hibited indentations of their luminal surfaces, probably as representations of their discharged secretion (Fig. 4).

In the early luteal phase (Fig. 2) the tubal epithelium had a surface topography similar to that found in the proliferative phase. The ciliated cells were more prominent on the summits of the tubal plicae, and this suggested that cilial transport occurs principally in the central tubal lumen with secretory activity being more concentrated in the plical crypts. The finding of a relatively uniform distribution of the various cell types throughout the entire length of the tube is in agreement with the report of Hashimoto *et al.* (*3*) and in contrast to those of Clyman (*2*) and of Woodruff and Paurstein (*1*) who reported more ciliated cells in the fimbria. The smaller peg cells, found by transmission microscopy to be compressed between the larger ciliated and secretory cells, have been identified with a reserve or precursor function. It may be that this is not so and that the peg cells represent exhausted secretory cells.

Secretory activity occurred in all portions of the oviduct. Small cytoplasmic buddings were occasionally seen on the surface of secretory cells. The secreted material, once detached from the cell, resembled mucus droplets and most likely correspond to the periodic acid Schiff test-positive, diastase-resistant (*5*) secretory substances seen in the secretory cells at the fine structural level (*2*, *3*).

Atrophy occurring after the menopause apparently affects both the ciliated and nonciliated cells of the oviduct (Fig. 3). Only rarely are tufts of cilia encountered. The greater part of the oviduct's surface is lined by cells having the domed apices with fine microvilli characteristic of the secretory cells. These rarely exhibit the bulging contours or collapsed appearances identified during the cycle as evidences of secretion. In scattered locations, this cobblestone pattern is interrupted by areas of flattened cells. The hexagonal pattern and prominent terminal bar attachments of these flattened cells resemble the arrangements of mesothelia (Fig. 3). The flattened cells probably represent atrophied tubal cells.

ALEX FERENCZY
RALPH M. RICHART
FREDERIC J. AGATE, JR.
MABEL L. PURKERSON
EDWARD W. DEMPSEY
Departments of Pathology and
Anatomy, Columbia University College of Physicians and Surgeons, New York 10032

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6. Supported by NIH grants GM 15289 and AM 50950 and a grant from the Association for the Aid of Crippled Children.

9 August 1971

Inhibition of Antibodies to Nuclear Antigen and to DNA in New Zealand Mice Infected with Lactate Dehydrogenase Virus

Abstract. *New Zealand mice developed antibodies to nuclear antigen leading to immune-complex nephritis. Humoral antibody was directed primarily against denatured DNA, although antibody to native DNA was also found. Persistent infection with lactate dehydrogenase virus significantly lowered antibodies both to nuclear antigen and to DNA in these mice. In addition, female (NZB × W)F_1 mice infected with lactate dehydrogenase virus were protected from the usual nephritic death occurring after the trapping of complexes of nuclear antigen and its antibody and of DNA and its antibody in the glomerular filter.*

New Zealand (NZ) mice serve as a model of human systemic lupus erythematosus. A certain proportion of these mice [NZB, NZW, (NZB × W)F_1] develop autoimmune disease manifested mainly by antibodies to nuclear antigen (ANA) and antibodies to red blood cells, subsequently accompanied by immune-complex glomerulonephritis and autoimmune hemolytic anemia, respectively (*1–3*). While the etiology of the development of the ANA response is not clear, it appears most likely that immunologic hyperresponsiveness of NZ mice to nucleic acid antigens is an important predisposing factor. In our laboratory we have shown that persistent infection with either of two dissimilar viruses, lymphocytic choriomeningitis virus (an RNA virus) and polyoma virus (a DNA virus), enhances ANA formation and aggravates the immune-complex glomerulonephritis (*1*, *4*). Others have reported that immunization with DNA or synthetic polynucleotides enhances both ANA and glomerulonephritis (*3*, *5*, *6*).

