Inhibition by Progesterone of Radiation-Estrogen-induced Mammary Cancer in the Rat

Albert Segaloff
Alton Ochsner Medical Foundation, New Orleans, Louisiana 70121

SUMMARY

The data from this preliminary experiment make it appear that continuous administration of progesterone protects animals from the synergism between estrogen and radiation. However, it also appears that the progesterone has by itself a protective effect against the individual carcinogenic agents since fewer tumors occurred on the opposite side as well as on the radiated side. In this preliminary experiment none of the animals that bore just progesterone pellets and were radiated developed mammary carcinomata.

INTRODUCTION

We have previously reported on the substantial synergism for mammary carcinogenesis between γ-radiation and continuous administration of diethylstilbestrol in the female A x C rat (1). It was important to learn whether the effects could be tempered by the simultaneous administration of the ovarian hormone progesterone. Accordingly, a preliminary experiment was designed to ascertain this.

MATERIALS AND METHODS

Female A x C rats weighing between 40 and 50 g at weaning were hysterectomized at 28 to 30 days to prevent fatal estrogen-induced uterine infections. The ovaries were left intact. The animals were divided into 3 groups. The 1st group received a diethylstilbestrol pellet and radiation in exactly the same fashion as in our previous study (1). The 2nd group received an estrogen pellet plus a pellet of progesterone plus radiation. The 3rd group received a progesterone pellet plus radiation. The pellets were all implanted intrascapularly when the rats were 8 weeks old. The 20-mg pellets contained either 25% diethylstilbestrol and 75% cholesterol, or progesterone. The pellets remained in place until the animals died or were sacrificed. Two days after pellet implantation all animals were anesthetized with urethan (ethyl carbamate), 1 mg/g of body weight, and the left mammary chain was radiated with 800 R of radiation in exactly the same manner and utilizing the same equipment as in our previous study.

RESULTS

There were 13 animals in Group 1 alive at the time the 1st tumor was observed. Ten of these developed multiple, grossly palpable tumors. The 1st tumor appeared on the radiated side at 22 weeks and the 1st tumor appeared on the nonradiated side at 34 weeks. There are subsequently more tumors on the radiated side (Chart 1). Of the 13 animals alive in Group 2 at the appearance of the 1st tumor, receiving both types of pellets and radiation, a single animal developed multiple tumors on both sides. The 1st tumor appeared on the radiated side at the 21st week and the 1st tumor on the nonradiated side appeared at 26 weeks. No tumors were observed on either the radiated or nonradiated side in the remaining 12 animals of this group. In Group 3, where the animals were radiated and received pellets of progesterone alone, none of the animals have developed tumors on either the radiated or nonradiated side.

All the animals in this study are now dead. The basic neoplasm is essentially the same as has been previously reported and is similar in all groups. The basic pattern is a poorly differentiated, solid (medullary), infiltrating carcinoma with little desmoplastic reaction, showing a variable degree of glandular and papillary differentiation. The predominant solid carcinomata contain large central zones
Table 1 shows the comparative incidence in the same fashion as the previous paper, and Chart 1 shows the total number of gross mammary tumors plotted against weeks after radiation.

ACKNOWLEDGMENTS

The author thanks Joseph Burfect, Roy H. Coleman, Albert Flores, and Peggy Hopkins for their technical assistance.

REFERENCES

Inhibition by Progesterone of Radiation-Estrogen-induced Mammary Cancer in the Rat

Albert Segaloff


Updated version
Access the most recent version of this article at:
http://cancerres.aacrjournals.org/content/33/5/1136

E-mail alerts
Sign up to receive free email-alerts related to this article or journal.

Reprints and Subscriptions
To order reprints of this article or to subscribe to the journal, contact the AACR Publications Department at pubs@aacr.org.

Permissions
To request permission to re-use all or part of this article, contact the AACR Publications Department at permissions@aacr.org.