agranular muscle fibers. The nerve endings are usually retracted in the dark, coarsely granular fiber and expanded in the light, agranular fiber, as revealed by gold impregnation of teased whole muscle fibers. The atrophy of disuse following tenotomy of the gastrocnemius muscle with nerve supply intact, is accompanied, during the first month, by the progressive loss of the narrow and dark, coarsely granular muscle fiber, and by a depletion of its innervation. The dark, granular muscle fiber is determined by the reciprocal interaction of the normally intact and attached muscle fiber with its normally functioning innervation. During the process of atrophy of disuse after tenotomy, small and giant fusiform neurosomes are discharged from the altered nerve endings. It is assumed that these giant fusiform neurosomes are the product of a retardation in the rate of the discharge, diffusion, and disappearance by hydrolysis, following tenotomy and disuse atrophy of the innervated gastrocnemius muscle. There appears to be a parallelism between the atrophy by disuse of tenotomized muscle and the loss of the normal discharge of neurosomes from the altered and progressively depleted innervation of the muscle. One factor in the atrophy of disuse of muscle appeared, therefore, to be the substantial loss of the discharge of neurosomes into muscle as well as the quantitative decrease of the myoplasm. The giant fusiform neurosomes that appear during the early period following tenotomy disappear when the living muscle *in situ* is adequately restretched prior to excision and gold impregnation.

15745

Ornithosis in Sea-Shore Birds.*

MORRIS POLLARD. (Introduced by Ludwik Anigstein.)
From the University of Texas, Galveston, Texas.

P. [illegible]ke infections have been reported [illegible] a [illegible]ge variety of wild and domesticat[illegible] birds including parrots, finches, pigeo[illegible] chickens and turkeys.[1] Because of the w[illegible]e prevalence of psittacosis-like infection among many species of wild and domesticated birds, Meyer proposed that the more general term "ornithosis" be employed in referring to nonpsittacine infections. It has not yet been found in partridges, quail or wild ducks.[2] Among the sea birds, the Fulmar petrel has been incriminated as the source of an epidemic of psittacosis in human inhabitants of the Faroe Islands.[3] Many of the avian species in which this disease is present ordinarily do not manifest clinical symptoms of the disease but carry the virus in "silent" fashion. This dormant infection of birds appears to be provoked to activity by conditions associated with crowding, malnourishment, or other effects of commercial aviary mismanagement. The disease might also be regarded as a natural population controlling factor, remaining inapparent until overpopulation and undernourishment permit the infection to become activated and aggravated in virulence.

Psittacosis (ornithosis) is an important public health problem; so it is important that the natural or potential reservoirs of the disease be known. A study of the incidence of ornithotic infections among sea shore birds

* Sincere appreciation is expressed to Drs. R. W. Strandtmann and C. U. Dernehl for their assistance in the capture and identification of the birds used in this study.

1 Meyer, K. F., Psittacosis and Ornithosis, in *Diseases of Poultry*, The Collegiate Press, Inc., Ames, Iowa, 1944.

2 Eddie, B., and Francis, T., Jr., PROC. SOC. EXP. BIOL. AND MED., 1942, **50**, 291.

3 Rasmussen, R. F., *Zentralbl. f. Bakt.*, I Abt., Orig., 1938, **143**, 89.

has been initiated in our laboratories, with results which indicate that the infection prevails among them.

There are two methods which may be employed for the detection of ornithotic infections in birds: one involves the inoculation of mice with the suspected avian spleen, liver and kidney tissue emulsions; the other method involves the serological detection of infection with the complement-fixation technic. That the latter technic is accurate is affirmed by the extensive data presented by Bedson,[4] and by Meyer *et al.*[5] Francis and Gordon[6] increased the specificity of the antigen by preparing it from the chorio-allantoic fluid of the infected chick embryo.

Methods. Antigen. A standard strain of ornithosis virus (P-4), obtained from Dr. J. E. Smadel, was propagated in the chorio-allantoic fluid of 7-day-old chick embryos. After 5 days further incubation at 35°C, the extra embryonic fluids were collected from the eggs and this material was then inactivated at 56°C/30 minutes. The agent was sedimented in an angle head centrifuge at 6000 r.p.m./1 hour after which the sediment was resuspended in saline to 1/10 its original volume. It was then titrated with a known potent antiserum and its complement fixing titer was set at 2 units. This antigen was prepared by us while at the 8th Service Command Laboratory, Fort Sam Houston, Texas, and was subsequently preserved in the lyophilized state at refrigerator temperature until used in this problem.

Serum. The blood specimens were collected by heart puncture and after storage overnight in the refrigerator, the serum was separated and inactivated at 56°C/30 minutes. Specimens of serum were collected from the following species of birds: Laughing gull *(Larus atricilla L.)*, Royal tern *(Sterna maxima B.)*, Least tern *(Sterna antillarum L.)*, Common tern *(Sterna hirundo L.)*, Skimmer *(Rynchops nigra L.)*, Willet *(Catotrophorus semipalmatus G.)*, Gull billed tern *(Gelochelidon nilotica L.)*, Glossy ibis *(Plegadis autumnalis L.)*, Reddish egret *(Dichromanassa rejuscens)*, Brown pelican *(Pelecanus occidentalis L.)*, Sooty tern *(Sterna fuscatus)*, and Sanderling *(Calidris leucophaea P.)*.

