(nurse to case T) succumbed to a streptococcic septicemia. The remaining animal of the series was sacrificed on the 41st day and again subinoculations yielded the psittacosis virus. In the meantime a second specimen of sputum from the same patient had produced typical infections in 4 mice.

These and many similar observations confirm the fact that the psittacosis virus has a remarkable tendency to remain latent in the spleen and liver of birds and mammals. The subclinical disease is largely influenced by the amount and the potency of the virus. Latent infections occur more frequently in heterogeneous transmissions, for example, from birds to mammals, than when passages between mammals of the same species are attempted. From a practical diagnostic point of view it is important to reinoculate the spleens which are enlarged. This procedure renders the search for the psittacosis virus slightly more complicated but improves the accuracy of the mouse test.

Mice with latent infections resist reinoculations with a very potent virus. This resistance is sometimes followed by a sterile immunity. But since the time of autosterilization is uncertain the immunization with formalinized spleen emulsions is generally preferred.

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Latent Psittacosis Infections in Shell Parrakeets.

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The existence of the psittacosis virus in the budgerigar or shell parrakeet (*Melopsittacus undulatus*) bred and raised in California was demonstrated by laboratory tests. Forty-five human cases of psittacosis infection recorded in California have followed exposure of patients to this species of bird. Secondary man to man infections, contact with infected canaries or foreign birds, or indirect contact with clothing worn in an infected aviary have resulted in 9 additional cases. Ten of the 54 infections ended fatally. Before and after the recognition of these facts psittacosis cases attributed to shell parrakeets from California have been observed in Kansas City, Portland, Oregon, New York City, Chicago, Boise, Idaho, Madi-

son, Wisconsin, Massachusetts, Michigan, Minnesota and New Jersey. The details of the epidemiological observations leave no doubt that the budgerigar, either visibly ill or apparently well, may transfer a disease to man indistinguishable from the malady which was attributed to the importation of infected parrots in 1929-1930. Parrot fever, due to indigenous shell parrakeets, has also been noted in England and Germany. It is surprising that the heterogeneous infection chain budgerigar or shell parrakeet—man has not received careful attention since Gordon[1] demonstrated the psittacosis virus in the spleen of a shell parrakeet obtained from the London Zoological Gardens.

Nothing definite concerning the origin or extent of the virus disease in the birds of Southern California is known. Obviously, before planning control measures to protect the public or to save the bird breeding industry from annihilation incident to the various quarantine measures by federal and state organizations it appeared desirable to survey a number of aviaries for the presence of infected birds.

At first, establishments were chosen in which human infections had occurred or had furnished disease-producing birds to the trade. More recently, the population of various aviaries has been sampled by a selection at random of 10% of the birds.

A total of 1,953 shell parrakeets from 66 aviaries has been subjected to detailed post mortem examinations and inoculation tests. Suspensions of the spleens and livers, later only the spleen, the mucous membrane of the nostrils and the filtered content of the cloaca have been injected into mice. When practicable, the spleens of each bird have been examined separately. Experience indicated that, as a rule, spleens measuring less than 4mm. in diameter were non-infectious. Consequently, the spleens of a certain diameter have been pooled and the suspensions in broth have been tested on mice. The presence of the psittacosis virus has been proved by the demonstration of sterile cultures from the enlarged spleen of the birds and mice, the typical anatomical lesions on the liver, the presence of elementary bodies (Levinthal-Cole-Lillie bodies) and by passage experiments of filtrates to mice and occasionally to ricebirds. Approximately 1,850 birds selected according to age in lots of from 6 to 450 originated from different pens of 65 aviaries and pet shops. Over 100 shell parrakeets either dead or sacrificed were collected from 1 aviary in which the epidemic course of avian

[1] Gordon, *Rep. on Pub. Health and Med. Subjects*, London, 1930, **61**, 106.

psittacosis is followed by a daily inspection of the pens. The 1,850 birds were classed by their owners as "healthy" budgerigars. They were chloroformed and the feathers dampened with cresol solution. An external examination showed in a small number of birds a few drops of mucous on the ceres and a soiled anus, more frequently a definite emaciation manifested in a marked atrophy of the pectoral muscles. In the latter class of birds the internal examination revealed a definitely enlarged, heavy and rather tough, slightly saffron or ochre yellow colored liver occasionally with healed necroses and a spleen varying in diameter from 5 to 18 mm. and rarely a few pneumonic patches in one or both lobes of the lungs.