We report now that persistent infection with lactate dehydrogenase virus (LDV), a relatively noncytopathic RNA virus, significantly decreased the production of both ANA and antibody to DNA in NZ mice. In addition, 9-month-old (NZB × W)F_1 females infected with LDV over a long period were protected from the nephritis and death that usually occurs as a result of nuclear antigen-ANA complexes or of DNA–antibody to DNA complexes being trapped in the glomerular filter.

The NZB and NZW mice were originally obtained from the Laboratory Animal Center, Medical Research Council, Surrey, England, and were inbred in our laboratory by brother-sister mating. The NZB females were crossed (at random) with NZW males and vice versa. Periodic testing indicated that the mice were free of infection with lymphocytic choriomeningitis virus, polyoma virus, and LDV. The persisting LDV infection was induced by inoculating 4-, 10-, or 12-week-old mice intraperitoneally with 10^5 infectious doses (ID_{50}) of LDV prepared from pools of isologous serums. Details of virus passage, inoculation procedures, and development and testing for LDV infection have been described (*7*, *8*). Tests for ANA and for clinical and immunopathologic changes associated with nephritis have been reported (*3*, *4*, *9*). Antibodies directed against denatured and native DNA were measured in samples of plasma from noninfected and LDV-infected NZ mice by means of a modified Farr technique (*10*). Briefly, the ability of various dilutions of NZ plasma to bind immunospecifically with H^3-labeled DNA prepared from either SV20 virus, adenoviruses, or mouse L cells was determined. By this procedure, it is possible to detect as little as 20 to 30 ng of antibody to DNA.

NZW mice have ANA and glomerulonephritis, but the incidence and severity are lower than in the NZB × W hybrid. Normally, in NZW mice the incidence of ANA increases with age. In contrast, NZW littermates infected with LDV over a long period show no increased incidence of ANA (Fig. 1).

Table 1. Effect of persistent LDV infection on antibody to DNA in NZW and (NZB × W)F_1 mice. Antibody to DNA was detected by a primary antigen-binding technique (*10*). Briefly, the ability of various dilutions of NZ mouse plasma to bind immunospecifically with [H^3]DNA was determined. Anti-N-DNA refers to antibodies directed against native DNA, and anti-H-DNA refers to antibodies directed against denatured DNA. In most cases, plasma from individual mice was assayed. Both NZW and (NZB × W)F_1 mice were infected with LDV at 1 month of age.

Group	Mice (No.)	Binding (μg antigen/ml serum)	
		Anti-N-DNA	Anti-H-DNA
NZW aged 9 months			
♀ Noninfected	24	0.14*	4.90
♀ LDV infected	17	0.00	0.30
♂ Noninfected	16	0.00	0.68
♂ LDV infected	22	0.00	0.00
(NZB × W)F_1 aged 3 months			
♀ Noninfected	25	0.02	5.90
♀ LDV infected	21	0.00	0.50
♂ Noninfected	32	0.10	0.90
♂ LDV infected	19	0.00	0.30

* Result is the arithmetic mean value of all mice in the group.

Hence, the percentage of control female NZW mice having ANA increased from 25 percent at 3 months to 35 and 52 percent by 6 and 9 months of age, respectively. In comparison, test female mice infected with LDV at 3 months of age showed no further increase by 6 or 9 months. Noninfected NZW males increased from an incidence of 5 percent ANA at 3 months to 21 and 27 percent at 6 and 9 months, respectively, whereas NZW males infected with LDV at 3 months maintained an incidence of 5 percent ANA through 9 months of age. There was no significant difference in the incidence of ANA between noninfected and LDV-infected NZB × W hybrids. However, the plasma from noninfected male and female (NZB × W)F_1 contained four to five times more ANA than that of LDV-infected mice, as judged by comparison of the arithmetic mean of the end-point dilutions needed for a positive test.

The antibody to DNA found in NZ mice was directed primarily against denatured DNA, although some antibody to native DNA was also found. In NZ mice, the amount of antibody to DNA found in LDV-infected mice was one-third or less than that found in noninfected controls (Table 1).

