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RÉSUMÉ

La griséofulvine qui a déjà défrayé plusieurs communications dans ce journal forme encore ici le sujet d'un rapport clinique de sept cas. Ces malades atteints de la teigne et autres infections fongiques de la peau et des ongles ont tous (sauf un) été améliorés par l'administration orale de ce nouveau médicament. Le *Tricophyton rubrum* était en cause dans plusieurs cas. Le seul échec dans cette série s'est vu chez un malade qui a accusé une légère amélioration au début du traitement. La maladie dont les progrès furent arrêtés temporairement sembla devenir réfractaire par la suite.

THE PREVALENCE OF COMPLEMENT-FIXING ANTIBODIES AGAINST PSITTACOSIS IN THE CANADIAN ARCTIC*

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ALTHOUGH it is now well recognized that the geographical distribution of ornithoses in birds is very wide, extending to such high latitudes as the Falkland Islands[1] and the Orkney and Faroe Islands,[2] and that human infections are not uncommon in employees of poultry farms,[3] it was still somewhat surprising to find antibodies in approximately 15% of apparently healthy adult Eskimos on the East Baffin Island coast.[4] This report deals with the prevalence of these antibodies in subsequent surveys of other northern populations.

POPULATIONS SAMPLED

Blood samples have been collected from populations shown in the map and listed in Table I. The

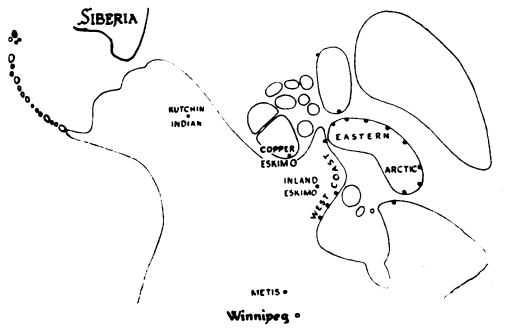

Fig. 1

Eastern Arctic studies previously reported are also included; the even distribution of persons with antibodies was shown in the original report.[4] The proportion of the total population sampled in this group was approximately 15%; all were adults. Similarly, it may be seen from Table I that the blood specimens collected from Eskimo Point, Rankin Inlet, Chesterfield and Igloolik on the west coast of Hudson Bay as well as those collected from Cambridge Bay were small samples of the adult Eskimo population. The blood specimens from Old Crow, however, were from a community of Kutchin Indians where blood was collected from most of the population over the age of four.[5] A small group of Cree Indians and a group of part-Indians from Southern Manitoba were also sampled. The part-Indians were Metis actively engaged in hunting, trapping and fishing at the south end of Lake Manitoba. The Indians were patients in the Brandon Sanatorium. Specimens shown in the table from Winnipeg and rural Manitoba were from patients with a suspected virus pneumonitis and were referred to the Manitoba Virus Laboratory for virological tests;

TABLE I.—DISTRIBUTION OF PSITTACOSIS ANTIBODIES

Location	*Race*	*Estimated population*	*No. tested*	*No. with psittacosis antibodies*
Baffin Island	Eastern Eskimo	1670	241	39
Cambridge Bay	Copper Eskimo	1400	54	44
West Coast Hudson Bay	Eastern Eskimo	1400	88	28
Garry Lake	Inland Eskimo	50	21	3
Old Crow, Yukon Territory	Kutchin Indians	170	108	75
St. Ambroise Lake Manitoba	Metis	300	20	0
Brandon San.	Eastern Eskimo		6	1
	Cree Indian		6	0
Winnipeg General Hosp.	European	urban and rural patients with suspected respiratory virus disease	1792	19*

*Including two cases diagnosed by rising titres in patients, one of whom was a poultry farmer and the other a pet-shop operator, and four household contacts of these two cases.[6]

