The biochemical characterization of the measles antigen with trypsin sensibility, ether sensitivity and resistance to RNA-ase and DNA-ase suggest that the antigen consists mainly of lipoproteins. These lipoproteins are probably constituents of the viral capsid.

It can be postulated that the small particles are debris of unused constituents appearing during viral synthesis.

The receptors for measles virus are destroyed by formalin treatment of red cells. Erythrocytes previously agglutinated by myxoviruses are still agglutinable by measles virus. These findings show that receptors for measles virus are different from the receptors for myxoviruses. In addition, measles virus does not elute spontaneously from the red cells unlike myxoviruses.

Summary. Hemagglutination of monkey red cells was demonstrated with a concentrated measles tissue culture antigen. The antigen was destroyed by trypsin and its activity was significantly reduced by ether. Heat inactivation at 60°C for 10 minutes destroyed the infectivity, while hemagglutinating activity was significantly reduced only after one hour. A small non-infectious hemagglutinating unit was also demonstrated. Both hemagglutinating particles are antigenic and their activity was inhibited by specific antiserum. The small particles appeared during viral multiplication in the cell. Receptors on the red cells were of a different nature than those of myxoviruses.

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Localization of Anti-Rat Kidney Antibodies in New-Born Rats.* (27556)

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Experimental nephrotoxic nephritis has been demonstrated by many investigators to occur in rats injected with anti-rat kidney antisera prepared in rabbits. Calcagno and coworkers(1,2) have reported that new-born rats do not develop the clinical disease even when injected with 4 times the dose (on a weight basis) of rabbit anti-rat kidney serum required to produce disease in adult rats. At 2 weeks of age clinical disease could be produced with twice the dose required for adults. Moreover, if the rats injected as newborn were reinjected at 3 to 14 weeks of age only about half of them showed clinical disease although previously uninjected 6-week-old animals all develop the disease at the dosage given.

Since the new-born rats could not develop clinical disease when injected with nephrotoxic serum which was capable of inducing experimental nephritis in the adult rat, studies were carried out to determine whether

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or not rabbit globulin from nephrotoxic serum fixed to the glomeruli of the new-born rat kidney. Further it would be of interest to determine the temporal and quantitative relationship of this antigen-antibody attachment.

Experimental. The rats used were of the Wistar strain. The anti-rat kidney serum was prepared in rabbits using the protocol described by Heymann *et al.*(3). New-born rats (1 to 3 hours of age) were injected intra-

