

An outbreak of common colds at an Antarctic base after seventeen weeks of complete isolation

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SUMMARY

Six of 12 men wintering at an isolated Antarctic base sequentially developed symptoms and signs of a common cold after 17 weeks of complete isolation. Examination of specimens taken from the men in relation to the outbreak has not revealed a causative agent.

INTRODUCTION

It has commonly been believed that on small Antarctic bases, isolated for many months, upper respiratory infections die out during the first few weeks of isolation and that the men are virtually symptom-free for the rest of the isolation period. With the arrival of the relief ship or aircraft, outbreaks of respiratory disease have been noted to occur (Taylor, 1960; Siple, 1960; Hedblom, 1961; Cameron & Moore, 1968; Holmes, Allen, Bradburne & Stott, 1971). This has tended to follow the pattern seen in other isolated communities (Paul & Freese, 1933; Shibli, Gooch, Lewis & Tyrrell, 1971).

Several studies of upper respiratory disease in men at isolated Antarctic stations have been undertaken. Sera obtained from the McMurdo Sound wintering party of 1958 were tested for the presence of antibodies to a number of respiratory viruses, but not including rhinoviruses, and showed no evidence of infection with any of the viral antigens tested (Chanock, R. M., quoted by Cameron & Moore, 1968). A systematic study of monthly serum specimens collected from the members of the South African National Antarctic Expeditions in 1961-62 showed no evidence of new virus infection (J. H. S. Gear, quoted by Cameron & Moore, 1968). In their 1968 study of the epidemiology of respiratory infections at Mawson, an Australian Antarctic Research Expedition station, Cameron & Moore (1968) made observations on infective diseases during the period of isolation, and found no diagnostic rises in antibody titre against influenza viruses A and B, mumps, adenovirus, herpes simplex and ornithosis. All attempts at virus isolation from throat, nose and faeces swabs were unsuccessful.

The apparent absence of respiratory infections for long periods during isolation in Antarctica has provided opportunity for basic epidemiological study, and

experimental inoculation of volunteers using easily traced viruses was started in 1968 with interesting results (Holmes *et al.* 1971). Further study along these lines was contemplated for the winter of 1969 at Adelaide Island Base, one of the British Antarctic Survey stations. During the preliminary observation period, after 17 weeks of isolation, upper respiratory symptoms occurred in one man and then spread to half the Base complement over the next week in the manner of an infectious disease. No virus had been artificially introduced at this time, and the outbreak was unexpected. Clinical records were kept, samples of serum and nasal washings were taken for analysis in the United Kingdom, and possible environmental factors were noted.

MATERIALS AND METHODS

Environment

The British Antarctic Survey Base on Adelaide Island (Latitude 67° S, Longitude 68° W) is situated on the south-west coast of the Antarctic Peninsula, and is about 1000 miles south of the Falkland Islands. The base acts as a centre for aircraft activities during the summer months, supporting remote bases and field parties working further south. Teams of husky dogs are sent to join the field parties, and set out soon after midwinter, with one man driving each team. Meteorologists make round-the-clock observations throughout the year.

The base is isolated from late March to mid-December each year, which coincides with the departure and arrival of the two Survey aircraft. There are one or two visits each summer from a relief ship.

The living quarters are dry wooden huts in the form of a small complex of buildings, heated by solid fuel burners, but the sleeping hut, made of fibreglass, tends to be damp and is heated with small electric convector heaters taking power from a diesel generator, and supplemented by paraffin stoves in the colder months. The sleeping hut is divided into cubicles and during the isolation period two men occupy each cubicle, with bunks 4 feet apart. The living rooms are more spacious, but because of the recurrent necessity for staying indoors because of inclement weather conditions, the men live in very close contact with each other.

Food comes mainly from packets or tins, and there are no fresh vegetables or fruit, but vitamin tablets are taken regularly. There is a daily bread bake, and special occasions merit the thawing of a small amount of meat from a -20° C. freezer or from an ice cave in the nearby glacier. The water supply is from snow or ice blocks melted in stainless steel tanks. Waste from the sink drains under the living huts where it freezes, and is washed out with hoses during the summer melt. Ashes and sweepings are deposited in the sea in summer, and in the crack between sea-ice and land-ice during the winter, as are the contents of the chemical closets.