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TABLE II.—DISTRIBUTION OF PSITTACOSIS ANTIBODY TITRES BY AGE GROUPS

Age	*<10 years*						*11 - 20 years*						*21 - 40 years*						*41 - 60 years*						*60 years*						*Total including those of unknown age*					
Titres	<4	4	8	16	32	64	<4	4	8	16	32	64	<4	4	8	16	32	64	<4	4	8	16	32	64	<4	4	8	16	32	64	<4	4	8	16	32	64
Cambridge Bay Copper Eskimos (54 sera)											1		4	2	5	8	6	3	2	2	5	2	2								10	7	11	13	10	3
West Coast, Hudson Bay Eskimos (88 sera)	5	2					14	5					24	6	1	2		1	10	5	2	1			2						60	19	3	5		1
Old Crow Kutchin Indians (108 sera)	5	10	4	2	1		8	6	4	4		1	7	7	4	10	4	2	5	4	2	3			8	1	3	1			33	29	15	22	6	3

some of these patients were suspected of having psittacosis clinically and in fact two cases of psittacosis were confirmed in the laboratory. These will be reported elsewhere in detail.[6]

METHODS

Blood samples were collected and the sera separated under sterile conditions. They were then transported and stored in the frozen state until tested.

A standard tube technique was employed for the complement fixation test using 2 units of psittacosis-human pneumonitis antigen* and 2 units of guinea pig complement. Overnight refrigeration at 4° C. was followed by a warm-up period of five minutes at 37° C. in a water bath before addition of 4 units of amboceptor with 2% sheep cells. Incubation at 37° C. was then continued for a further 20 minutes. The total volume of the test was 0.6 ml.

RESULTS

Table I demonstrates the prevalence of these antibodies in both Indian and Eskimo communities in the Western Arctic, the lower incidence in the Eastern Arctic and Inland Eskimos and the still lower incidence in both rural and urban white Manitobans.

The distribution of antibodies by age groups and titres is shown in Table II for the communities in which antibodies were detected in some numbers. It may be observed that antibodies occur in all age groups.

DISCUSSION

As the relative lack of specificity of this test is well known, consideration was first given to those diseases which are associated with antibodies that may cross with the psittacosis-human pneumonitis antigen.[7-11] Cat scratch fever and lymphogranuloma venereum were excluded for clinical reasons; syphilis was ruled out by the absence of clinical stigmata of the disease and also by the uniformly negative serological tests for syphilis. Eskimos and Indians frequently have corneal opacities,[12-15] and the possibility of trachoma was also considered; this disease has not been reported as a factor in the etiology of corneal scarring in the Arctic, and a recent eye survey of the Central Eskimos with this possibility in mind failed to show any evidence of trachoma.[16] This survey was carried out on the west coast of Hudson Bay, an area having a moderate incidence of psittacosis antibodies (Table I). At Old Crow a large proportion of the population had antibodies but corneal scarring was seen in only a few cases. In this community no patients were seen with acute conjunctivitis, nor was there any historical evidence of its occurrence;[5] this would tend to exclude inclusion conjunctivitis as the etiology of these antibodies.

The possibility of a genetically determined protein acting either as the antibody or as an anti-complementary agent has been considered. Although this explanation cannot be entirely ruled out, it seems unlikely: the presence of antibodies and their concentration cannot be correlated with racial purity in the two groups with the highest incidence of antibodies, Old Crow and Cambridge Bay.[17] Although each of the groups sampled may be genetically distinct, the fact that antibodies occur with high frequency in one Indian community and one Eskimo community but at a moderate or low incidence in other Indian and Eskimo communities does not support this genetic hypothesis. In addition, tests carried out by Ross on 16 of the sera from the Old Crow Indians using a group specific meningo-pneumonitis antigen for both complement-fixing[18] and hæmagglutinin-inhibiting antibodies yielded results similar to our own.[19]

The sera were also tested for antibodies to other viruses such as influenza, mumps, adenovirus, herpes simplex and the enteroviruses, and antibodies were found to occur in these populations;[20, 21] there is no correlation between the incidence of these antibodies and the incidence of the psittacosis antibodies. It has been reported that the antibodies of brucellosis and tularæmia react with the psittacosis virus.[10] The sera were also tested against brucella and tularæmia antigens and no correlation was demonstrated.