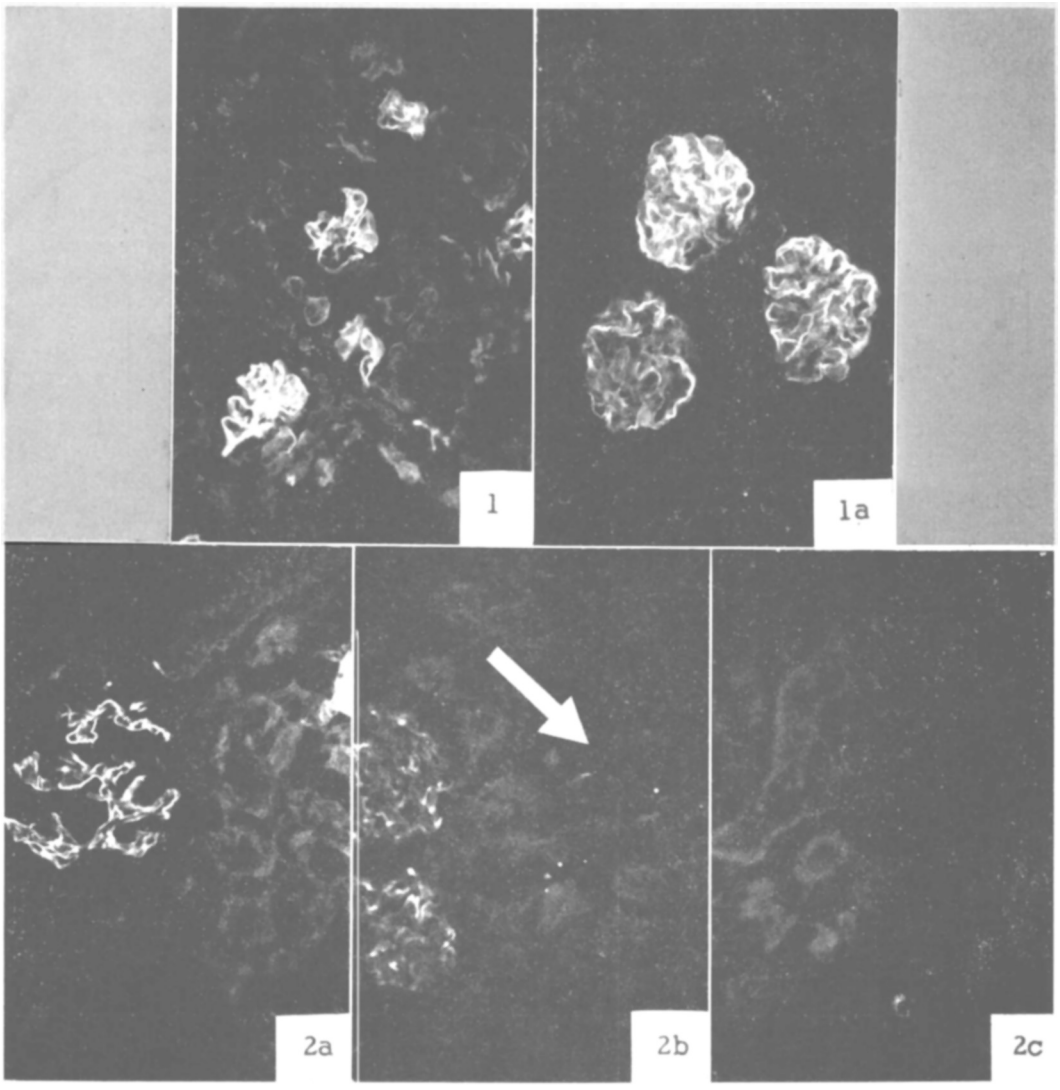

FIG. 1. Kidney section from a new-born rat inj. with anti-rat kidney serum and sacrificed 24 hr later. The glomeruli show fluorescence. They are smaller in size and not as well developed as in adult animals.

FIG. 1a. Kidney section from a 2-wk-old rat inj. with anti-rat kidney serum and sacrificed at 24 hr. The glomeruli show fluorescence. The glomeruli are larger and more developed than those in the newborn.

FIG. 2. A kidney section from a 6-wk-old rat which had been inj. with G-anti-RK at birth. The section was treated with fluorescein labeled horse anti-rabbit globulin. The single glomerulus visible in the field on the left (2a) shows intense fluorescence. This field was situated closest to the medulla of the kidney. As one approaches the cortex less fluorescence of the glomeruli is seen (center field, 2b). Two partially stained glomeruli are visible in the field. A third glomerulus with almost negligible streaks of fluorescence can be seen to the right (arrow). The field on the right (2c) is from the outer region of the cortex. None of the glomeruli showed fluorescence.

venously with 0.2 ml of anti-rat kidney serum. Injection was into the superficial temporal vein.

The kidneys were removed at the appropriate time, frozen without perfusion, sectioned, washed, and stained with fluorescein or rhodamine labeled horse anti-rabbit globulin antibodies according to the procedure described previously(4).

Results. To determine whether antikidney antibodies are fixed in the kidney of new-born rats, 2 were injected intravenously with 0.2 ml of rabbit anti-rat kidney serum prepared against adult rat kidney. As a control, 2 new-born rats were similarly injected with normal rabbit serum. The rats were sacrificed 24 hours after injection. The kidneys of the rats receiving the antikidney serum showed good glomerular localization (Fig. 1). The glomeruli were small and not as well developed as those in adult rats. All the glomeruli appeared to be involved. The control animals receiving normal rabbit serum showed no localization of rabbit globulin in any of the glomeruli. Rats, 2 weeks old similarly injected with antikidney serum, showed good localization in the glomeruli (Fig. 1a).

In view of the reported observation that only some of the rats injected with anti-RK when born develop the disease when injected again 6 weeks later, 2 new-born rats were injected with anti-RK serum as above and allowed to survive 6 weeks, to determine the fate of antikidney antibodies after fixation in new born. At this time one was challenged with antikidney serum. Then both were sacrificed and their kidneys assayed.

