Retinoic acid (RA) is a regulator of epidermal cell growth and differentiation (1). RA shows its effects by binding RA receptors (RAR) and retinoid X receptors (RXR) (2). Aim of this study was to investigate the effects of excessive retinoic acid on cell proliferation and RAR alpha expression in skin. We used a total of 12 adult female balb-c mice in control and experimental group. In experimental group 80mg/kg/day 13-cis RA was applied for 5 days. 5 days later 50mg/kg BrdU injection was done intraperitoneally. Skin biopsies were fixed with %10 formalin, embedded in paraffin. Collagen fibers were stained with Van Gieson. For immunohistochemistry, sections were stained with anti RAR alpha and BrdU antibody and evaluated with light microscopy. Type III collagen (thin) filaments were observed in RA applied group in sections stained with Van Gieson. There was no difference between experimental group and control group in BrdU labeling. In control group, RAR alpha immunoreactivity was observed intensively in dermis. In experimental group, weak labeling was seen in dermis. Schwartz et al. (3) stated that type III collagen synthesis which is found with type I collagen fibers is increased with topical treatment of RA. Type III collagen fibers participate in type I collagen synthesis (4). We observed that type III collagen fibers stained with yellow with Van Gieson stain in RA treatment group. All-trans RA represses RAR alpha in cells transfected with RAR alpha and increases collagen synthesis by repressing increased MMP-1 synthesis through RAR alpha pathway (5). We observed that RAR alpha labeling is decreased in RA treatment group. Our findings are compatible with the studies of Watson et al. (5) and stimulative effect of RA on collagen synthesis may be through RAR alpha receptor and MMP synthesis. Further studies are needed to investigate the effects of different retinoic acid receptors and MMP types on this process.

References