Of all the NZ mice (NZB × W)F_1 females develop the severest immune-complex glomerulonephritis. Thus, by 9 months of age, uninfected control (NZB × W)F_1 females have an expected mortality due to immune-complex glomerulonephritis of 72 percent. Table 2 shows that (NZB × W)F_1 females persistently infected with LDV from 4 to 10 weeks of age on had only a 12 or 44 percent mortality at 9 months, respectively. Hence, long-term infection with LDV significantly protected (NZB × W)F_1 females from death. Among the dying mice, the primary histopathologic finding was severe glomerulonephritis in both noninfected and LDV-infected mice.

Our observations indicate that LDV has done something to decrease ANA and antibody to DNA. As LDV has been reported to elevate some serum enzymes (*7, 11*), the possibility exists that nuclear antigens, particularly DNA, may be rapidly degraded by an increase in serum deoxyribonuclease or a decrease in deoxyribonuclease inhibitor. In addition, LDV may directly affect the host's immune response in other ways. LDV grows in cells of the reticuloendothelial system, presumably macrophages, and is associated with a proliferation of germinal center lymphocytes and a hypergammaglobulinemia (*12, 13*). Unlike the leukemia viruses which cause nonspecific immunosuppression of the immune response to a variety of antigens (*14*), LDV may cause either immunoenhancement or immunosuppression. In a yet unknown way, LDV can increase the primary immune response and prevent the development of immunologic tolerance to human gamma globulin (*12, 15*). On the other hand, LDV inhibits allograft rejection and the graft-versus-host reaction (*16*). Hence, it is apparent that LDV can profoundly influence the immune response of the host.

Release of both specific and nonspecific antiviral factors during LDV infection probably plays little, if any, role in the suppressed ANA response. LDV infection is associated with only a weak and transient production of interferon, while injection of interferon inducers in NZ mice potentiates immune-complex glomerulonephritis (*6*).

Greenwood and Voller (*17*) reported

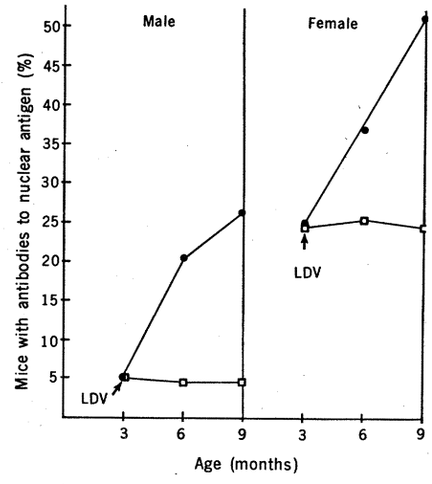

Fig. 1. The effect of persistent LDV infection on the ANA response of NZW mice. NZW mice were infected intraperitoneally at 3 months of age with 10^5 ID_{50} of LDV prepared from pooled isologous serums. ANA was detected by the indirect immunofluorescent antibody technique with acetone-fixed mouse kidney sections as the target cells (●—●, noninfected; □—□, LDV-infected).

the suppression of ANA and glomerulonephritis in (NZB × W)F_1 female mice infected at 4 weeks of age with the malaria parasite, *Plasmodium berghei yoelii*. In that the parasitemia lasted for 2 weeks only, while suppression of autoimmune phenomena continued throughout the animal's life, their results may not have been due to the malaria parasite per se, but rather to a contaminating agent in their red blood cell inoculum. Actually, LDV has been a frequent contaminant of tissue- or blood-passaged murine preparations (*7, 11*).

Our observations, in addition to the previous report of viruses enhancing ANA responses (*1, 4*), have significant importance for those workers studying NZ mice and may explain some of the variability in ANA responses reported among various laboratories. The wide dissemination and relative asymptomatic course of mice infected throughout their life with LDV, lymphocytic choriomeningitis, polyoma, or other such previously unsuspected viruses make them a particular problem. In ad-

Table 2. Survival of 9-month-old (NZB × W)F_1 female mice infected intraperitoneally with 10^5 ID_{50} of LDV at 4 or 10 weeks of age.

