AUTHOR'S FOREWORD

We started our inquiry into the basic chemistry of neoplastic and virus parasitisms in 1916 when facts were few and laboratory methods crude—long before Warburg's classical work was reported. The clinical approach therefore promised to be more comprehensive and reliable as the major source of information. Thus it was evident that neither cancer nor virus infection took place unless function was first impeded, impaired, or exhausted. We concluded therefore that the "front line" energy producing oxidations were protective and burned fuel substrates and toxins in much the same manner. The appropriate process thereto is entirely overlooked in the laboratory attack which still recognizes the Krebs system of oxidations and hydrolytic glycolysis as the only sources of energy for function and growth. On the other hand, both of these systems support virus and neoplastic parasitisms. Thus restrictions in the laboratory approach deprived it of its chance to even make "first base" in the search for the survival factor.

We aimed to identify and duplicate the details of this protective process and found it could be initiated by activated dehydrogenator carbonyl groups and carried along by free radicals and peroxide free radicals. The state of activation of the carbonyl groups determined the range and efficacy of the protection. The process could be blocked by groups that abolish the Pasteur Effect, such as sulphydryl, guanidines and amidines extractable from spermatogenic organs, extracts of tissues of animals that died in great fear, cancer extracts, but not by normal tissue extracts. The process could be terminated at will by reagents that block polymerizations, such as inert free radicals and electrophilic double bonds. Thus its chain reaction character is identified. But the laboratory has no suspicion of its existence as yet. In the experimental production of cancer, applications of complex tars stimulated tissue functions, reproductions, glycolysis, and Krebs cycle oxidation, (Deotto, 1936) while pure synthetic carcinogens depressed the oxidations of