The May and June issues of *Cancer Research* contain no less than 96 individual papers reporting cancer research results. While the total contents should be scanned for specific interests, the following articles should catch the eye of a clinically oriented oncologist.

In the May issue: (1) Structural and immunological relationships of isoferritins in normal and malignant cells, by Arosio et al. (Tufts University School of Medicine, Boston, Massachusetts); (2) Effect of bleomycin on deoxynucleotide polymerizing enzymes from human cells, by DiCicco and Sahai Srivastava (Roswell Park Memorial Institute, Buffalo, New York); and (3) Clinical evaluation of Floraful, by Valdivieso et al. (M.D. Anderson Hospital and Tumor Institute, Houston, Texas).

In the June issue: (1) Development of an assay for aryl hydrocarbon hydroxylase in human peripheral blood monocytes, by Bast et al. (National Cancer Institute, Bethesda, Maryland); (2) Plasma hormone profiles in populations at different risk for breast cancer, by Hill et al. (American Health Foundation, Valhalla, New York); and (3) Evaluation of Papanicolaou smear and effect of sample biopsy in follow-up of cervical dysplasia, by Youkeles et al. (University of California, Los Angeles, California).

Selected for comment here are two extensive reviews on topics of current interest, which attempt to synthesize the information as well as to marshall it.

**May**

Apffel (Pondville Hospital, Walpole, Massachusetts) presents a review on "Nonimmunological Host Defenses" against cancer. It is timely to highlight this broad area, since the great interest in cancer immunology may convert a research bandwagon into a juggernaut. It has become too popular to attempt to explain all effects upon tumor development and growth in immunological terms. Apffel points out that there are many other host defenses, of many diverse types. Some are directed against already transformed cells, and others prevent or inhibit initial events of carcinogenesis.

Chalones and oncolytic factors in sera and exudates are agents of containment. Under appropriate circumstances, the autoxidation of thiols and the formation of mixed disulfides lead to a destruction of tumor cells in vitro and in vivo. Both processes involve the generation of superoxide radicals and of hydrogen peroxide that, in turn, activate the peroxide-peroxidase-halide system. Thioldisulfide ratios and interchange co-determine the antioxidative activity of cellular
membranes, thus bearing on carcinogenesis. Many aliphatic and aromatic antioxidants have anticarcinogenic properties, and may be either endogenous or exogenous in foods. Antioxidant activity, linked with the ergastoplasm, is a part of a homeostatic mechanism that prevents self-accelerating chain reactions that could lead to membrane damage.

Carcinogens can be inactivated by microsomal enzymes belonging to biochemical mechanisms of detoxification. The activity of these enzymes is affected by diet and the state of nutrition, and can be increased with various inducers.

June

Marsh (Yale University School of Medicine, New Haven, Connecticut) reviews the "Effects of Cancer Chemotherapeutic Agents on Normal Hematopoietic Precursor Cells."

It is now three decades since the beginning of the modern era of cancer chemotherapy, during which most of the chemicals have been characterized by bone marrow toxicity. In fact, the hematologic effects have to be balanced against the antineoplastic effects in order to obtain optimum treatment regimens. There arose a series of tumor cell and hematopoietic cell assays, which the author gathers for presentation. He also attempts to derive generalizations regarding the hematopoietic precursor cells (HPC). Over 200 references are cited. The review is of considerable importance to normal hematology, as well as to further progress in cancer chemotherapy.

The assays for HPC are functional rather than morphologic, depending on the formation of hematopoietic colonies in vivo or in vitro. Some dozen such tests are described, all being affected by the experimental conditions of the cells, the host or medium. The interpretations, as in most biological assays, depend on the dose-response relationships that are elicited.

The effects of a given antineoplastic agent on HPC may depend on the dose, schedule and route of administration of the drug, proliferative state of the target cell and the age, sex and species of the host. The metabolism of the drug, uptake by the target cell and its stage in the cell cycle are also critical determinants.

HPC assays will undoubtedly be helpful in clinical applications, such as reduction of risk of toxicity to single new agents and to the increasingly popular combinations of agents. Such assays will continue to be useful also to define the normal physiology of HPC, with respect to maturation sequence, proliferative state and response to depletion of various populations of hematopoietic cells.