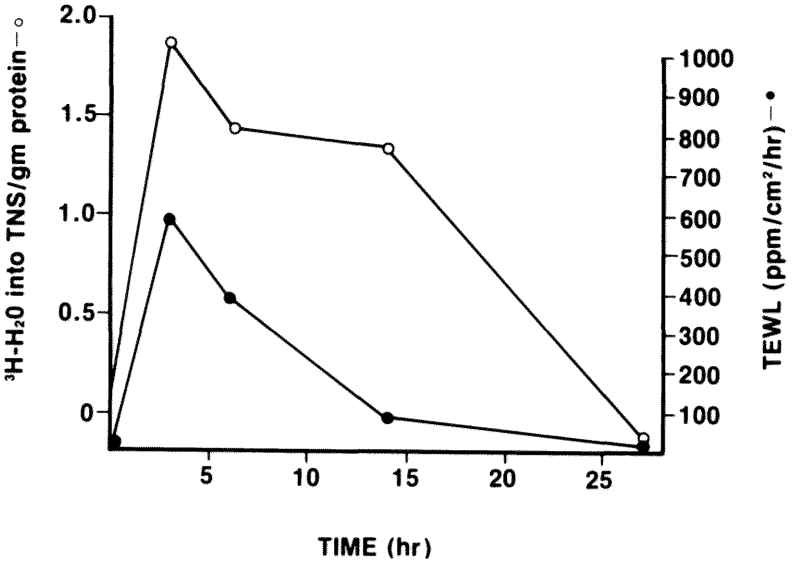

Figure 2.3 The time course of epidermal lipid biosynthesis and barrier function after treatment with acetone. Note that transepidermal water loss (*TEWL*), total nonsaponifiable lipid biosynthesis (*TNS*), and cholesterol (C) synthesis exhibit a parallel return toward normal over 24 hr (from Ref. 39, reprinted with permission).

and, then, to correlate barrier status with both an assessment of lipid replenishment in oil red O- and nile-red-stained frozen sections, and with rates of lipid biosynthesis (from tritiated H_2O) in the same samples. These results have shown (1) that the epidermis is a major site of sterol synthesis, accounting for about 30% of total cutaneous sterologenesis (38); and (2) that both epidermal sterol and fatty acid synthesis are stimulated by perturbation of the permeability barrier (39,40). Moreover, such stimulation is localized to treated sites, is limited to the epidermis (39–41), corrects as barrier function returns to normal (39; Fig. 3), and correlates, first, with removal and, subsequently, with repletion of lipids in the stratum corneum (39,41). (3) Epidermal sterologenesis is stimulated in essential fatty acid deficiency in relation to the defect in barrier function, (i.e., lipogenesis rates normalize with occlusion despite persistence of the underlying deficiency state; 42). (4) Synthesis is normalized when an impermeable membrane is applied to the perturbed skin (39–41). (5) Finally, the relationship of epidermal lipogenesis to barrier function is underscored further by the lack of