Weather conditions are variable, and changes often very rapid. Outside temperatures vary from a maximum just above zero centigrade, to a minimum of -35° C. These temperatures relate to still air, and their cooling effect is much enhanced by increasing wind speeds. The average wind speed around the base area is about 14 knots. Frequent gales occur, often reaching wind speeds of 100 knots. Relative humidity outside during the year ranges from about 60 to 90 %.

At this latitude there is continual daylight during the summer months, and in winter months very little daylight or none at all. There are few animals present during the isolation period other than the husky dogs. The local Adelie penguins and most of the other bird life migrate north at the end of the summer season, leaving a small number of petrels and the occasional seal.

Logistics

In 1969, 14 men wintered at Adelaide Island Base. They arrived by sea and air at various times between December 1968 and March 1969, and relieved the previous complement of men. The Base members were all between the ages of 21 and 35 years, and were in good health. One of the men (I. W.) had spent the preceding winter at Adelaide Island Base, and two men had wintered on other Antarctic bases and transferred to Adelaide Island Base (M. B., B. T.).

The last aircraft left on 18 March and flew north for the winter, after which the Base was completely isolated from the outside world, except by radio.

Between 30 June and the time of the arrival of the first aircraft in early December, five husky dogs were present in the Base area. They were used for local transport and small expeditions into the field, and were fed on alternate days with seal meat from a pile of dead seals in the Base area.

Clinical data and specimen collection

The medical history of each Base member was noted throughout the period of isolation, and when symptoms and signs appeared they were recorded on daily observation charts as used at the Common Cold Research Unit at Salisbury, Wiltshire.

Serum and nasal samples were taken monthly from the men throughout the 8-month period of isolation, except during the outbreak of respiratory disease, when nasal secretions were taken on alternate days.

Serum from clotted blood specimens was stored in 2 ml. vials at -20°C . in an electric freezer. Nasal secretions were collected by running 5 ml. sterile saline into each nostril and collecting the sample into sterilized disposable petri dishes. Samples were transferred from the petri dishes into duplicate vials containing 50% nutrient broth and Ampicillin 0.1 mg., and stored at -20°C .

At the end of the isolation period, all samples were taken aboard the relief ship and stored for 10 weeks at 4°C ., after which they were stored at -20 or -70°C . at the Common Cold Research Unit at Salisbury.

Serological tests

Sera taken 3 weeks after the outbreak were examined at the Public Health Laboratory, Salisbury, for CF antibodies against common respiratory pathogens. Those sera showing high antibody titres were compared in further tests with sera taken 2 weeks before the outbreak.

Similar pairs of sera were examined using microtitre methods for CF antibodies against coronavirus OC43, 229E and MHV3, and for HI antibodies against

coronavirus OC43, coxsackievirus A21 and influenza viruses A2/Eng/12/64 and A2/Eng/344/68.

Sera for coronavirus CF and HI tests and coxsackievirus HI tests were inactivated at 56° C. for 30 min. Sera for influenza-virus HI tests were diluted 1/5 with cholera filtrate, left overnight at 37° C. and inactivated at 56° C. for 30 min. Sera taken 2 weeks before and 3 weeks after the outbreak were examined in the Microbiology Department, Northwick Park Hospital, for antistreptolysin-O antibodies and anti-DNase B antibodies.

Tissue and organ cultures

Virus isolation was attempted, from single samples and from pools of these samples, from the nasal secretions of those men who showed symptoms during the outbreak. A range of tissue cultures was used, grown in roller bottles at 37° C. and seeded onto glass tubes to produce a monolayer. Tubes were rolled at 33° C. after inoculation of 0.2 ml nasal secretion.

