Rosengren, A.-M. & Rosengren, E.: Excretion of 5-S-cysteinyldopa in humans with genetically dark skin. Acta Dermatovener (Stockholm) *59:* 69, 1979.
4. Agrup, G., Agrup, P., Andersson, T., Hafström, L., Hansson, C., Jacobsson, S., Jönsson, P.-E., Rorsman, H., Rosengren, A.-M. & Rosengren, E.: 5 years' experience of 5-S-cysteinyldopa in melanoma diagnosis. Acta Dermatovener (Stockholm) *59:* 381, 1979.
5. Anton, Aa. & Sayre, D. F.: The distribution of dopamine and dopa in various animals and a method for their determination in diverse biological meterial. J Pharmacol Exp Ther *145:* 326, 1964.
6. Aubert, Ch., Rosengren, E., Rorsman, H., Rouge, F., Foa, C. & Lipcey, C.: 5-S-cysteinyldopa in diagnosis and treatment of human malignant melanomas and ultrastructual observations. Eur J Cancer *13:* 1299, 1977.
7. Rorsman, H., Rosengren, A.-M. & Rosengren, E.: A sensitive method for determination of 5-S-cysteinyldopa Acta Dermatovener (Stockholm) *53:* 248, 1973.

The Effect of Short-term Occlusion on the Cutaneous Flora in Atopic Dermatitis and Psoriasis

Georg Rajka,[1] Raza Aly, Charlene Bayles, Yajarayma Tang and Howard Maibach

[1]*Department of Dermatology, Rikshospitalet, Pilestredet 32, Oslo, Norway and Department of Dermatology, University of California, San Francisco, CA 94143, USA*

Received August 18, 1980

Abstract. The effect of occlusion on the cutaneous microbial flora in patients with atopic dermatitis and psoriasis was measured. Significant increases in the density of *Staphylococcus aureus* and lipophilic diphtheroids were noted after occlusion in eczematous and psoriatic skins. While the incidence of *S. aureus* increased in psoriatic skin after occlusion, this trend was not noted in dermatitic skin.

In atopic dermatitis, colonization with *Staphylococcus aureus* predominates in the involved skin and coagulase-negative staphylococci in the uninvolved skin (2). Coagulase-negative staphylococci dominate in psoriatic skin (6).

When reviewing studies on cutaneous microbial colonization, it is necessary to separate incidence (which may refer to few or many bacteria per patient) and quantity (the number of organisms per unit area, usually colony-forming units (CFU/cm^2)).

Many atopics carry *S. aureus* in their involved and putatively normal skin: the counts per unit area frequently reach 10^6 (CFU/cm^2) in involved skin and 10^5 in "normal skin". Among factors influencing experimental skin infection in man and animal, occlusion (yielding heat and moisture and altering pH), CO_2 emission rate, bacterial antagonism and skin surface lipids, are important (7).

Prolonged occlusion of normal skin increases microbial density and qualitatively alters the aerobic microflora (8, 9). No published data exist on the effect of short-term occlusion on the bacterial flora in pathological skin conditions. This study investigates the changes in the microflora of the skin in atopic dermatitis and psoriasis after short-term occlusion.

MATERIALS AND METHODS

Four male and 6 female outpatients with clinically well established atopic dermatitis and 9 with psoriasis were studied. We excluded patients receiving systemic and/or topical antibiotics for a minimum of a month. Several psoriatics received tar treatment, which was stopped 3 days before the study. All patients had active skin disease at investigation. Overtly inpetiginized patients were excluded. The sites for bacterial sampling were inflamed lesions, and adjacent intact (about 5 cm away from inflamed lesions) areas of the forearm.

Occlusion. The forearm was wrapped with three layers of vinylidine polymer plastic film (Saran Wrap, Dow Chemical, Midland, Michigan) tightly secured at the wrist and below the elbow with paper adhesive tape (Micropore, 3M, St. Paul, Minn.). Samples were taken by the detergent scrub technique (10). The surface was rubbed with a teflon "policeman" in 1 ml of wash solution (0.075 M phosphate buffer with 0.1% Triton X-100) for 1 min; the sample fluid was aspirated with a pipette and serially diluted in a 0.037 M phosphate buffer containing 0.05% detergent. Samples (0.1 ml) of varying dilutions were cultured on appropriate media. The bacteria were identified by Gram stain and appropriate biochemical tests, as described earlier (2).

