ened papers were decolorized. It was suggested this substance is in the nature of a quinone.

The significance of this volatile substance may be, then, that it acts like an "antibiotic" or defensive mechanism for mycelial growth; that it inhibits fructification unless it is modified in the casing soil; and that it affects normal growth when allowed to accumulate. It is of interest to note that pathogens which do not attack the mycelium in the compost, do so in the casing soil—that is, in the medium where the antibiotic value of this volatile substance is at the lowest ebb. The intensity or high potential of the substance diffusing from the mycelium was shown by embedding oxidation-reduction indicators in agar slants (*5*). It appears that the oxidizing intensity of this substance is so great that it prevents the thickening of the hyphae from which sporophores develop. The necessity for adjusting the soil at a relatively high pH for best fructification tends to show that the soil must be a medium suitable for rapid oxidation-reduction reactions. A movement of air or oxygen-ventilation is, of course, required for such reactions. Accordingly, at least one of the functions of the casing soil is to provide an alkaline-oxygenated medium for the destruction of this volatile substance. A fuller presentation will be published elsewhere.

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The Medical Examination of Hiroshima Patients with Radiation Cataracts[1]

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Radiation cataracts, the first late manifestation of exposure to atomic bomb radiation reported in man, have been described by Cogan, Martin, and Kimura (*1*), and Cogan, Martin, Kimura, and Ikui (*2*). Kimura (*3*) reported 98 Hiroshima patients with radiation cataracts. Eighty-five of the 98 were among 922 survivors 1000 m (1094 yards) or less from the hypocenter,[3] an incidence of 9.8%.

This group of 98 patients with radiation cataracts is a select group because all were unquestionably exposed and all show unmistakable delayed response to radiation injury. Seventy-eight of them have been examined by the medical department of the Atomic Bomb Casualty Commission Clinic in Hiroshima in an effort to discover if other late manifestations of radiation injury exist. The examinations were performed approximately five years after exposure.

The group comprised 45 men and 33 women between the ages of 12 and 69 years, and they had been from 150 to 1240 m from the hypocenter; 64 were between 700 and 1099 m (710–1202 yards). Detailed radiation and medical histories were obtained, and physical examinations were performed. Roentgenograms of the chest and blood, and urine and stool specimens were examined. Thirty-four patients were proctoscoped, and sternal marrow specimens were obtained from 27 patients. Additional diagnostic studies were performed as indicated.

Symptoms presumably produced by acute radiation were vomiting, fever, diarrhea, oropharyngeal lesions, bleeding gums, purpura, epilation, amenorrhea, and abnormal periods after amenorrhea. At least 50% of the patients experienced fever, purpura, epilation, amenorrhea, and vomiting on the day of exposure, suggesting that at least half of them received severe irradiation.

Forty-six of the 78 patients received mechanical injuries described as minor lacerations and penetrating wounds from flying glass and other small objects. Several patients were bruised by falling beams and roofs, but the only major injury was a depressed skull fracture. As far as could be determined, all injuries resulted from indirect blast effects. Small thermal flash burns occurred in 15 patients. Five of the burned patients were free from flash burn scars, suggesting partial shielding from thermal injury. One scar resembled a keloid.

Seventy-seven of the 78 patients experienced scalp epilation. This observation suggests that a cataractogenic dose will produce some degree of scalp epilation in the majority of patients.

Two males and one female above 39 years of age had diastolic blood pressures of 100 mm Hg or higher. The remaining individuals had blood pressure recordings within the usual range. Thirty-six patients had scars from burns and/or wounds sustained at the time of the explosion. The majority were minor and showed only small amounts of scar tissue. Radiation cataract was the only physical finding attributed to the late effects of the atomic bomb.

At the time of the study the hematological findings did not disclose any blood dyscrasias. Two patients with radiation cataracts have developed acute leukemia, one of them subsequent to her examination in this study and one child not included in this report. The aspiration sternal marrow specimens obtained on 27 patients were compatible with the peripheral hematological findings.

Chest films, stool and urine examinations, and serological tests for syphilis revealed no abnormalities that could be attributed to the atomic bomb. The histories did not reveal any information which suggested late effects of the atomic bomb other than visual complaints.

The shielding factor was not studied. The greatest

[1] Sponsored by the Atomic Bomb Casualty Commission, National Research Council, with funds supplied by the U. S. Atomic Energy Commission.

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[3] Point on the ground directly below the explosion.

number of patients with cataract formation in this series was 700–1000 m from the hypocenter. No cataracts have been found in the survivors in this report that were in the Fukuya Department Store, a reinforced concrete building 800 m from the hypocenter, where shielding was afforded.