Infection	Died	Survived	Mortality (%)	*P*
None	70	27	72	
LDV, 4 weeks	3	22	12	> .001
LDV, 10 weeks	24	30	44	> .001

dition, understanding the mechanism by which LDV decreases ANA and antibody to DNA may offer possible leads for the suppression of ANA responses in man.

MICHAEL B. A. OLDSTONE
FRANK J. DIXON
Department of Experimental Pathology, Scripps Clinic and Research Foundation, La Jolla, California 92037

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5 August 1971; revised 6 October 1971

Photosynthetic Adaptation to High Temperatures: A Field Study in Death Valley, California

Abstract. *The photosynthesis of* Tidestromia oblongifolia *(Amranthaceae) is remarkably well adapted to operate at the very high summer temperatures of the native habitat on the floor of Death Valley. The photosynthetic rate was very high and reached its daily maximum when the light intensity reached its noon maximum at the high leaf temperatures of 46° to 50°C which occurred at this time. At the intensity of noon sunlight the rate decreased markedly when the leaf temperature was experimentally reduced to below 44°C. The optimum rate occurred at 47°C. At this temperature the photosynthetic rate was essentially directly proportional to light intensity up to full sunlight.*

The intense heat and the very limited supply of water that prevail during the summer in many of the world's desert regions impose special demands on plants occupying such habitats. Most species escape the requirement of efficient photosynthesis and other metabolic activity under such severe conditions by carrying out most or all of their photosynthesis and growth during milder periods of the year. However, some higher plant species grow primarily during the hot summer. Obviously these species are uniquely suited for investigations of photosynthetic adaptation to high temperature. We report here the results of a study of the photosynthetic performance of one such species, *Tidestromia oblongifolia* Wats (Standl.) in its native habitat on the floor of Death Valley, California, during the first week of July 1970. The results show that the photosynthetic apparatus of this species is remarkably well adapted to function at the extremely high temperatures prevailing in its native habitat; even at leaf temperatures as high as 50°C the plant was capable of photosynthetic rates as high as those reported for any species under much more favorable conditiions and far greater than for other species growing in hot, arid environments.

Tidestromia oblongifolia is a low-growing herbaceous perennial which occupies dry sandy places, such as washes, below an altitude of 700 m throughout the Colorado and eastern Mojave deserts of the southwestern United States. It is very common on the floor of Death Valley, particularly on the foot of alluvial gravel fans. Earlier observations in this habitat indicated that this plant carries out most of its growth during May through August and dies back to at most a few basal leaves during the mild winter.

The floor of Death Valley is the hottest and driest part of the Western Hemisphere and one of the most severe habitats in the world. Long-time weather records show that it receives an average of 42 mm of rain per year, of which 75 percent falls during November through April (*1*). Air temperatures are consistently very high during the months of May through September, and a temperature of 50°C or higher is not uncommon during these months. The long-time average daily maximum temperature for the hottest month, July, measured at the U.S. Weather Bureau Station at Furnace Creek (54 m below sea level) is 47°C, and the average daily mean temperature is 39°C. Temperatures measured at Badwater, 85 m below sea level and 26 km south of Furnace Creek, are as high as or even higher than those at Furnace Creek. Ground surface temperatures may be more than 25°C higher than those measured at standard height (*1*).

Our experimental site was located on a gravel fan, 60 m below sea level and 2.5 km south of Furnace Creek, just off the road to Badwater. The meteorological data measured at Furnace Creek should therefore be approximately valid also for the experimental site. Our temperature measurements at this site during the period of the investigation closely agreed with those recorded by the U.S. Weather Bureau Station at Furnace Creek.

The vegetation of the gravel fan on which the site was located is characterized by three species: *T. oblongifolia, Atriplex hymenelytra,* and *Euphorbia* sp. These species occur predominantly in small washes crossing the gravel fan. Except in phreatophytic zones, plants are sparsely distributed on the gravel fans in this area at densities of about 125 per hectare (*2*). Anatomical examination of the leaves showed that all three species at this