WI38 cells, MRC5 cells, L132 cells and human embryo kidney cells were maintained in Eagle's basal medium, containing 0.088% sodium bicarbonate, 2% fetal calf serum, and penicillin and streptomycin (100 i.u./ml.). Extra magnesium (0.03 M) was used in the maintenance medium for HeLa cells, and Eagle's medium containing 1% fetal calf serum, penicillin and streptomycin (100 i.u./ml.) and buffered with HEPES was used to maintain African green monkey kidney cells.

Organ cultures of human embryo nasal mucosa were prepared and maintained according to the method of Hoorn & Tyrrell (1969).

Broth cultures

Nasal washings from those men who showed symptoms were inoculated into Todd-Hewitt broth, and incubated at 37° C. for 7 days, to detect the presence of streptococci. The tubes were visually inspected every day, and were subcultured on the seventh day.

Volunteer experiments

These were carried out at the Common Cold Research Unit, Salisbury, Wiltshire, and the methods of study used there have been described previously (Andrewes, 1951). A pool of nasal secretions from men affected in the outbreak was inoculated into ten volunteers, and then a pool of nasal secretions from those volunteers was inoculated into a further seven volunteers. Sera were taken before and 2 weeks after inoculation.

RESULTS

Clinical observations

Between February and March 1969, new personnel arrived at Adelaide Island Base by air and ship from other parts of the Antarctic, and colds continued to occur in both old and new personnel.

The last aircraft left on 18 March and total isolation ensued. During the next 17 weeks no colds were observed by the 14 men at the Base who had been asked

to report any symptoms, and no respiratory disease was detected clinically. Two men left the Base with their dog teams at the end of June for 6 months, during which time they suffered from no respiratory symptoms.

Twelve men were left at the Base, and on 14 July 1969 one man presented with respiratory symptoms closely resembling those of a mild to moderately severe cold as described by Tyrrell (1965). During the next 2 weeks, eight out of the 12 men at the Base suffered similar respiratory symptoms, and a further two had attacks of sneezing. The symptoms and signs were charted (Fig. 1).

Quantitative assessment of nasal discharge by counting paper handkerchiefs used as suggested by Roden (1958) was largely impracticable, but was possible in one man and demonstrated a considerable increase in nasal discharge. In two men discharge was severe enough to warrant the use of two cotton handkerchiefs on the same day.

Of those affected, the average duration of symptoms was 5 days. The symptoms and signs of the outbreak did not resemble those of a streptococcal sore throat, as outlined by Christie (1969). Two men had unilateral posterior cervical adenitis of a mild nature.

Of the four men who showed minimal or no symptoms, two (H. B. and D. H.) worked in a separate hut during the daytime and shared the same sleeping cubicle, and one (J. N.) tended to work at night and sleep during the day, thus reducing contact with other Base members.

The man who first showed symptoms (M. B.) had spent the previous year at a four-man station at Fossil Bluff, further south, during which time no respiratory symptoms were noticed, but he had developed a cold in the few days after being transferred by air to Adelaide Island Base during the relief period.

The symptoms in one man (D. S.) may have been related to over-exertion in the cold air. He returned from a field trip 3 days after the start of the outbreak, and 2 days later complained of a stuffy nose and slightly increased nasal discharge lasting for 3 days. One day after this he developed a headache and a wheezy chest, with inspiratory and expiratory rhonchi in both lung fields. He recovered from the chest trouble in 1 week without the use of antibiotics and had no further chest complaints during the remainder of his stay. He gave no previous history of asthma or chest trouble.

Environmental observations

The outbreak occurred in the second half of July 1969, just over 3 weeks after midwinter, and was preceded by 2 weeks of bad weather conditions. In the first half of the month there were 5 days of gales, 12 days of blowing and drifting snow, and 7 days of snowfall. During this time most people stayed in the safety of the central hut, and social contact was thus greatest in the 2 weeks before the outbreak of respiratory disease.

There was minimal daylight for the first half of the month and there was no sunshine until 24 July, 10 days after the appearance of the first symptoms. With an average daily temperature of -13.4°C . the first half of the month was a little warmer than the last half, which gave an average daily temperature of -17.5°C .

