

THE ANTIPROMOTING EFFECT OF ALKYLOGLYCEROLS FROM SHARK LIVER OIL

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Carcinogenesis is a pathological process which may be mimicked in experimental models. The entire process may be subdivided into three steps: initiation, promotion and progression. The three steps of this model are routinely generated in mouse skin tissue, using the carcinogen 7,12 dimethylbenz [a]anthracene (DMBA) as an initiator, and cocarcinogens such as 12-O-tetradecanoylphorbol-13-acetate (TPA), which is present in croton oil, or other diterpene esters for promotion. The aim of our study was to verify the effect of alkyloglycerols from shark liver oil (Ecomer, Exposan AB, Sweden) on mouse skin papilloma development at the promoting step of carcinogenesis.

For the induction of skin papillomas, inbred 8-week-old female BALB/c mice were used. Papillomas were initiated by painting the skin of the mice with an acetone solution of carcinogen DMBA in a single dose (100µg/mouse). For the promotion of papilloma growth, croton oil (10µl for a single painting) dissolved in acetone was applied twice a week for 22 weeks. Alkyloglycerols from shark liver oil were applied to the mices' skin a day after croton oil painting.

Alkyloglycerols from shark liver oil gave a four-fold decrease in the number of papillomas found on the skin 24 weeks after initiation. They also prolonged the period of latency from 7 to 10 weeks. When applied to mice treated with DMBA, but without the promotion step, only one mouse developed papilloma. The one papilloma developed in the group treated only with alkyloglycerols from shark liver can be treated as a random effect.

In comparison to the control (croton oil treatment), we can state that alkyloglycerols from shark liver oil cannot be promoters for papilloma growth. The obtained results suggest that the alkyloglycerols displayed an antipromoting effect.