The kidneys of the animal that had received anti-rat kidney serum only at birth showed good localization of the antikidney antibody which had been administered 6 weeks prior. Localization was seen in all the glomeruli situated close to the medulla of the kidney whereas no fluorescence was seen in glomeruli situated toward the periphery of the kidney (Fig. 2). This experiment shows that antikidney antibody produced against adult rat kidney is fixed in the glomeruli of new-born rats. Antibodies are still present in glomeruli even after 6 weeks, which is in accordance with the long half-life of antikidney antibody previously observed in adult rats(5). The kidneys of the rat receiving the second injection showed antikidney antibody localized in all glomeruli.

This experiment is in accord with the observation of Arataki(6) that the kidneys of new-born rats contain about 10,500 glomeruli and the number increases to 24,200 in 24 days. Therefore, from birth to weaning at 20 days, the number has more than doubled. The increase occurs in the periphery. Our results are easily explained by this observation. The antibody injected in the new-born rat remained in those glomeruli in which it localized originally. In the meantime new glomeruli became functional on the periphery resulting in the picture we found, that is, antibody localization was demonstrated in the glomeruli near the medulla, but none in the glomeruli in the periphery of the cortex. In the animal reinjected at 6 weeks there was localization in the glomeruli.

The failure to demonstrate attached antibody in all of the glomeruli 6 weeks after administration of nephrotoxic serum to new-born rats suggests a delay in functional maturation of some glomeruli or alternatively a dissociation of any antigen-antibody attachment during the 6-week interval. The last possibility is contrary to the observed long life observed for antibody fixed in rat kidney (5).

The failure to produce clinical disease and biochemical abnormality in new-born rats injected with nephrotoxic serum suggests the following possibilities: (a) The antigens giving rise to cytotoxic antibodies are not present in the young new-born rats, therefore the rabbit antibody observed to be fixed at this time in the glomeruli is not the one which is associated with cytotoxic effects. (b) The young rats are deficient in complement(7)† which is a necessary component to induce cytotoxicity. (c) There is no secondary pro-

† We have compared dilutions of pooled serums from new-born rats with the respective mothers and pooled normal guinea pig serum, and in every case the new-born serum although showing the presence of complement failed to lyse sensitized sheep erythrocytes to the extent as the adult rat or guinea pig serums.

duction of rat anti-rabbit globulin antibodies in the new born, such as is known to be a factor in the development of disease in adult rats(8). (d) The failure to produce clinical disease in newborn rats injected with nephrotoxic serum may be due to the fact that new glomeruli open as the animal matures.

Summary. Rabbit antibodies against adult rat kidney were found to be fixed in the kidneys of new-born rats. Since new-born rats do not develop clinical disease when injected with anti-rat kidney antibodies, this suggests either that the antigens giving rise to cytotoxic antibodies are not present at this early date or the new-born rats are deficient in complement. Six weeks after new-born rats are so injected they still have the rabbit globulin (antibody) in the glomeruli. However, rabbit globulin was demonstrated in the glomeruli on the medullary side of the cortex and not in those of the periphery indicating that new glomeruli in the peripheral region become functional as the animal matures. The increase in functional glomeruli may be the reason for the observation that new-born rats injected with anti-rat kidney serum do not develop clinical disease.

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Hormonal Control of Mammary Gland Involution in the Rat.* (27557)

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Previous studies in this laboratory have employed desoxyribonucleic acid (DNA) as a quantitative index of normal and experimental growth of rat and mouse mammary glands(1,2,3,4,5). Present investigation was undertaken to study effects of oxytocin (OXT), lactogen‡ (LGH), and hydrocortisone acetate (HCA) on mammary gland involution in the rat. Morphological methods (6) have demonstrated that all these hormones will retard retrogression of lobule-alveolar system of rats. The question arises whether the hormones actually retard cellular degeneration or only maintain milk synthesis in remaining secretory cells. Since DNA is an index of cellular number, it is possible to answer this question by its determination.

Materials and methods. Primiparous Sprague-Dawley Rolfsmeyer rats were employed in present experiments. Young were separated from control and experimental animals on day 3 of lactation, and dams were injected for a 10-day period. Experimental animals received (a) 2 USP units of OXT, either intraperitoneally (ip) or subcutaneously (sc), 3 × daily (b) 1 and 2 mg LGH sc (c) 100, 500 and 1000 μg of HCA sc from day of weaning. Animals were killed on day

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