Average values of all groups for 25 hours after total body irradiation were still elevated but considerably lower than those for 11 hours post-irradiation. At 49 hours post-irradiation the average values for all groups were the same as that observed for pre- and one hour post-irradiation. After 73 hours average GOT values were similar to the pre-irradiation level.

While the above work was in progress, Milch and Albaum published studies of SGOT levels in x-irradiated animals, using dose levels of 500, 750 and 1000 r. Their data demonstrate significant increase in serum glutamic oxaloacetic transaminase activity within 6 hours after receiving 500 r and within 3 hours after receiving 750 and 1000 r. An exact comparison is not possible, because of differences in time intervals used, but our data indicate an average increase 5 hour post-irradiation. Some animals in all groups did not show any response.

All animals that survived the total body x-irradiation were kept for a maximum of 2 weeks post-irradiation. There was no correlation between GOT changes and survival time, (Table II). The data indicate that some animals survived after an increase of several hundred SGOT units while others died showing only mild GOT increase. More animals in the groups which received dosages of 1250 and 1500 r died during the first 24 hours. Although increased SGOT activities may qualitatively indicate tissue damage inflicted by irradiation, survival of the animal appears to depend on ability to overcome and repair the resultant damage.

Summary. New Zealand strain rabbits were given varying dosages of total body x-irradiation. SGOT determinations were made at various intervals in the post-irradiation period up to 168 hours following irradiation. Initially the blood specimens were drawn by direct cardiac puncture and the results in these instances were too variable to be used for diagnostic or prognostic purposes. When the free bleeding technic was used for obtaining blood specimens, more consistent results were obtained. Average SGOT values for pre- and one hour post-irradiation were of the same order of magnitude. An increase in GOT activity was observed as early as 5 hours after total body irradiation and remained elevated for 25 hours. After 49 hours post-irradiation SGOT levels returned to normal. There was no correlation between SGOT activity and total irradiation or survival time of the animal.

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Effect of Histone on Plasma Unesterified Fatty Acids.* (23990)

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Recognition of the physiological importance of unesterified fatty acids (UFA) followed the observation of the UFA-increasing effect of heparin(1,2,3), and reports establishing UFA as available metabolites(4,5,6,7). Protamine was found to counteract the effects of heparin(7,8,9). In the present studies the effects of histone on UFA metabolism were investigated. Histone, like protamine, is a positively charged molecule, but unlike protamine it is widely distributed physiologically.

Methods. Alimentary lipemia was pro-

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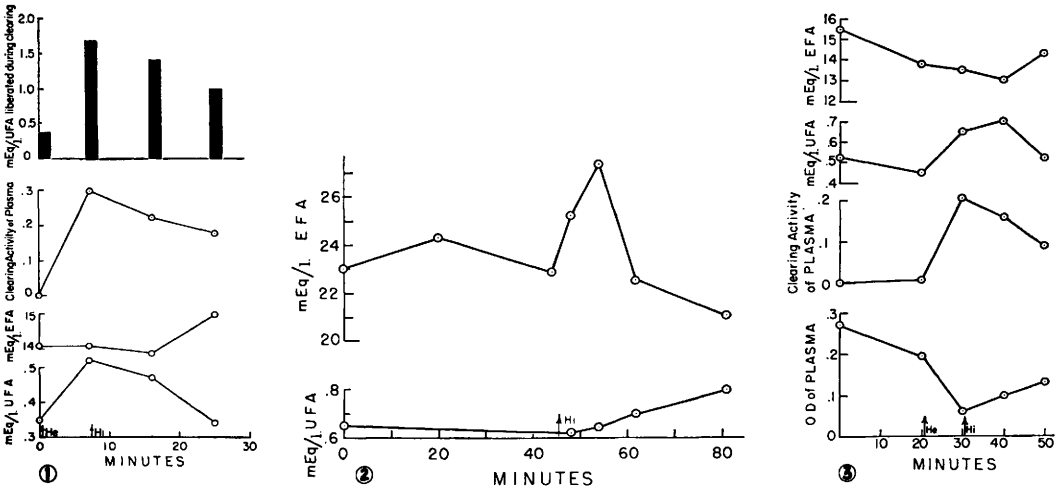

FIG. 1. Effects of 100 mg histone (Hi) following 6 mg heparin (He) in a nonlipemic dog.
FIG. 2. Effects of 90 mg histone (Hi) in a lipemic dog.
FIG. 3. Effects of 50 mg histone (Hi) following 6 mg heparin (He) in a lipemic dog.

