

McIntosh obtained in 1933 four tumors in fowls treated with tar, three of which could be propagated readily by filtrates prepared from the original neoplasms. Sarcomas produced in chickens by the injection of tar or 1.2.5.6-dibenzanthracene proved to be of the non-filtrable type (Sturm and Murphy (1928); Peacock (1933); Mellanby (1934); Gye and Purdy (1934); and Andrewes). Andrewes observed that pheasants, into which the chicken tar tumors could be inoculated successfully, developed antibodies which would neutralize the virus of Rous sarcoma no. 1. This investigator concluded from this evidence that tar fowl sarcoma contains a virus, though this cannot be directly demonstrated by infections with filtrates.

Andrewes, Ahlström, Foulds, and Gye; and Ahlström and Andrewes found that the intravenous injection of the rabbit fibroma virus (Shope) into rabbits, which had previously received a single intramuscular injection of tar, resulted in a generalized fibromatosis, an effect never obtained before with the virus only, and caused in one instance a polymorphous cell sarcoma. The spontaneous regression normally occurring in these virus fibromas was much delayed in rabbits which were inoculated intradermally or subcutaneously with the virus after an intramuscular injection of tar.

Very detailed and extensive experimental studies on the interrelation between the cancerigenesis by tar and the action of the Shope papilloma virus have been carried out by Rous and his coworkers, Beard and Kidd. Kidd and Rous observed, after the intravenous injection of the Shope papilloma virus into rabbits whose ears had been tarred previously for a period of 1.5 to 3 months, that there was a rapid development of numerous papillomas in the tarred areas. Some of the neoplasms were malignant, while the ears of tarred control rabbits showed only warts, which receded spontaneously later on. The investigators concluded from this evidence that the virus activates tar warts and may convert some tar papillomas into malignant ones, depending upon the localization of the virus in the skin. The virus acts in this connection, according to Rous, only in an adjuvant capacity, by exerting a driving and formative effect upon the cells rendered neoplastic by tar. Rous argued that as tar cancer cannot be produced at will, because this neoplasm affects only relatively few places in large areas subjected to the carcinogenic stimulation, as the incidence of tar cancer varies notably from individual to individual, as their origin is punctate, and as no experimental procedure thus far employed has caused them to appear as diffuse processes or in unexampled multitude, though their number may increase with continued tarring, some agent, probably a virus, must be present where tar cancers arise (indigenous virus). It was pointed out by Rous that there does not exist any serological relationship between papillomatous tumors produced by tar and those caused by virus, in spite of a close histological similarity.

This observation received support from a study of Höra, who found that the development of papilloma virus lesions was not hastened when the ears