〈教育セミナー〉
（アクネケア最前線）

アクネの発症メカニズム

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Pathogenesis of Acne

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Abstract

Acne vulgaris is a skin disorder of sebaceous follicles that commonly occurs in adolescence and in young adulthood. The major pathogenic factors involved are hyperkeratinization, obstruction of sebaceous follicles which result from abnormal keratinization of the infundibular epithelium, stimulation of sebaceous gland secretion by androgens, and microbial colonization of pilosebaceous units by *Propionibacterium acnes*, which promotes perifollicular inflammation. Follicular keratinocytes in comedones can be seen to possess increased numbers of desmosomes and tonofilaments, which result in ductal hypercornification. The increased activity of sebaceous glands elicited by androgen causes proliferation of *P. acnes*, an anaerobe present within the retained sebum in the pilosebaceous ducts. The organism produces several biologically active mediators which may contribute to inflammation, for instance by promoting leukocyte migration and follicular rupture. To examine the participation of neurogenic factors in the pathogenesis of acne, we quantitatively assessed the effects of neuropeptides on the morphology of sebaceous glands *in vitro* using electron microscopy. Substance P, which can be elicited by stress, promoted the development of cytoplasmic organelles in sebaceous cells, stimulated sebaceous germinative cells and induced significant increases in the area of sebaceous glands. It also increased the size of individual sebaceous cells, and the number of sebum vacuoles for each differentiated sebaceous cell, all of which suggests that substance P promotes both the proliferation and the differentiation of sebaceous glands. An improved understanding of the pathogenesis of acne should lead to a rational therapy to successfully treat this skin disease.

Key words: acne vulgaris, sebaceous follicle, *Propionibacterium acnes*, inflammation, substance P.