Very high acetaldehyde levels up to 960 nmol/l were formed by the bacteria studied at ethanol concentrations known to exist in sweat during normal social drinking. The corresponding bacterial cytosols showed significant ADH activity both at low and high ethanol concentrations. The amount of acetaldehyde related to the number of bacteria (mean ± SE) after at least four separate determinations was: P. acnes (59 ± 23 nmol/10⁶), S. pyogenes (118 ± 15 nmol/10⁶), S. epidermidis (106 ± 10 nmol/10⁶) and S. aureus (160 ± 22 nmol/10⁶). The P. ovale fungi associated with seborrheic dermatitis, however, neither produced acetaldehyde (0.8 ± 0.2 nmol/10⁶) nor showed ADH activity. This primary observation of bacterial production of acetaldehyde – a highly toxic and reactive substance – could offer an explanation for the deleterious effect of alcohol on various skin diseases and warrants for further in vivo studies.

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REFERENCES

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