Date	14	15	16	17	18	19	20	21	22	23	24	25	26	27	
MB	<u>Headache</u> <u>Nasal stuffiness</u> <u>Nasal discharge</u> <u>Sore throat</u> <u>Post-nasal discharge</u> <u>Sneezing</u>														
TA	<u>Headache</u> <u>Nasal stuffiness</u> <u>Nasal discharge</u> <u>Sore throat</u> <u>Sneezing</u> <u>Post-nasal discharge</u>														
DB	<u>Nasal discharge</u> <u>Nasal obstruction</u> <u>Sneezing</u> <u>Nasal stuffiness</u>														
SW	<u>Nasal discharge</u> <u>Nasal stuffiness</u> <u>Post-nasal discharge</u> <u>Sneezing</u> <u>Headache</u> <u>Sore throat</u> <u>Cough</u>														
BW	<u>Nasal stuffiness</u> <u>Post-nasal discharge</u> <u>Hoarseness</u> <u>Malaise</u> <u>Chill</u> <u>Pyrexia</u> <u>Sneezing</u>														
IW	<u>Nasal discharge</u> <u>Nasal stuffiness</u>														
DS	<u>Nasal discharge</u> <u>Nasal stuffiness</u> <u>Wheezy chest</u> <u>Headache</u>														
BD	<u>Sneezing</u> <u>Headache</u> <u>Malaise</u> <u>Nasal stuffiness</u>														
JN	<u>Sneezing</u>														
HB	<u>Sneezing</u>														
BT	} No symptoms														
DH															

Fig. 1. Symptoms in 12 men in June 1969 after 17 weeks of isolation. The line under each symptom denotes the length of time that symptom was present.

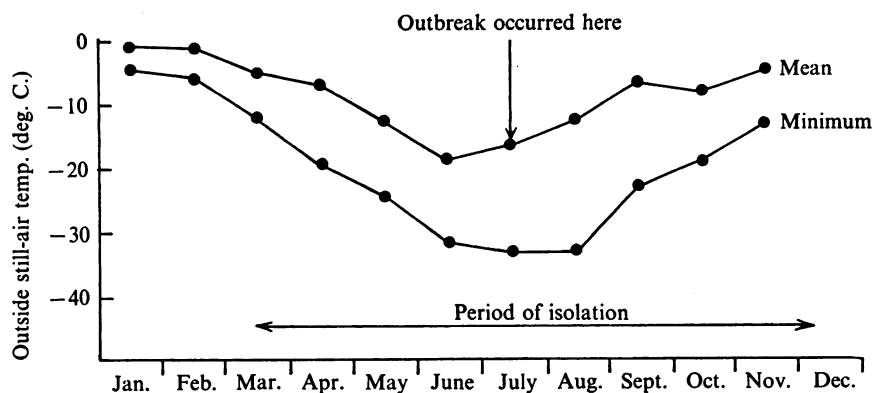


Fig. 2. Occurrence of outbreak in relation to mean and minimum monthly temperatures during 1969.

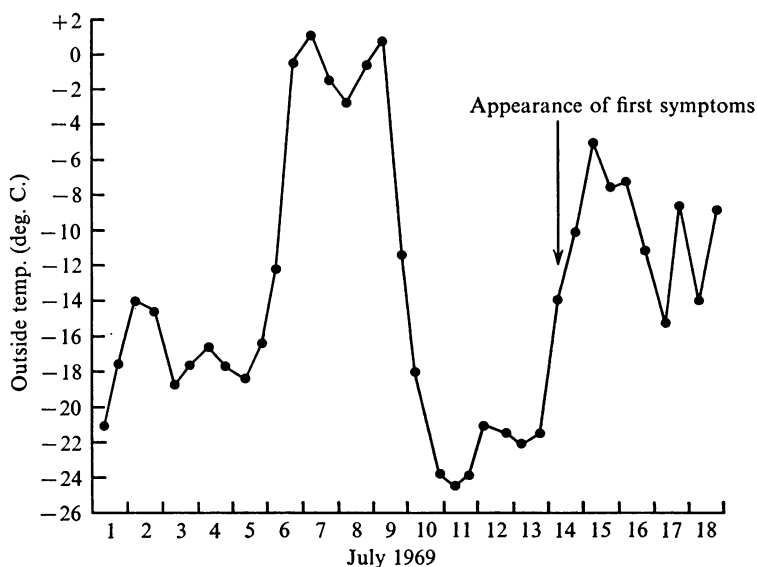


Fig. 3. Occurrence of outbreak in relation to bidaily temperatures during July 1969.

