have been tested for teratogenic properties. 3. When injected into the yolk sac of embryos carrying chorioallantoic grafts of the mouse sarcoma 180 and Toolan's human epidermoid carcinoma #3, "triazene" causes inhibition of tumor growth and cellular injury. This effect is similar to that produced by nitrogen mustard and other polyfunctional alkylating agents. Because "triazene" shows certain similarities in tumor-inhibiting activity when compared to nitrogen mustard and because of its distinctly different chemical structure, it warrants consideration as a candidate agent for clinical cancer chemotherapy.

We wish to express our appreciation to Dr. Christian S. Rondestvedt, Jr., of the University of Michigan for supplying several of the triazenes, and to Dr. Helene W. Toolan, of the Sloan-Kettering Institute for conducting the bioassays of the H.Ep. #3.

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Received September 12, 1955. P.S.E.B.M., 1955, v90.

Serum Glycoprotein Concentrations in Rats Infected with the Cestode, *Hymenolepis diminuta*. (22075)

HENRY E. WEIMER, MARIETTA VOGE, FRANCES A. QUINN, AND JEAN REDLICH-MOSHIN
(Introduced by Charles M. Carpenter.)

Department of Infectious Diseases, School of Medicine, University of California, Los Angeles.

The serum protein response in a large number of infectious diseases has been studied by many investigators employing electrophoretic or salting-out methods. Gutman(1), Fisher(2) and Stauber(3) have summarized the significant data in their reviews. In contrast, few reports are available on effects of infectious diseases on carbohydrate-containing proteins of serum. Studies concerned with the influence of infection on protein-bound carbohydrates of serum have been briefly reviewed by Weimer *et al.*(4). Diseases in which serum glycoproteins have been determined are characterized by invasion of tissues by the etiologic agent and marked, systemic host reactions. It was of interest to investigate the serum glycoproteins in an experimental infection in which the parasite is localized and invasion of the tissues does not occur.

The present investigation was undertaken to study the effects of experimental infection with *Hymenolepis diminuta* on serum concentrations of total glycoprotein, seromucoid (mucoprotein), γ-globulin polysaccharide, total protein, and γ-globulin protein in rats.

Hematocrit values and concentrations of hemoglobin were also determined in oxalated blood specimens.

Materials and methods. Twenty male and 7 female adult rats of the Long-Evans strain with an average weight of 305 g were infected. Thirteen male and 7 female rats served as controls. Tapeworm eggs were recovered from the feces of rats infected with *H. diminuta,* Rice Institute strain. Cysticercoids were obtained by feeding the eggs to laboratory-reared beetles, *Tenebrio molitor.* The rats were lightly anesthetized with ether and 5 cysticercoids were administered *per os* with a pipette. The animals were housed 2 per cage and were maintained on a diet of Purina Laboratory Chow and tap water, *ad libitum.* Thirty-two days after inoculation the infected rats were sacrificed by exsanguination from the heart and air embolism. The animals were autopsied and the tapeworms counted. Blood samples were obtained from the normal animals by cardiac puncture. Two ml of each of the blood specimens were added to tubes containing a dried mixture of potassium and ammonium oxalates. The remainder of the blood sample was allowed to clot and the serum separated. Total serum glycoprotein, seromucoid, γ-globulin polysaccharide, total serum protein, and γ-globulin protein were determined by methods previously reported(4,5). Hematocrit values were determined in Wintrobe tubes by the procedure outlined by Kolmer(6). Hemoglobin was determined by an acid-hematin method in a Klett-Summerson photoelectric colorimeter (6). The mean, standard error of the mean, t, and probability values were determined by standard statistical methods(7).

Results. A summary of the data is presented in Table I. No significant differences occurred between the normal and the infected group for the serum determinations. The increases in the hemoglobin and hematocrit values were significant at the 5% level.

The lack of effect of a well established infection with *H. diminuta* on the serum glycoproteins and proteins of the rat is in marked contrast to changes observed in other pathologic states of infectious etiology. The increase in hematocrit and in hemoglobin values might suggest a stimulation of the hemapoietic system, or reflect loss of water and plasma protein. Further studies are indicated.

TABLE I. Summary of Results.

