

1 (HSV-1). Two of these isolates were made from the same individual, (February and August isolates), who reported no RTI symptoms during the winter period, but developed cold sores on both occasions 1 week following specimen collection and virus isolation. Two HSV-1 isolates were obtained from personnel with respiratory symptoms, one from a subject with a sore throat (February isolate) and one isolate from a subject with short-duration (i.e., less than 24 hours) "flu-like" symptoms in May. These symptoms were thought to be due to the inhalation of fumes from a blow-torch and not of viral origin. All subjects from whom the virus was isolated reported histories of recurrent oral herpetic lesions. Four additional subjects experienced cold sores, but not RTI symptoms, at intervals during the winter isolation period. No virus was recovered from throat swabs of these individuals, and the lesions were not cultured. No other cytopathic or hemadsorbing viruses were recovered from specimens collected during the 1983 winter. Of the 19 participants, 12 (63 percent) had detectable neutralizing antibody to HSV-1. All individuals possessed neutralizing antibody to parainfluenza virus types 2 and 3. The individual who reported the RTI in April had a four-fold rise in antibody to parainfluenza virus type 2, suggesting a recent infection. However, without virus isolation, the specificity of this response cannot be confirmed.

Although upper respiratory infections again occurred during the winter of 1983 at South Pole Station, they appeared to be limited to a few individuals and with little evidence of horizontal transmission. No viral agents could be identified conclusively as the etiologic agents of these illnesses. HSV-1 has

been shown to be a persistent infection of man, causing recurrent oral herpetic lesions, and although this virus has also been implicated as a cause of RTI's in young adults (Glezen, Fernald, and Lohr 1975; Lindgren, Douglas, and Couch 1968), HSV-1 did not appear to be clearly related to the RTI's observed during the 1983 winter at South Pole.

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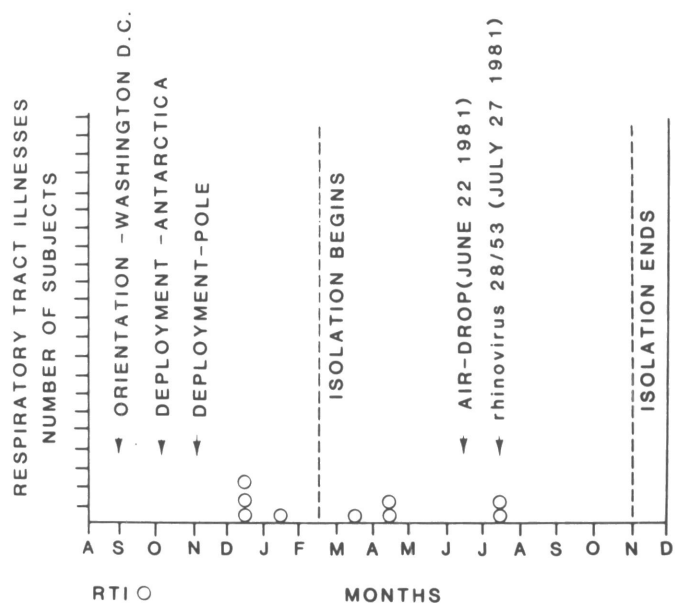
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Rhinovirus respiratory tract infections during isolation at South Pole Station

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Episodes of respiratory tract infections (RTI) occurred among the 17 personnel wintering at South Pole Station during 1981 (figure). One subject (subject 13) reported an RTI 10 March 1981, 4 weeks after station closing and the beginning of the winter isolation period. A second subject (subject 1) reported two separate RTI's, one beginning 6 April and a second beginning 27 April 1981. Another episode of RTI's began 23 July, 4 weeks after the 1981 midwinter air-drop, and involved two subjects (subjects 3 and 5). Symptoms varied from mild—including sore



Numbers of respiratory tract infections (RTI) reported during 1980-1981 at South Pole Station.

throat and nasal congestion lasting less than 2 days (subjects 3 and 13)—to moderate—including initial sore throat, nasal congestion, coryza, and malaise lasting from 4 to 6 days (subjects 1 and 5).

Throat swabs were collected from only the few subjects with symptoms. These swabs were frozen in a virus transport medium consisting of Hanks balanced salts solution with 0.5 percent gelatin added and were stored at -70°C for the duration of the winter. Serum specimens were collected monthly from all volunteer subjects. These were also stored at -70°C throughout the winter period.

After station opening on 1 November 1981, the throat swabs were inoculated into semicontinuous (MRC-5) and continuous (Hep-2, Hela M, LLC-MK2, and MDCK) cell cultures for virus isolation. A virus producing cytopathic effect in MRC-5 cells only was recovered from subject 5 from a specimen collected 27 July 1981, 4 days after the onset of symptoms. The virus was grown to high titer, lyophilized, and shipped to the U.S. in dry ice. The virus was subsequently shown, by M.K. Cooney, Department of Pathobiology, University of Washington, Seattle, Washington to be equally neutralized by monotypic antisera prepared against rhinovirus prototype viruses 28 and 53. No other cytopathic or hemadsorbing viruses were recovered from acute specimens collected during the 1981 winter.