RESULTS

Atopic dermatitis

Density. The mean lesional density of *S. aureus* increased from 2.3×10^6 to 2.2×10^9 (Table I). This increase was also noted on the adjacent normal skin (4.2×10^4 to 1.2×10^9/cm^2). On 2 patients, tiny pustules or crusts were seen after occlusion. Similar increases of coagulase-negative staphylococci were noted. Micrococci noted before occlusion were not detected after a 24-hour occlusion. Gram-negative

Table I. *Average microbial counts in patients with atopic eczema or psoriasis before and after 24 h occlusion*

Organism	Normal skin before occlusion	Normal skin after occlusion	Lesional skin before occlusion	Lesional skin after occlusion
Atopic eczema				
Staphylococcus aureus	6.76×10^{3}	1.90×10^{8}	3.79×10^{5}	3.56×10^{8}
Coagulase-negative staphylococci	3.24×10^{4}	4.33×10^{8}	9.46×10^{4}	4.37×10^{8}
Micrococci	3.02×10^{2}	0	9.76×10^{2}	0
Streptococci	0	0	0	0
Non-lipophilic diphtheroids	0	0	0	0
Lipophilic diphtheroids	2.65×10^{2}	2.28×10^{7}	12.2	8.62×10^{6}
Bacillus sp.	0.41	8.13×10^{5}	1.79	1.79×10^{6}
Gram-negative rods	6.50	3.41×10^{2}	0.98	7.32×10^{2}
Yeasts	0	0.49	0	0
Psoriasis				
S. aureus	2.18×10^{5}	2.63×10^{6}	3.64×10^{5}	5.17×10^{6}
Coag.-neg. staph.	9.48×10^{3}	5.12×10^{7}	9.15×10^{4}	5.06×10^{7}
Micrococci	1.04×10^{2}	1.21×10^{3}	3.707×10^{1}	2.76×10^{4}
Streptococci	0	0	0	0
Non-lipo. diph.	3.76×10^{1}	0	2.67×10^{2}	1.11×10^{7}
Lipo. diph.	1.05×10^{2}	2.19×10^{6}	3.63×10^{2}	1.93×10^{7}
Bacillus sp.	2.37×10^{1}	0	1.265×10^{1}	0
Gram-neg. rods	0.18	3.85×10^{1}	1.95	3.25×10^{2}
Yeasts	0	0	0	0

rods increased in numbers following occlusion. A dramatic increase of 3 to 4 logs of lipophilic diphtheroids occurred with occlusion. A substantial increase (4 logs) in the bacterial count was noted after occlusion.

Incidence. The carriage rate of *S. aureus* was the same before and after occlusion of the lesion (80%) and was slightly elevated (from 63% to 75%) on normal skin after occlusion (Table II). The incidence of coagulase-negative staphylococci de-

Table II. *Percent incidence of microorganisms in 10 patients with atopic eczema and 9 patients with psoriasis before and after 24 h occlusion*

Organism	Normal skin before occlusion	Normal skin after occlusion	Lesional skin before occlusion	Lesional skin after occlusion
Atopic eczema				
Staphylococcus aureus	63	75	80	80
Coagulase-negative staphylococci	100	88	90	70
Micrococci	63	0	20	0
Streptococci	0	0	0	0
Non-lipophilic diphtheroids	0	0	0	0
Lipophilic diphtheroids	13	25	10	20
Bacillus sp.	38	13	30	10
Gram-negative rods	25	50	20	40
Yeasts	0	13	0	0
Psoriasis				
S. aureus	44	89	33	89
Coag.-neg. staph.	89	100	100	100
Micrococci	44	22	22	11
Streptococci	0	0	0	0
Non-lipo. diph.	33	0	22	22
Lipo. diph.	33	22	22	22
Bacillus sp.	22	0	11	0
Gram-neg. rods	11	22	11	33
Yeasts	0	0	0	0

creased in both locations (on normal skin from 100% to 88%, and on lesions from 90% to 79%). Micrococci disappeared during the 24-hour occlusion; gram-negative rods increased slightly. The carriage rate for lipophilic diphtheroids remained slightly elevated on involved and uninvolved skin after occlusion. Streptococci and yeasts were not observed in the lesional skin.