Test. The inactivated serum was diluted in saline serially from 1:5 to 1:160 in 0.2 cc amounts, 2 units of antigen, and 2 units of complement was added, and this mixture was incubated at 37°C/1 hour. The amboceptor and 2% sheep red blood cells were then added and further incubated at 37°C/30 minutes. At the end of this period the results were recorded, providing the serum, antigen and complement controls were satisfactory.

Results. It may be noted in Table I that 40% or more of the laughing gull, skimmers and willets showed complement-fixing antibodies for ornithosis in their blood serum. There was a lower incidence of antibodies in the blood serums of the terns and sanderlings, although the total numbers of the species were smaller. The majority of serum titers were in 1:5 and 1:10 dilutions; however, some went as high as 1:80 and 1:320 dilution. Of 165 birds of all species examined, 61 (36%) showed evidence of complement-fixing antibodies for ornithosis in the blood serum.

This serological evidence must necessarily be confirmed by actually demonstrating the agent in organs of naturally infected birds. Thus far, a virulent ornithosis-like agent has been isolated in mice from the pooled livers and spleens of 2 willets. This agent (W-ornithosis strain) is lethal for mice by intracerebral and intraperitoneal routes; in either case, nests of elementary bodies can be observed in the mononuclear cellular exudate of the spleen and meninges. The agent kills 7-day-old chick embryos in 4 to 5 days, in which case the chorio-allantoic fluid contains numerous minute elementary bodies. An antigen was prepared from the chorio-allantoic fluid, as described for the P-4 psittacosis antigen above, and it fixed complement with psittacosis serum, lymphogranuloma venereum serum, and its homologous rabbit serum. This latter serum also fixed

4 Bedson, S. P., *Brit. J. Exp. Path.*, 1936, **17**, 109.

5 Meyer, K. F., and Eddie, B., *J. Inf. Dis.*, 1939, **65**, 225.

6 Francis, R. D., and Gordon, F. B., PROC. SOC. EXP. BIOL. AND MED., 1945, **59**, 270.

TABLE 1.
Ornithosis in Seashore Birds by Complement Fixation Test.

Species	No. Tested	No. AC	No. +	% +	Complement fixation serum titers 1:5	1:10	1:20	1:40	1:80	1:320
Laughing gull	60	3	24	40	10	8	1	2	2	1
Willet	37	3	17	45.4	7	6	4			
Skimmer	15	0	8	53	4	2	2			
Sanderling	8	1	4		4					
Royal tern	15	0	1	6			1			
Least tern	8	0	2			1		1		
Common tern	6	0	3		1	11				
Gull billed tern	6	0	1	16		1				
Ring billed gull	1	0	0							
Glossy ibis	1	0	1						1	
Brown pelican	4	1	0							
Sooty tern	1	0	0							
Reddish egret	3	3	0							
	165	11	61	36	26	20	8	3	3	1

complement with lymphogranuloma venereum antigen and with psittacosis antigen.

The significance of finding evidence of ornithosis in sea-shore birds serves to add to the spectrum of information regarding the distribution of this disease in nature. While it may not be a great hazard to the human population, since these sea-shore birds are not generally confined in close association with man, nevertheless, this disease appears to exist in a multitude of avian species. Under proper circumstances these infected birds might contribute directly, or through other birds, to the morbidity rate of human ornithosis.

Summary. Serological evidence of ornithosis-like infections among several species of sea-shore birds is presented. Over 40% of the serums of sea gulls, willets, and skimmers which were examined showed complement-fixing antibodies for ornithosis. One strain of an ornithosis-like agent has been isolated from the willet species.

15746

Specific Complement-Fixing Diagnostic Antigens for Colorado Tick Fever.

CARL J. DE BOER, LAWRENCE J. KUNZ, HILARY KOPROWSKI, AND HERALD R. COX.
From the Section of Viral and Rickettsial Research, Lederle Laboratories Division, American Cyanamid Company, Pearl River, New York.

In the past Colorado tick fever[1,2] was believed to be a mild form of Rocky Mountain spotted fever.[3] However, largely due to the work of Florio and his colleagues,[4,5] it is now known that Colorado tick fever is not a rickettsial infection but a viral disease,[5] presumably tick-borne,[6] which apparently is quite distinct from Rocky Mountain spotted fever[1,3,7] and dengue fever[8,9] although resembling the latter both clinically and hematologically.

1 Becker, F. E., *Colorado Med.*, 1930, **27**, 36, 87.

2 Toomey, N., *Ann. Int. Med.*, 1931-32, **5**, 585.

3 Florio, L., Mugrage, E. R., and Stewart, M. O., *Ann. Int. Med.*, 1946, **25**, 466.

4 Florio, L., Stewart, M. O., and Mugrage, E. R., *J. Exp. Med.*, 1944, **80**, 165.

5 Florio, L., Stewart, M. O., and Mugrage, E. R., *J. Exp. Med.*, 1946, **83**, 1.

6 Topping, N. H., Cullyford, J. S., and Davis, G. E., *Publ. Health Rep.*, 1940, **55**, 2224.

7 Shaffer, F. C., *Colorado Med.*, 1935, **32**, 226.

8 Florio, L. Hammon, W. McD., Laurent, A., and Stewart, M. O., *J. Exp. Med.*, 1946, **83**, 295.