Determination	Normal (20)	Infected (27)
Wt at bleeding (g)	343 ±14.0*	345 ±13.8*
No. of worms		3.9± .2
Total serum glycoprotein (mg %)	138 ± 2.0	143 ± 2.0
Seromucoid (mg %)	18 ± 1.2	18 ± .9
γ-globulin polysaccharide (mg %)	31 ± 1.7	30 ± .6
Total serum protein (g %)	6.6± .12	6.6± .9
γ-globulin protein (g %)	1.9± .06	1.9± .06
Hemoglobin (g %)	14.6± .4	16.1± .4
Hematocrit (%)	44.5± 1.2	47.7± .8

* Stand. error of the mean.

Several factors have been suggested as possible causes for the increased concentrations of serum glycoproteins in a wide variety of pathologic and physiologic states: tissue degradation(8), tissue proliferation and repair(9), protein synthesis(10), and nonspecific stress(11). Regardless of the cause of elevated serum glycoprotein levels, it is apparent that none of the responsible factors was operative in the present investigation.

It may be inferred from the results reported above, that a localized infection without tissue invasion has no significant effect on the level of the serum glycoproteins and proteins of the rat.

Summary. The effects of experimental infection with *Hymenolepis diminuta* on the serum concentrations of total glycoprotein, seromucoid, γ-globulin polysaccharide, total protein, γ-globulin protein and on hemoglobin and hematocrit values have been determined in Long-Evans rats. No statistically significant differences occurred in serum values between the normal and the infected groups. An increase in hemoglobin and hematocrit values, significant at the 5% level, was observed in the infected group.

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Received September 13, 1955. P.S.E.B.M., 1955, v90.

Role of Hyperlipemia in the Genesis of Hypercholesteremia.* (22076)

MEYER FRIEDMAN AND SANFORD BYERS.

Harold Brunn Institute, Mount Zion Hospital, San Francisco, Calif.

It has long been known that an hypercholesteremia, either occurring clinically or induced experimentally, is usually accompanied by a rise in the concentration of plasma phospholipid and sometimes also of neutral fat. This frequently observed association of hypercholesteremia with hyperlipemia has not been investigated in respect to the possible *sequential* relationship of these multiple lipid rises. Instead it has been more or less assumed, in lieu of direct evidence, that the lipid rises observed in hypercholesteremia are merely associative phenomena. Recently, however, Allbrink, Man and Peters(1), noting the physical association of plasma cholesterol with neutral fat in chylomicra, have posed the question of whether an excess of plasma neutral fat might not dissolve additional cholesterol thus preventing its inclusion in soluble lipoprotein, leading thereby in some manner to a state of hypercholesteremia.

In view of this last possibility, it was decided to induce a sustained state of hyperlipemia in the rat by continuous parenteral injection of 2 types of lipid mixture (containing only neutral fat and lecithin) and then observe the possible changes in plasma cholesterol resulting therefrom. The results obtained in this study, as herein reported, indicate that a state of hypercholesteremia quickly follows the induction of a prolonged hyperlipemia.

Materials and methods. Male rats (Long Evans strain) 12-16 weeks of age were used in all of the experiments. (a) Production of sustained hyperlipemia was accomplished in rats by rapid intravenous administration of 3 ml of either of 2 different lipid emulsions (B† and Z‡) followed by *continuous* intravenous administration of the same emulsion at 0.9 to 1.2 ml/hour for 12 hours. This continuous intravenous administration to an unanesthetized rat was made possible by its enclosure in the Bollman type of cage(2). Prior to the experiment these rats had been placed for 7-15 days on a diet comprised of Purina Chow enriched with cholic acid (2%) and olive oil (2%). Each rat also received an additional 3 ml of olive oil by stomach tube at 72, 48 and 24 hours prior to the start of experiment. All food was withheld from rats for 24 hours immediately prior to and during the experi-

* Aided by Grants from the Life Insurance Medical Research Fuund, the San Francisco Heart Assn. and Grant H-119 from National Heart Institute, National Institute of Health, Public Health Service.

† B lipid emulsion (kindly furnished by Don Baxter) is composed as follows: Sesame Oil, U.S.P. 10; Dextrose, U.S.P. 4.5; Purified Soya Bean Lecithin 0.65; Water, q.s. 100 ml. Lipid is present as finely divided particles of 1 μ or less in diameter.

‡ Z lipid emulsion (kindly furnished by D. B. Zilversmit of University of Tenn.) is composed as follows: Coconut Oil 10; Purified Soya Bean Lecithin 1.0; Dextrose 5.0; Water, q.s. 100 ml. Lipid particles are approximately the same diameter as in B emulsion.