Serum neutralization assays were carried out using standard methods (Lennette and Schmidt 1979), to determine the rhinovirus $28/53$ specific antibody levels in sera collected throughout the year from all volunteer subjects. The person from whom the rhinovirus was recovered (subject 5), demonstrated a sustained serologic response when the acute serum antibody titer (July 1981, less than 2) was compared to convalescent serum antibody titers (August 1981, 32) (table 1), confirming rhinovirus $28/53$ to be the etiologic agent of this RTI which occurred 25 weeks after station closing. Additional serologic responses to rhinovirus $28/53$ were detected in asymptomatic subjects (subjects 1, 9, 10, and 12–15) both during the austral summer prior to station closing, and periodically throughout the winter isolation period. Serum neutralization titers to human parainfluenzavirus type 3 were also determined on all subjects (table 2), and a significant serologic response was demonstrated in one symptomatic subject (subject 1) following his RTI which

occurred in April. In addition serologic responses were demonstrated in asymptomatic subjects prior to and during the winter isolation period. No antibody response by any subject was detected by neutralization assays to parainfluenzavirus type 2. While the observed serologic responses suggest infection by parainfluenzavirus type 3, the absence of virus isolation does not allow confirmation.

While human parainfluenzaviruses have been implicated as etiologic agents of mid-isolation RTI's at South Pole Station in 1976 (Muchmore et al. 1978) and 1978 (Muchmore et al. 1979), this is the first time a rhinovirus has recovered from any subject during the winter at South Pole Station. The origin of rhinovirus $28/53$ within this community is unknown. Other rhinovirus types and parainfluenzaviruses types 1 and 3 have been recovered frequently during the outbreaks of RTI which occur during the austral summer seasons each year at McMurdo and South Pole Stations (Parkinson et al. 1976; Parkinson, Muchmore, and Scott 1979; Meschievitz et al. 1982). The presence of serologic responses in South Pole Station subjects to both parainfluenzavirus type 3 and rhinovirus $28/53$ during the 1980–1981 austral summer, suggests the presence of these viruses within this community before the station closed for the winter in February 1981. While prolonged survival of rhinovirus within the indoor station environment is unlikely, long-term virus survival in the cold, dry outdoor south polar environment or the introduction of rhinovirus type $28/53$ to this community during the 22 June midwinter air-drop cannot be excluded. However, the presence of antibody, together with specific serologic responses to rhinovirus $28/53$ among asymptomatic subjects after station closing and before the midwinter air-drop, suggests survival and persistence of virus within this community by person-to-person transmission of asymptomatic infection.

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Table 1. Geometric mean neutralization titers of subjects with serologic responses to rhinovirus type 28/53 from November 1980 through October 1981 and September 1982

Subject	Austral summer				Winter isolation								
	11/80	12/80	1/81	2/81	3/81	4/81	5/81	6/81	7/81	8/81	9/81	10/81	9/82
1	<2	<2	6 ^a	<2	<2	<2	<2	<2	<2	<2	<2	<2	— ^c
5	<2	<2	<2	<2	<2	<2	<2	<2	<2 ^b	32	32	16	16
9	32	24	32	16	12	4	16	8	4	4	8	6	—
10	2	<2	<2	2	<2	<2	<2	12	<2	2	2	2	—
12	12	12	24	16	16	12	24	4	4	32	64	16	—
13	4	6	4	4	—	2	2	8	8	3	2	6	—
14	4	4	16	6	4	6	8	16	4	6	8	8	—
15	2	<2	2	<2	2	<2	2	2	<2	<2	<2	4	—

^a The underlined numbers indicate a change in titer fourfold (two tubes) or greater.

^b Respiratory tract infection (rhinovirus type 28/53 isolated).

^c "—" denotes no data available.

Table 2. Geometric mean neutralization titers of subjects with serologic responses to parainfluenzavirus type 3 from November 1980 through October 1981

Subject	Austral summer			Winter isolation								
	11/80	12/80	1/81	2/81	3/81	4/81	5/81	6/81	7/81	8/81	9/81	10/81
1	240	320	240	320	480	<u>60^a</u>	240	<u>960</u>	240	160	160	480
2	80	120	160	>2560	120	160	<u>120</u>	<u>480</u>	80	160	<u>160</u>	<u>640</u>
3	120	<u>40</u>	<u>160^b</u>	240	120	80	80	240	120	160	80	240
6	80	240	<u>120</u>	<u>480</u>	120	120	<u>120</u>	<u>480</u>	60	120	240	640
7	—	120	320	960	640	320	<u>80</u>	<u>640</u>	320	480	240	640
10	40	<u>20</u>	<u>120</u>	160	80	60	<u>60</u>	<u>480</u>	40	60	<u>80</u>	<u>320</u>
14	120	320	320	160	120	<u>120</u>	<u>480</u>	480	420	120	120	320
15	60	<u>20</u>	<u>160</u>	160	80	80	60	120	60	80	<u>40</u>	<u>240</u>
16	—	60	120	120	160	60	60	<u>80</u>	<u>320</u>	240	120	240
17	—	—	40	30	60	40	40	<u>40</u>	<u>160</u>	80	30	30

^a Respiratory tract infection.

^b The underlined numbers indicate a change in titer fourfold (two tubes) or greater.

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