NUTRITION CANCER

Edited by

Jan van Eys, M.D., Ph.D. M.D. Anderson Hospital & Tumor Institute Houston, Texas

Mildred S. Seelig, M.D., M.P.H. New York University Medical Center New York, New York

and

Buford L. Nichols, Jr., M.D. Baylor College of Medicine Houston, Texas



New York • London

Editorial Foreword to Part II

Selected research and review communications of direct relevance to nutrition and cancer, the subject of the plenary sessions of the Eighteenth Annual Meeting of the American College of Nutrition, are included in Part II of this volume. Those dealing with learned food aversions and its evaluation and treatment in patients with cancer (Bernstein et al.; Fotopoulos et al.; and Settle et al.) complement the paper on anorexia and the cancer patient.

The papers dealing with minerals and neoplasms present evidence on abnormal levels of trace metals and magnesium in patients with cancer, and consider the roles of these abnormalities in the pathogenesis of the neoplastic process. Flynn, who reported elevated serum levels of copper in patients with advanced solid tumors, noted normalization of copper levels that was directly related to favorable response to therapy. Lower selenium levels were found in women with breast cancer than in normal women (McConnell et al.), but much wider than the normally narrow range of selenium levels was found in patients with other cancers, a finding suggested as possibly related to the type and stage of neoplasm and the treatment (Broghamer et al.). Shamberger and Willis considered the evidence that optimal small amounts of selenium might have an antineoplastic effect. They also surveyed the evidence that, because of zinc's importance in normal cellular metabolism, it might enhance growth of neoplastic cells—which growth might be inhibited by zinc depletion. On the other hand, they point out that zinc's inhibition of certain viruses (and thereby possibly of tumor viruses) might lead to inhibition of certain tumors. Collery et al. presented new data on the plasma and erythrocyte magnesium levels of patients with advanced neoplastic disease, and correlated low plasma magnesium levels in such patients with their susceptibility to thrombotic events. They suggest that a rise in erythrocyte magnesium in cancer patients might reflect a rise in cancer cell magnesium content, such as is seen in malignant cells. They consider the antimetabolic effect of clinical magnesium depletion, but recommend caution in depleting the body of this essential nutrient.

As in the case of minerals, some vitamins seem to have some anticarcinogenic activity in some test systems, whereas inhibition of others has long been known to exert antineoplastic effects (Shamberger and Willis). Leklem et al. present new data on the vitamin B_6 status of breast cancer patients and review the evidence that abnormalities in the pyridoxine-dependent tryptophan metabolic steps might be contributory to the disease process in patients with breast or bladder cancer.

The correlation of chronic alcoholism with increased risk of certain types of cancer (Feldman and Kissin) is of interest, in view of the general malnutrition and specific mineral deficiencies (e.g., magnesium and zinc), excess (copper), and vitamin deficiencies. Thus, additional to the direct tissue damage that is produced by alcohol abuse, induced imbalance of trace and major substances might contribute to the neoplastic process.

Mildred S. Seelig, M.D., M.P.H., F.A.C.N.
President-Elect, American College of Nutrition (1979-1981)
Chairman, ACN Council on Magnesium and Trace Substances