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Experimental Folliculitis with *Pityrosporum orbiculare*: The Influence of Host Response

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Goodfield MJD, Saihan EM, Crowley J. Experimental folliculitis with *Pityrosporum orbiculare*: The influence of host response. *Acta Derm Venereol (Stockh)* 1987; 67: 445-447.

The aetiology of the folliculitis associated with seborrhoeic eczema is unclear, though the yeast, *Pityrosporum orbiculare* has been implicated. *P. orbiculare* was applied under occlusion to normal forearm skin of patients with seborrhoeic eczema (SE), seborrhoeic eczema and folliculitis (SEF), and normal controls. There were significant differences in response to occlusion between the three groups. Those patients with previous clinical evidence of folliculitis (SEF) developed folliculitis at the site of occlusion more frequently than either of the other two groups ($p < 0.001$), in whom only one patient developed skin changes. This difference was not explained by the response to occlusion alone, nor by natural carriage of yeasts. These results suggest that the yeast *P. orbiculare* is necessary for the development of folliculitis, but that the nature of the host response determines those patients prone to follicular inflammation. (Received February 19, 1987.)

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Seborrhoeic eczema (SE), and the folliculitis associated with it (SEF), are common disorders of uncertain aetiology. The role of the yeast, *Pityrosporum orbiculare*, has been much discussed in this context (1). *P. orbiculare* is a lipophilic yeast which exists as a commensal in 90% of normal individuals (2), but in SE and SEF yeast overgrowth has been demonstrated (1, 3), though mycelia are rarely found, in contrast to other yeast diseases (4). This aetiology is further supported by the response to topical and oral antifungal therapy (3, 5 and 6). Despite the proposed common aetiology, folliculitis is not found in all patients with SE, however, and there must be other factors determining those prone to folliculitis.

In a series of experimental infections, Faergemann showed that following application and occlusion of pityrosporal yeasts, those people with previous pityriasis versicolor

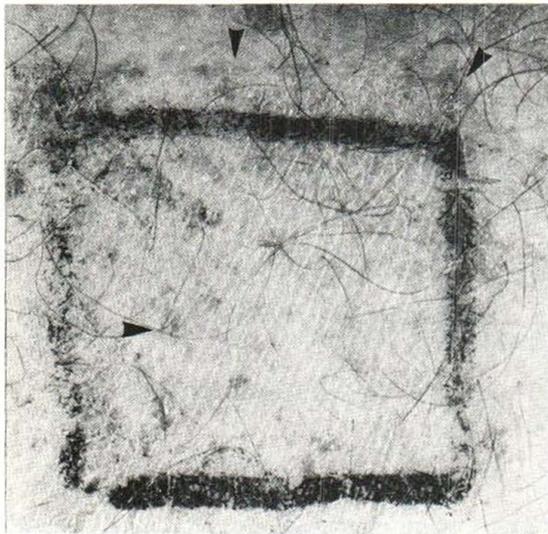


Fig. 1. Close up photograph of area occluded with yeast after removal of occlusion. Arrows indicate papular inflammation of follicles within and at the edge of the area to which yeast was applied, indicating spread of the liquid medium beyond the edge of the inert plastic square.

developed skin changes more often than normal controls (7). This suggests differences in the host response to the organism.

This study was intended to demonstrate the responses of volunteers with and without SE to occlusion with *Pityrosporum* yeasts.

PATIENTS AND METHODS

Thirty-three volunteer subjects formed 3 groups: seborrhoeic eczema (11). SE and folliculitis (12) normal controls (10). The *Pityrosporum orbiculare* used was isolated from an SEF patient. The organism was maintained on Sabouraud's agar with an olive oil overlay, and sub-cultured to Glycerol/Tween Agar (8) without oil overlay, for use in the occlusions. A suspension in saline was made and adjusted to approximately 10^8 yeasts/ml with the aid of a haemocytometer.

Of the yeast suspension in saline 0.1 ml was applied to an area of clinically normal skin on each patient's forearm. It was covered by an inert impermeable polythene square (2×2 cm), followed by a semi-permeable plastic dressing (Opsite) to secure it. The edges were secured with Micropore tape. Normal saline (0.1 ml) was applied to a control area of adjacent skin and secured in the same way. The dressing was left in place for 72 h. The skin was then examined for evidence of follicular inflammation and scaling and sampled for microscopy and culture to re-isolate any remaining *P. orbiculare*. Isolation of the organism from the uninoculated area was taken as an indication of natural carriage.

Statistical analysis was by a 2 by 3 contingency table, using the χ^2 statistics.

RESULTS AND COMMENT

The development of folliculitis in an occluded area was a positive result. In all cases this was a papular eruption (Fig. 1), without pustules. Table I shows the frequency of these in each group. It is clear that patients with previous clinical evidence of folliculitis develop follicular inflammation following occlusion (10 of 12 patients). This is not the case for patients without folliculitis previously (1 of 11), nor for the normal controls (0 of 10) ($p=0.0001$). Folliculitis developed equally frequently in the areas without yeast in all groups ($p=0.5$), indicating no effect of occlusion alone. The recovery of yeast from both

Table I. Numbers developing folliculitis (F+) under occlusion in each group

Figures in parentheses indicate numbers in whom *P. orbiculare* isolated from occluded area after occlusion removed). F- indicates patients not developing folliculitis

Group	<i>P. orbiculare</i>		Saline	
	F+	F-	F+	F-
SEF	10 (9)	2 (1)	3 (1)	9 (0)
SE	0	11 (7)	1 (0)	10 (4)
Normals	0	10 (6)	0	10 (6)

sites was similar in all groups, indicating similar rates of survival (of occluded yeast) and of natural carriage. No mycelial elements were seen amongst the recovered yeasts from any patient.

The study indicates that there is a significant difference in host response to occlusion between the groups, not explained by differences in natural carriage or yeast survival. This is of such a magnitude that the artificial environment of occlusion is also unlikely to explain it.

Other pityrosporal conditions may be related to detectable differences in immunity (9), and SEF patients have raised levels of pityrosporal antibodies (10). The development of both SE and SEF in AIDS (11) suggests that impaired cell-mediated immunity may be implicated. Alternatively, a more local abnormality due to sebum abnormalities previously described (12) may be implicated, although this should be common to all of those with SE.

It is likely then that nature of the host response is the determining factor in the development of SEF. The yeast *P. orbiculare* is necessary for this inflammation to occur, but is not the only factor. Further investigation should be directed towards establishing the immunological abnormality which allows this condition to develop.

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