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Histochemical Demonstration of Protein-bound Sulfhydryl Groups[1]

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Protein-bound or fixed sulfhydryl groups are essential for the activity of many enzymes, serve as linkages between proteins and some prosthetic groups, are involved in the contractile phenomena of muscle, and are important in the coagulation of blood, in cell permeability, and in hormone synthesis and activity (*1*). Methods for the detection as well as estimation of sulfhydryl groups of proteins are based on reactions with either oxidizing, reducing, alkylating, or mercaptide-forming agents. Some of these methods are not colorimetric, and most of them lack specificity because functional groups other than sulfhydryl are quite reactive toward these agents (*1*). However, modifications of some colorimetric techniques have been used histochemically to demonstrate sulfhydryls in tissue sections. For this purpose ferricyanide (*2*) and nitroprusside (*3, 4*) have been widely used, although no oxidizing agent other than cystine can be called specific for the oxidation of sulfhydryl groups in proteins (*1*). A method based on a new mercaptide-forming agent, 1-(4-chloromercuriphenylazo)-naphthol-2 (*5*), gives weak color reactions in tissue sections.

In order to improve the sensitivity of sulfhydryl histochemistry by increasing the color value of the final compound and by increasing the specificity of the reaction for sulfhydryls, a reagent was developed which contained a disulfide linkage, the specific oxidative group, and a naphthol moiety for coupling to form an azo dye. As shown in Fig. 1, the reagent,[3]

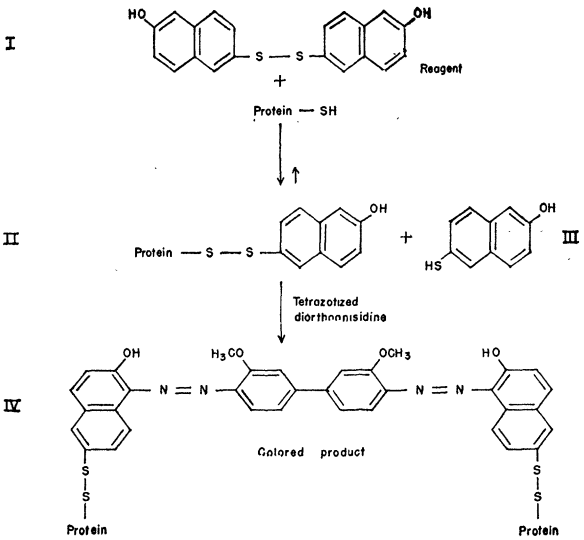

FIG. 1.

2,2′-dihydroxy-6,6′-dinaphthyl disulfide (I), when used in excess at pH 8.5, reacts with active sulfhydryl groups of fixed tissue proteins to form a colorless substance (II), which can be converted into an intensely colored azo dye (IV) by coupling with tetrazotized diorthoanisidine. The colorless oxidation product (II) was insoluble in both water and ether-alcohol, so that the excess of reagent (I) as well as the reduced reaction by-product (III) could be washed out of the tissues with organic solvents. Subsequent treatment of the tissues with tetrazotized diorthoanisidine resulted in the rapid development of a red color (monocoupling) or a blue color (dicoupling) at the sites of protein sulfhydryl groups (IV). Monocoupling (red or pink) was taken to indicate sparse, widely separated sulfhydryl groups, whereas dicoupling (blue) indicated a greater concentration of sulfhydryl groups.

The reagent (I) was prepared from sodium 2-hydroxy-6-naphthalene sulfonate. The hydroxy group was protected by conversion to the carbethoxy derivative, and the sulfonate group was converted to sulfonyl chloride with PCl_5 (*6, 7*). Reduction to a sulfhydryl group was accomplished with zinc dust and hydrochloric acid in alcohol, a method used for other naphthalene derivatives (*8*). Oxidation with ferric chloride gave the disulfide, and the carbethoxy group was hydrolyzed with hot alkali.

The required sodium-2-carbethoxy-6-naphthalene sulfonate (*6, 7*) was prepared by the slow addition of 84 ml ethyl chlorocarbonate to a vigorously stirred solution of 197 g sodium 2-hydroxy-6-naphthalene sulfonate (Eastman technical grade) and 32 g sodium hydroxide in 800 ml water. The stirring was continued for an additional hour, after which the mixture was cooled to complete the precipitation of the carbethoxy derivative. It was collected with the aid of suction and dried by warming on the steam bath. The crude product (177 g) was ground with an equal weight of phosphorus pentachloride and was heated on the steam

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[2] Acknowledgment for technical assistance is due Edith Herman and Ralph Gofstein.

[3] This reagent (DDD) is now available from the Schwartz Laboratories, Inc., New York City.