the temperature of sensitization; preparations obtained from thyroid powder sensitized at 4°, 23°, and 37° C. were equally potent. Eluates from similarly treated powder of human organs other than thyroid did not contain thyroid antibodies. Serological reactions with eluates of thyroid antibodies were weaker than the reactions with the corresponding original sera.

The first elution was never complete and two to three consecutive eluates still showed marked antibody activity. Antibodies against human serum proteins present in rabbit anti-human thyroid sera were reduced to traces in eluates.

The eluates of human thyroid autoantibody effectively inhibited the activity of rabbit anti-human γ-globulin serum in bringing about the agglutination of Rh^+ red blood cells sensitized by incomplete Rh antibody. In addition, sera of two rabbits immunized intravenously with these eluates acted as potent Coombs reagents. These experiments established the fact that eluates contained γ-globulin.

If desired, the eluates of thyroid antibodies might be concentrated by freeze-drying or evaporation and further properties of the tissue-reactive antibodies studied.

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Transfer of Nephrotoxic Glomerulonephritis in Rats by means of White Blood Cells

Transfer of experimental glomerulonephritis on originally healthy rats has been accomplished in this and other laboratories by means of parabiosis[1,2]. Transfer was observed in random-bred as well as in inbred rats, either in short-term experiments or after intervals up to 71 days between institution of nephrotoxic serum glomerulonephritis in the primarily nephritic animal, and parabiotic conjunction with the originally healthy partner[3–5]. Fluorescein-labelled antisera were used to exclude the involvement of the primarily injected heterologous (rabbit) antibodies in the transfer phenomenon[6]. From these experiments it was derived that a secondary nephritis-producing factor, appearing as a consequence of the nephrotoxic serum glomerulonephritis in the circulation of the primarily nephritic rat, was transferred to the primarily healthy animal capable of attacking the corresponding organs of the latter.

This conclusion was supported by recent experiments which were an attempt to localize the transfer factor in question. First, the parabiosis was substituted by continuous cross-transfusion (that is, bilateral carotid artery-jugular vein anastomosis), and glomerulonephritis was transferred on 5 out of 15 healthy rats following cross-circulation of 2·5–4 days duration[7]. Secondly, plasma and white blood cells from nephritic rats were injected (for 4–15 days) or infused (by using a modification of the technique of Armin *et al.*[8] for continuous infusion of solutions in unanæsthetized animals) into healthy recipients. As in the former experiments, a highly inbred strain of Sprague–Dawley rats was used consistently. Development of experimental glomerulonephritis was observed in 5 out of 31 recipients following transfer of 210–450 million white blood cells from 4–10 nephritic donors, only in 1 out of 32 animals receiving 10–35 ml. of plasma from the same donors, the controls, 52 recipients of plasma and white blood cells from healthy donors, being completely negative.

Since in rats 75–80 per cent of the white blood cells are lymphocytes and transfer of experimental 'autoimmune-nephrosis' on immunologically tolerant rats recently was achieved by injecting lymph node cells[9], the results reported herewith are in favour of a lymphocyte-bound nature of the secondary nephritis-producing factor in nephrotoxic serum glomerulonephritis likewise in rats. In contrast to the transfer of an autoimmune-nephrosis, however, Freund's adjuvants was not used in our experiments. They suggest, on the other hand, the induction of an autologous hypersensitivity reaction of the delayed type following a passive heterologous anaphylaxis in the glomeruli. Possibly this secondary mechanism is responsible for the progression of the disease, once it has started. Further work is required for elucidating the immunological nature of the secondary nephritis-producing factor in question.

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BIOLOGY

Significance of Mitotic Duration in evaluating Kinetics of Cellular Proliferation

Mitotic rate or 'rate of cell renewal' can be defined as the number of cells which complete their mitosis per unit time.

$$\text{Mitotic rate} = \frac{\text{mitotic count}}{\text{mitotic duration}}.$$

As can be seen from this equation, and as also emphasized by Leblond and Walker[1], mitotic count can be used to estimate mitotic rate only if mitotic duration is not significantly influenced by the experimental conditions.