generative epithelial proliferations the xylol, used as the solvent, plays an important causative role. Gawronski (1904) reported equally unsuccessful experiments in which dogs were employed. Ten years later Davis mentioned that he failed to elicit neoplastic responses in the skin of animals by the application of slack-wax and pressed distillate. In 1922, Leitch succeeded in producing cancers in the skin of mice after the repeated painting of various fractions of crude shale oil (green oil, blue oil, unfinished lubricating oil, and unfinished gas oil [the first three fractions mentioned contain paraffin]). Rats, guinea pigs, and rabbits, subjected to the same treatment proved refractory. Twort and Ing (1928) and Wood (1930), who applied various brands of purified, liquid paraffin to the skin of mice, did not elicit any neoplastic lesions.

Leitch reported in subsequent studies (1922, 1923, and 1924) the production of cutaneous malignancies in mice which had received applications of various refined oils obtained from shale oil and different types of petroleum, demonstrating that refined mineral oils, such as used for the lubrication of machinery, can cause cancer of the skin. Rats and guinea pigs treated similarly failed to develop cutaneous tumors. Rabbits showed only transient warts after eleven months of painting.

The most thorough and extensive experimental investigations of carcinogenic qualities of various mineral oils were conducted by Twort and Twort; Twort and Fulton; Twort and Lyth; and Twort and Ing. In a series of experiments, extending over a period of many years, these investigators demonstrated on large series of mice the carcinogenic property and potency of various crude and refined mineral oils obtained from naphtha and shale distillates. The results of these experiments were mentioned briefly before. As one of the by-products of these studies, Twort and Lyth developed a biological method for measuring the carcinogenicity of mineral oils. When 0.5 cc. of a certain mineral oil to be tested was injected intraperitoneally into mice, and the oil was recovered from the abdominal cavity one to 10 weeks later, and examined for its refractivity, the degree of reduction in refractivity ran parallel to the carcinogenic potency of the original oil injected. This reaction was related directly to the degree of unsaturation of the oil. Twort and Lyth attributed the decrease in refractivity of the oil, observed after its stay in the abdominal cavity, to the occurrence of chemical changes (reduction or oxidation).

The potency of a particular oil depends upon various factors (Twort and Twort): (*1*) the concentration of carcinogenic units in the agent; (*2*) the amount of the agent applied; (*3*) the nature of the diluent, if one was used (lanolin; purified paraffin oil; animalic and vegetable oils reduce the carcinogenic activity, while chloroform, benzol, and other organic solvents, which remove the natural fat of the skin, increase the carcinogenic activity of the oil tested); (*4*) possibly the area covered by the applications; (*5*) the total number of applications; (*6*) the frequency of applications; (*7*) the