The timing of the outbreak in relation to the mean and minimum monthly temperatures is shown in Fig. 2, and in relation to outside temperatures taken at 6 a.m. and 6 p.m. during the first 18 days of July is shown in Fig. 3. A sudden change in weather between 9 and 10 July brought the outside temperature down from a high of 1.2°C . to a low of -24°C ., and reduced the relative humidity outside from 93% to 79% over the same period.

Laboratory observations

Organ culture. In two experiments, in which pools of nasal washings were inoculated into cultures of human embryo nasal mucosa, there was no loss of ciliary activity compared with controls.

Tissue culture. Nasal washings from two men, who had been affected in the outbreak, produced cytopathic effects in WI38 cells, and these effects were reproduced in the next passage into WI38 cells. Although the effects resembled those produced by some coronaviruses, they could not be positively distinguished from non-specific cytopathic effects. Further passages into WI38, L132 and African green monkey kidney tissue cultures produced no cytopathic effects.

Harvests from the organ cultures produced no cytopathic effects in human embryo kidney cells, but passage from these into WI38 cells produced cytopathic effects again resembling those seen previously, which were not present in passed controls. Further passages did not produce any convincing cytopathic effects.

Electron microscope studies

Tissue culture cells from those tubes which showed cytopathic effects were examined with the electron microscope. No virus was seen.

Volunteer experiments

One doubtful cold was produced in one out of ten volunteers given pooled nasal secretions from the outbreak. Inoculation of a pool of nasal secretions from these volunteers into a further seven volunteers produced no symptoms.

Sera taken before and after inoculation from all volunteers were tested for coronavirus OC43 HI antibody, in view of the cytopathic effects seen in tissue culture. No rises in titre were demonstrated.

Broth cultures

Nasal washings taken from men with symptoms during the outbreak yielded no pathogenic bacteria.

Survey of CF antibodies against a range of respiratory pathogens

Sera from all the men at the Base taken 3 weeks after the start of the outbreak showed low or undetectable CF antibody titres against influenza B virus, respiratory syncytial virus, adenovirus, *Mycoplasma pneumoniae*, psittacosis, Q fever and coxsackie virus A.

Most of the sera had CF antibody titres of 1/20 against influenza A virus, but comparison with sera taken before the outbreak showed no rises in antibody titre. The sera of two men taken three weeks after the outbreak showed CF antibody titres of 1/100 and 1/50 against coxsackie virus B, but further comparison of similar pairs of sera showed no significant rises in CF antibody titre.

Further tests against specific viral and bacterial antigens

No HI antibody rises between paired sera, against the 1964 and 1968 strains of influenza virus A2, or against coronavirus OC43 were detected. The results of the CF antibody tests for coronavirus were obscured by anti-complementary activity and irreproducibility, and were thus unreliable. Further CF tests using sera treated with complement, and also sera treated with 1% chloroform gave unsatisfactory results. No significant rises in anti-streptolysin-O antibodies were detected. AntiDNase B titres were all low or absent.

DISCUSSION

There is little doubt that an outbreak of respiratory disease occurred at an Antarctic base after 17 weeks of complete isolation. The symptoms occurring in six of 12 men were totally unexpected, and are most likely to have been of infective or allergic origin. The close resemblance of the symptoms to those of a common cold, the absence of common plant allergens, and the low level of dust in the huts, make it unlikely that the symptoms were of allergic origin. Finally, the sequential nature of the outbreak, suggesting person to person spread, indicates the presence of an infective agent.