duced in mongrel dogs by feeding about 40 ml of cod liver oil. The oxalated blood samples were promptly refrigerated and centrifuged in the cold. UFA were determined by the method of Grossman(10). Esterified fatty acids (EFA) were estimated by the procedure of Stern and Shapiro(11). All analyses were performed in duplicate. Histone, from calf thymus nuclei (Mann Research Laboratories), was injected intravenously in 2.5 to 5.0 mg/kg doses, either with or without previous intravenous injection of heparin (about 0.3 mg/kg). Animals that did not receive heparin showed an untoward reaction to histone that lasted about one minute. Clearing activity of the plasma was determined by clearing in optical density units of 1 ml of cottonseed-oil emulsion by 0.5 ml of plasma after 2 hours' incubation at room temperature.

Results. Administration of histone to nonlipemic dogs did not influence either UFA or EFA levels of plasma (4 exp.). When histone was administered following injection of heparin into nonlipemic dogs, the heparin-induced changes, such as clearing activity of plasma and elevation of UFA, were counteracted by histone (3 exp.), (Fig. 1).

In 8 experiments on lipemic dogs, histone alone increased visible lipemia, and, in most cases, blood EFA, but it had no consistent effect on UFA (Fig. 2). When heparin preceded administration of histone in lipemic dogs, the heparin-induced changes were again counteracted by histone; visible lipemia and EFA were increased, clearing activity of plasma and UFA were decreased (Fig. 3) in 6 experiments. In control experiments on both normal and lipemic animals the effect of heparin lasts over 30 minutes (Figs. 2-6 and 9-11 of ref. 7, and Figs. 1 and 3 of ref. 9).

Discussion. UFA is liberated by clearing factor(1,2,3) or by tissue lipases(12,13,14,15) and is removed by tissues(4,5,7). Both clearing factor(8,9) and tissue lipoprotein lipase (12,13) are inactivated by protamine. Histones, which unlike protamines, are physiologically widely distributed, have an action similar to that of protamine on lipid transport. The latter finding makes it very tempting to speculate that UFA-liberation may be under the influence of an intracellular lipase-histone equilibrium.

Summary. Histone influenced lipid transport in the same manner as protamine. In nonlipemic dogs, it failed to influence unesterified or esterified fatty acids, reversed the action of heparin on clearing activity, and opposed the elevation of unesterified fatty acids. In lipemic dogs, histone increased lipemia without affecting the unesterified fatty acids. In similar animals after heparin, histone reversed the action of heparin, increasing li-

pemia and decreasing clearing activity of the plasma and unesterified fatty acids.

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Effect of Zinc upon Growth and Incidence of Perosis in Turkey Poults.* (23991)

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Todd *et al.*(1) and Stirn *et al.*(2) reported that zinc is indispensable in the nutrition of the rat. Follis *et al.*(3) studied the histology of zinc deficient rats and noted a parakeratosis as a characteristic symptom. Tucker and Salmon(4) reported that a parakeratosis syndrome was noted in swine on rations containing 34 to 44 ppm of zinc. The incidence was increased by increased levels of calcium and/or phosphorus. The syndrome was prevented and growth improved by supplementing the ration with zinc. Zinc was also found to be effective in preventing the parakeratosis at lower levels of calcium in practical rations by Luecke, *et al.*(5) and purified protein (Drackett) diets by Conrad and Beeson(6). Morrison *et al.*(7) obtained no growth response to zinc with chicks fed a purified ration containing an isolated soybean protein (Drackett C-1). Mehring *et al.*(8) also failed to observe any increased growth by addition of zinc to a practical type ration for chicks. O'Dell and Savage(9) observed increased growth of chicks fed a Drackett protein basal ration with relatively high levels of calcium and phosphorus when zinc carbonate was added. Norris *et al.*(10) also obtained a slight growth response in chicks with a similar type ration, except that lower calcium and phosphorus levels were used and concluded that the requirement of chicks for zinc was in the order of 20 to 25 ppm. Hunt *et al.*(11) and Butters and Scott(12) noted an enlarged hock condition in turkey poults fed purified rations with isolated soybean protein (Drackett 220 or C-1) as source of protein. It could be prevented by substituting soybean oil meal for Drackett protein or by adding soybean oil meal ash to purified rations(12). Slinger *et al.*(13) noted no improvement in growth of poults by addition of zinc to a practical ration. Supplee *et al.*(14) observed that zinc and potassium additions to a purified ration containing Drackett protein were effective in reducing the incidence of the enlarged hock disorder and in improving growth. The pres-

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