Psoriasis

Density. The density of *S. aureus* did not change significantly on the uninvolved skin; in the involved skin one log increase (2.2×10^6 to 3.2×10^7/cm²) was noted after a 24-hour occlusion (Table I). Dramatic increases in the density of lipophilic diphtheroids were noted in the normal skin and in the plaques. Gram-negative rod counts rose.

Incidence. A significant carriage rate for *S. aureus* occurred in the plaques and the adjacent normal skin after occlusion (Table II). This change was from 33% to 89% in the plaques ($P=0.0531$) and 44% to 89% on the normal skin after occlusion ($P=0.1336$). Coagulase-negative staphylococci remained unchanged. Reverse trends were noted with micrococci, i.e. the incidence decreased with occlusion. Lipophilic diphtheroids and gram-negative rods also increased.

DISCUSSION

Short-term occlusion on normal skin

Earlier studies using one day's occlusion with plastic film showed an average 11.1 million/cm² bacteria on normal skin, compared with 3800/cm² control value (1). Aly et al. (9) found an increase from 1.8×10^2/cm² (before occlusion) to 1.4×10^6/cm² after 24 hours of occlusion. After 24 hours' occlusion, coagulase-negative staphylococci rose from 90% to 100%, micrococci from 30% to 70%, and non-lipophilic diphtheroids remained unchanged (50%) and gram-negative rods rose from 0 to 10%. Coagulase-positive staphylococci, beta hemolytic streptococci and *Pseudomonas aeruginosa* did not appear under occlusion.

Atopic dermatitis. Several authors emphasize the lesional *S. aureus* colonization in atopic dermatitis patients (1–3). Aly et al. (2) observed that *S. aureus* was the predominant organism; this was detected in 93% for lesions (mean density, 7.5×10^4/cm²) and constituted 91% of the total aerobic flora. Clinical infection was lacking in these patients. It has been suggested that a density of more than 1×10^7/cm² of *S. aureus* is required for clinical infection (13). The incidence of *S. aureus* in non-occluded uninvolved skin of atopics was 76%, with a mean density of 7.1×10^3/cm². On normal skin of these patients, coagulasenegative staphylococci predominated over *S. aureus*.

In the present study, the carriage rate of *S. aureus* did not change in the lesion, or increased slightly in uninvolved skin after occlusion. However, the average density of this organism rose to the range that might produce clinical disease (13). This was noted for both involved and uninvolved skin. Although the carriage rate for coagulase-negative staphylococci decreased during the occlusion, their average number increased significantly. Similarly, a 5 to 6 log increase in density was noted with lipophilic diphtheroids after 24 hours of occlusion in normal and dermatitic skin.

Psoriasis. The carriage rate of *S. aureus* was 50% in the lesions of outpatients (5). Noble & Savin (4) reported 44% of psoriatic inpatients yielded *S. aureus* from plaques. Their outpatients with psoriasis seldom demonstrated *S. aureus* on their skin (4). In a later study, the incidence of *S. aureus* was 20% (mean density: 3×10^2 per square cm) on the lesional skin, and 13% (average number 1.5×10 per square cm) on uninvolved skin (6). This quantitative difference was not statistically significant. The predominant organisms on the involved/uninvolved skin of psoriatics were coagulase-negative staphylococci (82%), followed by non-lipophilic diphtheroids (30% on lesional and 23% on non-lesional skin). Lipophilie diphtheroids were 4% on plaques and 30% on non-involved skin (6).

In this investigation, the most prevalent finding was the marked increase in the carriage rate of *S. aureus* in the psoriatic patients and their concurrent increase in density after short-term occlusion. This increase in incidence was not noted with atopic dermatitis. No significant changes in the incidence of lipophilic diphtheroids were noted after occlusion in atopic dermatitis and psoriatic plaques. When present, a significant increase in lipophilics was noted after occlusion.

