

**PATHOPHYSIOLOGY
OF
DERMATOLOGIC
DISEASES**

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S E C O N D E D I T I O N

One of the editors (N.A.S.) dedicates this book to his mother, Mildred Elizabeth Soter, and the other editor (H.P.B.) dedicates this book to his family.

PATHOPHYSIOLOGY OF DERMATOLOGIC DISEASES

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Biology of the Sebaceous Gland and the Pathophysiology of Acne Vulgaris

John S. Strauss

BIOLOGY OF SEBACEOUS GLANDS

Sebaceous glands are found everywhere on the human skin except for the palms, soles, and dorsa of the feet. The sebaceous glands are part of the pilosebaceous unit, and their secretion (sebum) flows through the sebaceous duct into the follicular canal. Sebaceous glands also are found on some mucous membranes. Those in the mouth empty directly onto the surface through the gland duct itself. There is great variation in the size and density of the glands; more glands and larger glands are found on the face and scalp compared with other areas of the body. On much of the face in adults, the sebaceous glands are the predominant portion of the follicle. These follicles are called sebaceous follicles (Fig. 13-1) and are the follicles that are involved in acne.

Sebaceous cells enlarge and contain more lipid as they move in a centripetal direction to the center of the sebaceous acini. Mitotic activity is restricted to the peripheral basal cell layer of the gland. Therefore sebaceous gland development involves both mitosis and lipogenesis, and there is evidence that these two processes are independently controlled. Sebaceous gland secretion is a continuous process and is holocrine in nature. However, the capacity of the unprotected skin to hold lipid is limited, and therefore excess lipid is lost. If the skin is protected, the lipid will continue to accumulate.



Figure 13-1 Sebaceous follicle from the human cheek. The sebaceous ducts, as well as the widely dilated follicular canal, are filled with keratinous material. A small vellus hair is present. [From TB Fitzpatrick et al (eds), *Dermatology in General Medicine*, 3d ed, New York, McGraw-Hill, 1987, p 186.]

Sebaceous gland growth is under endocrinologic control.¹⁻³ The prime stimulus for growth in the male is testicular testosterone and its metabolic products such as dihydrotestosterone. In the female, ovarian and adrenal androgens are probably responsible for gland growth. While the glands are large at birth, they undergo atrophy soon afterward and do not enlarge again until the beginning of puberty. Since the sebaceous glands are androgen sensitive, their growth is one of the earliest signs of the pubertal spectrum. After puberty, the glands remain stable in size. In women, they undergo atrophy during menopause, but in men sebaceous gland activity

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is maintained until approximately 70 years of age. While androgens are of prime importance, there is an important pituitary influence on sebaceous gland activity, and in hypopituitarism, sebaceous gland activity is markedly diminished.

Physiological amounts of estrogens do not influence sebaceous gland development and, therefore, are unimportant in the normal control of the sebaceous gland. However, pharmacologic amounts of estrogen do decrease sebaceous gland activity.⁴ This effect is mediated through central inhibition of androgen production. This finding is used in the treatment of severe acne in women.⁵

Because the sebaceous glands are deep in the dermis and do not open directly to the skin surface, it has not been possible to collect sebum, the product of the sebaceous glands, directly. Therefore, skin surface lipids have been used to study sebaceous gland secretion and sebum composition. The skin surface lipids represent a two-compartment system in that they originate from both the epidermis and the sebaceous glands. Since sebaceous and epidermal lipids are distinctly different, the composition of the skin surface lipids will vary depending upon whether the major portion is derived from the epidermis or the sebaceous glands.⁶ The approximate composition of sebum and epidermal lipid is given in Table 13-1. Triglycerides and their breakdown products make up approximately two-thirds of both epidermal and sebaceous lipids. Therefore, the concentration of triglycerides and their breakdown products does not vary significantly as the percentage of sebaceous and epidermal lipids changes. On the other hand, since triglycerides are hydrolyzed by bacterial lipases, the concentrations of triglycerides, diglycerides, and free fatty acids may vary greatly in a given individual under various conditions.

Besides triglycerides, the epidermal lipids are cholesterol and cholesterol esters. In contrast, less than 5 percent of sebaceous lipids are cholesterol and cholesterol esters. Wax esters and squalene are uniquely of sebaceous origin and represent the major nontriglyceride fraction of sebum. Therefore,

Table 13-1
APPROXIMATE COMPOSITION OF SEBUM AND
EPIDERMAL LIPID

	Sebum, wt (%)	Epidermal Lipid wt (%)
Glycerides and free fatty acids	57.5	65
Wax esters	26.0	—
Squalene	12.0	—
Cholesterol esters	3.0	15
Cholesterol	1.5	20

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