A virus is most likely to have been the cause of this outbreak. However, in about 10% of minor respiratory disease a β -haemolytic streptococcus may be discovered (Tyrrell, 1965). Our clinical and laboratory results indicate that streptococci were not involved. The range of viral agents responsible for the common cold syndrome is large, and the symptoms in each syndrome tend to overlap, and thus there are no sharp distinctions. Also the colds produced in Antarctica are not necessarily similar to those produced by the same viral agent under non-isolated conditions, so that the identification of a causative agent, on purely clinical grounds, is impossible. Viral antibody studies were limited to the range of respiratory antigens available, and HI and CF tests showed no evidence of infection with any of the viruses used. However, no studies of antibody against rhinovirus could be attempted, because of the multiplicity of serotypes, yet these viruses are thought to produce up to 50–60% of upper respiratory infections in adults (Rhodes & van Rooyen, 1968), and could have been responsible for this outbreak.

Attempts at isolation of a viral agent using tissue and organ culture techniques were unfruitful, as was electron microscopy. The cytopathic effects seen in several passages of nasal washings in WI38 cells, although they appeared non-specific in character, could equally well have been the effects of a coronavirus, but such a possibility was not confirmed by serological tests on specimens taken during the outbreak and volunteer trials. Inoculation of volunteers with nasal washings taken during the outbreak produced negative results, and might indicate that if virus were present it was no longer viable, reflecting previous experience with such specimens (Cameron & Moore, 1968).

The occurrence of a common cold during isolation, when the chances of introduction of new infection from the outside are virtually nil, implies that in some way virus persisted, either in the environment or in the men. The possibility that husky dogs act as an animal reservoir of human respiratory virus was suggested by Holmes *et al.* (1971), but subsequent studies in Antarctica have failed to support this hypothesis (Allen & Holmes, in preparation). In addition, no common human respiratory virus is known to have been transmitted from an animal host to man with the production of disease and it seems unlikely that animals were involved in causing this outbreak.

Persistence of respiratory virus on inanimate objects must be considered. At midwinter many new boxes were opened, and suits were worn which contained

soiled but usable handkerchiefs. Colds have occurred at this time among other men isolated in Antarctica (Allen, in preparation), but in contrast to the present outbreak the colds did not apparently spread. The interval of 3 weeks between midwinter and the start of the outbreak makes it unlikely that infection occurred at midwinter, especially as the two men who left the Base soon after the celebrations had finished showed no evidence of respiratory disease.

Virus might have persisted in the respiratory tract of one or more men at the Base. If such were the case it would be necessary to postulate a triggering mechanism to precipitate symptoms, and it is interesting to note that symptoms occurred 4 days after a precipitous fall in outside temperature, and during one of the coldest months of the year, which Hope-Simpson (1958) and Lidwell, Morgan & Williams (1965) have correlated with increased incidence of respiratory disease. There is disagreement about whether the viruses which cause common colds can be carried by adults, and how important this is in epidemiology. The pattern of virus infection revealed by long-term studies, such as the virus watch programme (Elveback *et al.* 1966), is of a series of short infections with different viruses, and in the case of influenza virus disappearance of the current strain when a new serotype appears. On the other hand, adenoviruses may be shed by children for periods of months and recovered from the tonsils in a high proportion of cases, without evidence of acute respiratory infection, and non-respiratory viruses such as those of the herpes group often persist for the lifetime of a man. Furthermore, observations in animals have shown that pigs can carry swine influenza and transmit infection to other pigs 3 months later (Blaškovič *et al.* 1970), turkeys may carry and shed influenza virus A after apparent recovery (Robinson, Easterday & Tumova, 1972), and cattle which have recovered from foot and mouth disease still reproduce virus in the pharynx, and can initiate epidemics on contact with non-immune cattle (Graves *et al.* 1971).

There are thus precedents in both children and animals for persistence of respiratory viruses, but in adults the laboratory evidence for carriage and reactivation of common cold viruses is weak. It may be that such evidence can only be found in rather unusual conditions of isolation and stress, such as occur in Antarctica. It is likely to be a rare phenomenon, but it might well be important in explaining the persistence of the large number of rhinovirus serotypes which make an appearance in many areas when the temperature falls.

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