With a few exceptions, the psittacosis virus was readily demonstrated in the organs of birds showing these lesions. However, more common were well nourished shell parrakeets in which merely the size of the spleen attracted attention. In fact, the degree of latent psittacosis infection in a given lot of budgerigars was readily predictable by the percentage of spleens exceeding in diameter the normal average of from 1 to 4 mm. Thus in aviary T a lot of 24 shell parrakeets, in which 14 were found to harbor the virus, and in another, C, a lot of 25 birds with no infection, the sizes of spleens were as follows:

	Aviary T Infected	*Aviary C Non-infected*
Size of the spleen	2 to 4 mm. = 7	24
	5 to 11 mm. = 17	0

Some relationship exists between the age of the bird, the size of the spleen and the latent infection. In a sample of 48 birds from an infected aviary 66% of the old birds had spleens varying in diameter from 1 to 4 mm. and only 13% were larger than 6 mm.; on the other hand, only 50% of the young birds (not older than 5 months) had small spleens while 37% had spleens greater than 6 mm. in diameter. In general, "non-capped" (young) birds are more frequently carriers of the virus than the "capped" or old birds. However, birds 2 to 3 years old may present very large (23x24x9 mm.) spleens resembling a myeloma in which the virus may be demonstrated.

Forty-four or 66% of the 66 aviaries housed budgerigars in which the psittacosis virus was demonstrated in the internal organs. The number of birds found infected in a sample or a pen varied greatly. Occasionally, one sample consisting of old birds revealed no latent infections, while the spleens of a subsequent sample consisting of young parrakeets yielded a high percentage of positive mice. For example, in March only 1 spleen of 6 birds of the

A. R. C. Aviary contained the virus while late in November 186 budgerigars of a sample of 450 presented enlarged spleens and a high incidence of latent infection. A 2% sample of the D. K. Y. Aviary yielded healthy birds in the spring but a 10% sample (31 birds) revealed 4 spleens considered suspicious for psittacosis. The virus was found in 2. The first test on Aviary M. Y. gave one positive bird while the examination of the total budgerigar population of 413 suggested latent psittacosis (22%) in 5 breeding pens. The virus was obtained in the pools of the spleen removed from the birds of every pen except pen 5.

The percentage of virus carrying birds in a sample or in a pen or in an entire aviary varied from 10 to 90%. Incomplete evidence indicates that the virus may be present in the nose or the liver and absent in the slightly enlarged spleen. It is therefore not unlikely that much higher figures would have been obtained if the liver, nose and spleen of every bird had been pooled and tested on mice. The evidence presented probably represents minimum and not maximum percentages. Notwithstanding this reservation it is definitely proved that avian psittacosis infection is a disease of eminently chronic character and as such exists in a high percentage of aviaries. Even breeding places managed under excellent sanitary conditions harbor infected birds. Their existence can only be recognized by a post mortem examination and by mouse inoculations. Judged by the short incubation time (5-6 days), the high fatality rate and the extent of the anatomical lesions produced in mice, a highly active virus in large quantity is usually present.

A study of the epidemiology of the psittacosis disease among parrakeets may answer the question: When are these carriers infectious for man and what factors contribute to human transmissions? It is difficult to correlate the wide and extensive occurrence of psittacosis-virus carrying birds with the relatively low incidence of recognized human infection in population groups which have close contact with birds.

Incomplete observations suggest that the virus leaves the birds much more frequently by way of the nostrils than the cloaca. Furthermore, it may be found in the egg yolk of breeding birds thus suggesting a germinative transmission. The period of autosterilization is not known; it is doubtless of many months duration. Despite the existence of a heavy latent infection rate the spread of the virus is confined to the birds of one pen. Parrakeets housed in an adjacent cage merely separated from the diseased by a wire

netting may be anatomically and by inoculation tests entirely healthy. Whether the latency is the sequel, precursor or an intermediary stage of the disease is now the subject of further analysis since exposure experiments with parrakeets from infected aviaries have so far yielded negative results.

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Toxicity, Distribution, and Excretion of Thallium.

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Thallium sulphate administered by mouth to quail, geese and ducks has been found to be fatally toxic in doses of 12, 15, and 30 mg. per Kg. respectively, calculated as thallium metal. Analyses were made of the distribution of thallium in various body tissues by a gravimetric iodide method, and by a new colorimetric method involving liberation of iodine from potassium iodide by thallium, and the estimation of the color intensity of the iodine in carbon bisulphide. The latter method is accurate to within 5% at a thallium concentration of 15 mg. per Kg. Thallium in liver, kidney, heart, and osseous tissue was estimated to be present in a concentration approximately equal to that which had been administered. Muscular tissue, however, was found to acquire a considerable higher concentration of thallium. Fat was found to contain practically none. Analysis of the tissues of a goose dying 15 days after the oral administration of 20 mg. per Kg. indicated a retention of 35-70% of the thallium given.

The first studies on the excretion of thallium were reported in 1890 by James Blake,[1] a physician residing in Middletown, California, who found by spectroscopic examination that thallium was eliminated in all secretions. Quantitative estimations of the rate of elimination of thallium in the urine after oral administration to dogs, by the colorimetric method noted above, indicated that 60% of the total amount given was excreted in 36 days, with a progressive diminishing rate of excretion.

Although thallium is very slowly excreted, and although the

[1] Blake, J., *Compt. Rend. Soc. Biol.*, 1890, **1**, 55.