Short-term occlusion of the skin of atopic dermatitis and of psoriasis

The experimental model of occlusion performed with plastic film on the forearm represents an exaggerated type of occlusion. Although in clinical

situations this duration of occlusion seldom occurs, some types of clothing and therapeutically applied dressings, bandages, tapes and several topically used vehicles including soft paraffin may result in occlusive or semi-occlusive conditions lasting for various periods of time, mostly as short-term occlusion. Therefore, the results of this study may not be fully comparable with the conditions of everyday life for these patients. Whereas occlusion promoting hydration of the usually dry epidermis in atopic dermatitis could be of some benefit for these patients, the quantitative increase in the skin microflora—particularly of *S. aureus*—after 24 hours' occlusion may become harmful to the patient and his/her environment. Similar considerations should be borne in mind regarding psoriasis: the high rate of desquamation and thus the dispersal of *S. aureus* as a public health hazard has been noted (6). According to these current findings, one should be aware of the possible need for antibacterial measures in the management of both skin diseases, especially for atopic dermatitis, when prolonged occlusion is required. The experimental data here concern 24 hours' occlusion; unfortunately we note (unpublished data) more than the uncommon staphylococcal infection with even our more limited occlusion (8 hours) utilized in clinical practice.

REFERENCES

1. Aly, R., Maibach, H. I. & Mandel, A.: Bacterial flora in psoriasis. Br J Dermatol *95:* 603, 1976.
2. Aly, R., Maibach, H. I. & Shinefield, H. R.: Microbial flora of atopic dermatitis. Arch Dermatol *113:* 780, 1977.
3. Aly, R., Shirley, C., Cunico, B. & Maibach, H. I.: Effect of prolonged occlusion on the microbial flora, pH, carbon dioxide and transepidermal water loss on human skin. J Invest Dermatol *71:* 378, 1978.
4. Leyden, J. E., Marples, R. R. & Kligman, A. M.: *Staphylococcus aureus* in the lesions of atopic dermatitis. Br J Dermatol *90:* 525, 1974.
5. Maibach, H. I.: Experimentally-induced infections in the skin of man. *In* Skin Bacteria and Their Role in Infection (ed. H. I. Maibach & G. Hildick-Smith), p. 33. McGraw-Hill, New York, 1965.
6. Marples, R. R.: The effect of hydration on the bacterial flora of the skin. *In* Skin Bacteria and Their Role in Infection (ed. H. I. Maibach & G. Hildick-Smith), p. 33. McGraw-Hill, New York, 1965.
7. Marples, R. R., Heaton, C. L. & Kligman, A. M.: *Staphylococcus aureus* in psoriasis. Arch Dermatol *107:* 568, 1973.
8. Noble, W. C. & Savin, J. A.: Carriage of *Staphylococcus aureus* in psoriasis. Br Med J *i:* 417, 1968.
9. Selwyn, S. & Chalmer, S. D.: Dispersal of bacteria from skin lesions: A hospital hazard. *77:* 349, 1965.
10. Williamson, P. & Kligman, A. M.: A new method for the quantitative investigation of cutaneous bacteria. J Invest Dermatol *45:* 498, 1965.

Blood Hyperviscosity in Psoriasis

Ronni Wolf,[1] Israel Machtey,[2] Eleasar J. Feuerman[1] and Draga Creter[3]

[1]Department of Dermatology, Beilinson Medical Center, Petah Tikva; Sackler School of Medicine, Tel Aviv University Tel Aviv; [2]Department of Medicine A and Rheumatology Service, Hasharon Hospital, Petah Tikva; and [3]Department of Haemostasis, Hasharon Hospital, Petah Tikva, Israel

Received July 23, 1980

Abstract. A study of platelet function and whole-blood viscosity in 11 patients with the confirmed diagnosis of psoriasis revealed a significant elevation in whole-blood viscosity (3.33±0.37 cP) as compared with that found in a control group (2.80±0.1 cP). The platelet count and platelet aggregation with ADP, epinephrine and collagen as well as platelet malonyldialdehyde were all within the normal range in all the patients. It is suggested that the increased blood viscosity may contribute to the higher incidence of occlusive vascular disease occurring in patients with psoriasis.

Key words: Psoriasis; Hyperviscosity of blood; Occlusive vascular disease

The high incidence of occlusive vascular episodes occurring in psoriatic patients treated with Azaribine prompted a number of investigators to carry out retrospective studies. In a recent study of dermatological patients, McDonald & Calabresi (4) noted that psoriatic patients have a significantly higher incidence of occlusive vascular disease than non-psoriatic patients.

The reports in the literature confine themselves largely to clinical observations and statistical analyses of such cases, and little has been done to elucidate the mechanism responsible for this phenomenon. We report here the findings of a study of platelet functions and whole-blood viscosity made in psoriatic patients.