AUTHOR'S FOREWORD

It might be taken as a rule that where sugar is vigorously burned in physiological processes, not only are fats burned too, but also every other combustible substance that enters the blood and tissues. Natural immunity destroys every virus, every disease germ poison, or structure, and every allergenic agent in this way. To force immunity to disease, therefore, one can do no better than to establish a vigorous oxidation mechanism. The present day understanding of this process is not sufficient to be of much help, and to work out the different steps that are traversed in the oxidation of sugar will take some years of additional effort. The dilemma was even worse twenty-five years ago when the problem came across my path. It was useless to think of clarifying the oxidation process step by step in the short time at my disposal. So instead of studying the sugar burning mechanism directly to learn what deficiencies might happen in the process to break immunity, I thought it more practical to ascertain what types of atomic groupings could exert toxic effects, and to identify them with different stages in sugar oxidation. In those days photochemistry was not developed to the point where it could be of much help, and besides, disease toxins were not recognized by their atomic groupings, but were regarded as protein molecules of quite large molecular weights and no one would see any relationship between oxidation and immunity. Lysins, precipitins, and agglutinins

were the rage. However I was strongly attracted by the possibilities of such unsaturated bonds as join the imid group of guanidin to the carbon atom, and occurs in histamine, and also by the double bonds of the ethylene group and between carbon and oxygen in the keto and particularly in the quinone groups. Personal experiments with fluorescent substances gave two startling disclosures; one, a substance that can produce allergically a sensation that continues for days after application to the skin, even after it is washed off. This substance is a halogen compound related in structure to caffeic acid, coumarin, and æsculin, thyroxin, and adrenalin. The other experience was the production of a faint blue light for a while by a mixture evolving exothermic energy in the presence of a fluorescent substance. It was in the attempts at explanation of these phenomena that the systems of sugar oxidation and the idea of allergy and immunity presented here were worked out. That the cancer abnormality is associated with the failure to utilize lime appears from the following facts. In cancer there tends to be a lipoid in water phase of colloid dispersion. Such conditions exist experimentally in water lipoid systems according to J. Loeb where there is a dominence of monovalent over divalent cations. In the case at hand it appears that calcium utilization is deficient. Calcium is certainly necessary to sugar oxidation, and the calcium fixing vitamins and hormones all are helpful in cancer treatment in my experience. Even powdered chalk is helpful. Calcium, phosphoric acid, and guanidine are lost in the urine after parathyroidectomy. Guanidine fits into the oxidation mechanism in two places and in connection with phosphoric acid utilization. Phosphoric acid in

anærobic glycolysis as occurs in cancer, appears to aid in hydrolysis toward lactic acid production with the aid of coenzymes and calcium; but in the ærobic burning of sugar I believe that it mediates a dehydration of the hexose molecules in several ways that will be described later. This dehydration permits direct peroxidation and burning of the molecule; and thus calcium deficiency and oxidation block run hand in hand in cancer metabolism. The calcium, one might conclude, plays its part not only in the dehydration and peroxidation of the straight and cyclic chains of more than one carbon atom but also in the utilization of and peroxidation of the formaldehyde produced from the straight unsaturated chains during their oxidation as described below, and particularly in the synthesis of unburned formaldehyde into the diketone described later, and even into hexose. At least this theory comes close to the basis of the present research. If it is in error, as well might be, the error is lucky indeed for through it we arrived at a useful treatment of otherwise incurable disease; and the recoveries are able to be permanent as twenty years of practical application now shows.

In the following discussion, photochemic definitions closely follow the wordings of Griffith and McKeown, "Photoprocesses in Gaseous and Liquid Systems," Longmans, Green and Co. Colloidal affairs are described in MacDonagh's terminology because of its familiarity to physicians. This does not mean that we agree with MacDonagh's thesis entirely; for instance, it is impossible to understand how the blood protein can vary in quantity to the enormous extent that the scarcity of particles in some microscopic fields and the large number in others in the same and different

humans would indicate. It would seem that the blood protein is represented by the homogenous material between the particles as well as the particles themselves, which possibly are artifacts produced by the contact with foreign conditions attending microscopic examination. The type and extent of artifact produced will be an index to colloidal stability or entropy and thus, though it does not represent the total blood colloid, it gives an estimate of conditions. We therefore follow MacDonagh's descriptive system trusting that the reader will permit our use of the terms according to this explanation. We might add that the lipoid changes he describes in cancer should find better explanation in Loeb's expositions of water lipoid systems as influenced by monovalent and divalent cations and by anion variations. The lipoid deposits in cancer cells are definitely the result of the inability to burn them, and therefore they depend upon the deficiency in and the crippling of the oxidation catalysis that forms the basis of our subject. Essentially the subject centers about the activation of oxygen by the free valences of the extremely labile metabolites of ærobic glycolysis on the one hand, and about the polymerization of and inactivation of these valences on the other, when oxygen supply is inadequate and stable peroxides of high molecular weight are present to favor polymerization. The allergenic diversion of energy, and the changes in colloidal and cation distributions that have the greatest clinical importance are dependant matters. Primary in my experience stands the inhibitory effects of animo and imino groups against the free valent activity of the carbonyl and ethylene groups in their catalysis of the oxidations as I describe them here.