Although further work is required to elucidate the reservoir and mode of transmission of the agent producing these antibodies, it is probable

*Markham Laboratories, 9246 South Vincennes Avenue, Chicago 20, Illinois.

that they result from exposure to ornithosis or some related virus harboured by birds or animals and producing a relatively mild disease in humans. The possibility also exists that the agent is directly transmitted from man to man.[22, 23]

SUMMARY

Tests for complement-fixing antibodies to a psittacosis–human-pneumonitis antigen in Canadian Arctic Eskimo and Indian communities showed a high incidence of antibodies ranging from 15 to 80% in different communities.

It is considered that these antibodies result from endemic exposure to a virus of the ornithosis group; reservoir and mode of transmission are not clear.

We are grateful to Drs. Bruce Chown, A. C. Wallace and F. J. Sellers for blood samples collected at Cambridge Bay, Garry Lake and west coast of Hudson Bay respectively; to Dr. M. R. Ross for his assistance in testing some sera with his own antigens; to Dr. K. F. Meyer for helpful comments; and to Dr. L. P. Lansdown for the serological tests for syphilis, tularæmia and brucellosis.

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RÉSUMÉ

Il est étonnant de se rendre compte qu'environ 15% des Esquimaux adultes et apparemment en santé possèdent des anticorps à l'ornithose. L'épreuve n'est pas des plus spécifiques mais les autres infections qui pourraient produire des anticorps comparables furent éliminées par l'examen clinique ou les épreuves sérologiques. Le réservoir et le passage restent encore obscurs.

Case Reports

INTOXICATION BY ETHCHLORVYNOL (PLACIDYL) REPORT OF FOUR CASES

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ANY DRUG which is taken for the relief of suffering or tension is potentially habit-forming. Whether the drug is called a hypnotic, a sedative or a tranquillizer and whether it is a barbiturate or a non-barbiturate does not matter; only if the drug has some unpleasant side effects is the danger of addiction minimized (as in the case of chlorpromazine).

One of the new drugs until recently sold without prescription is ethchlorvynol (Placidyl).† The manufacturers state: "Placidyl is especially recommended for hypnotic effect in simple insomnia due to tension, mild anxiety, mild excitement or agitation. It is also useful for daytime tranquillization."[1] Many persons are probably taking reasonable quantities of this drug, but the following case histories indicate the danger of excessive intake. The first three patients did not consult a physician but obtained ethchlorvynol at the local drug store.

CASE 1.—This 44-year-old married woman, with no children, was a housewife. The history of ethchlorvynol intake is indefinite, no reliable informants being available. It is known that the capsules had been used "in large numbers" since August 1958, because of insomnia. She was reported to have been drowsy at times, had been noted to be staggering, and may have had "blackouts". The patient had no history of taking other medication except occasionally aspirin with codeine tablets.

She was admitted to the Verdun Protestant Hospital on October 10, 1958. At that time she was noted to be somewhat confused and disoriented; she was lethargic and apathetic, and walked with an unsteady gait. No other neurological abnormalities were noted. The symptoms cleared rapidly within the first few days. On November 22, she was allowed to go home for a week-end; when she returned the following day she was staggering, showed slurring of speech, had a number of minor injuries, and could not explain what had happened. Several days later it was discovered that she had been taking ethchlorvynol capsules during the week-end. The patient was removed from the hospital against medical advice on November 29, but had to be readmitted on December 11, because on three occasions she had been stuporous, apparently because she had been taking ethchlorvynol again.

CASE 2 (courtesy Dr. H. B. Durost).—This man, aged 45, married with two children, was a manu-

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†This drug, together with many other tranquillizers, was placed on Schedule F in July of this year.