# The History Of Chimpanzee Coryza Agent (Renamed As Respiratory Syncytial Virus RSV) And Acute Respiratory Disease In Children.
## By Viera Schiebner, PhD

Morris et al. (1956) described the recovery of a cytopathogenic agent that produced acute respiratory illness in chimpanzees and possibly in human beings. During October 1955, a respiratory illness characterised by coughing, sneezing, and mucopurulent nasal discharge occurred in a colony of *20 "normal" chimpanzees at the Walter Reed Army Institute of Research*. They named it the chimpanzee coryza agent.

Chanock (1956) wrote on the association of a new type of cytopathogenic myxovirus with infantile croup. The viruses produced an unusual "sponge-like" cytopathogenic change in monkey kidney tissue culture, isolated from the pharyngeal swabs from 2 of 12 infants with croup. The infants from whom the agents were isolated and 3 additional patients developed significant increases in neutralising or haemoagglutination-inhibition and complement fixing of all 3 varieties of antibodies during convalescence.

Chanock et al. (1957) and Chanock and Finberg (1957) reported on two isolations of a similar agent from infants with severe lower respiratory illness (bronchopneumonia, bronchiolitis and laryngotracheobronchitis). They also found serological evidence of infection in a number of additional children from whom they could not isolate the virus. This infection was shown to occur in a significant portion of outpatients with respiratory infections. *The two human viruses were indistinguishable from an agent associated with an outbreak of coryza in chimpanzees (CCA virus) studied by Morris et al. (1956).* A person working with the infected chimpanzees subsequently experienced respiratory infection and, although virus isolation attempts were unsuccessful, a rise in antibody for CCA virus was observed during convalescence. They mused that there was a suggestion that CCA was a virus of human origin, which produced an outbreak of mild respiratory illness when introduced into a susceptible population of chimpanzees. They proposed a name for this agent "respiratory syncytial virus."

Beem et al. (1960) isolated the virus from inpatients and outpatients seen in the Bobs Roberts Memorial Hospital for Children of the University of Chicago during the winter of 1958-1959, and provided additional evidence that this agent is associated with acute respiratory illness in humans.  They named it Randall virus.  It had an unusual cytopathic effect on H Ep-2 cells characterised by the formation of extensive syncytial areas and giant cells.  Over the course of the next five months, 48 similar agents were isolated from 41 patients.  Complement-fixation tests and cross-neutralisation tests using serums from rabbits made hyperimmune to Randal virus and the Long and Sue strains of chimpanzee coryza virus showed antigenic similarities between all three viruses. The youngest patient from whom the Randall virus was isolated was 3 week and the oldest 35 years. *The clinical diagnoses included acute respiratory disease, croup, bronchiolitis, pneumonia and asthma.  Randal virus, antigenically similar to chimpanzee coryza virus produces infection and illness in humans*.  It could be demonstrated in respiratory-tract secretions of children with acute respiratory disease but not from such material obtained from a control group without respiratory illness.  The occurrence was not linked to any other microorganisms, such Myxovirus parainfluenza, types 2 and 3, of beta-hemolytic streptococci, Diplococcus pneumoniae and H. influenzae.  The illnesses of patients from whom the virus was isolated ranged from mild coryza symptoms to fatal bronchiolitis.  The rate of isolation was particularly high among infants less than six months of age (46%), compared with older patients (16 %).  Beem et al. (1960) hypothesised that the difference in age distribution was due to a smaller likelihood of isolating virus from infected older patients.  In general, the clinical illness of infants was more severe and protracted.  The reader now knows better.

In Australia, Lewis et al. (1961) and Forbes et al. (1961) isolated further viral specimens which corresponded in tissue-culture behavior and clinical association to respiratory syncytial virus.  They wrote that for several years, prior to July 1960, the influenza and parainfluenza virus groups were the predominant causes of epidemic respiratory infections. *In July 1960, the pattern changed abruptly, with a sudden increase in number of infants admitted with bronchiolitis and bronchitis and a concomitant increase in infants with pneumonia*.   These were not paralleled by an increase in croup or by a similar increase in pneumonia in older age groups.  Prior to this, the diagnosis of bronchiolitis was infrequent.  Two hundred and forty-four children under the age of four years were admitted to hospital with the diagnosis of bronchiolitis, bronchitis or pneumonia during this epidemic.  Fifty eight percent were less than 12 months old, and patients under the age of four years

dominated the group of respiratory infections at that time.  The major clinical feature of the group was the large proportion of infants with "wheezing" or "bronchospasm" associated with other evidence of pulmonary infection.  This epidemic pattern was repeated in April 1961, when 237 patients under the age of four years were admitted, of whom 55% were aged less than one year. It was shown by Dr Ferris and his colleagues, that while the croup in 1960 was due predominantly to parainfluenza virus infection, and that in 1961 to parainfluenza and influenza type B viruses, the *infants with brochiolitis and severe bronchitis yielded a respiratory syncytial agent not previously isolated.*  The majority of infants had a history of two to three days' respiratory illness, often coryzal in onset.  In milder cases, the diagnosis of bronchitis was used, but there were many more severe infections, with dyspnoea with expiratory wheezing (different from that seen in croup), tachycardia in all cases, with inspiratory rhonchi and rales.  All this indicated inflammatory narrowing rather than muscular spasms of bronchioles.  There was a disparity between seriousness of symptoms and temperature, which was either normal or only slightly elevated.  There was also a relative absence of x-ray findings.  Some cases had a paroxysmal cough.  Two deaths occurred during each epidemic. According to Kravetz et al. (1961), during their study it was found that *the simian kidney vacuolating virus SV40 was present in the RS virus inoculums and caused inapparent infection in some volunteers*. [It would be interesting to find out how many later on developed any characteristic tumours caused by SV40 virus.  Unfortunately, we shall never know].

Rogers (1959) wrote an essay on the changing pattern of life-threatening microbial disease.  He wrote that the hope that antimicrobial drugs would abolish infections as a cause of death has not been adequately realised.  Microbial infections continue to pose life-threatening problems and antibiotic drugs do not offer susceptible human beings significant protection from certain types of microbial disease.  During the course of intrahospital survey it became apparent that the specific microbial causes of serious disease in hospital practice and the actual contribution of infection to deaths in hospitalised patients in 1959 were somewhat different from those suggested in the current literature.  In this paper, Rogers (1959) analysed the incidence and nature of infections contributing to deaths on the medical service of The New York Hospital, contrasting the period 1957-1958 with the period 1938-1940.  The clinical records, laboratory data and autopsy protocols of 200 consecutive medical-service patients subjected to post-mortem examination between 1938 and 1940 and an equal number of those dying in 1957 and 1958 were analysed in detail.  While pneumococcal infection played a major role in the earlier pre-antibiotic times, pneumococcal infection played a minor role in the antimicrobial era.  Streptococcal infection was directly responsible for the deaths

of 14 patients during 1938-1940.  Significant antecedent disease was present in all 14 patients.  In contrast, streptococcal infection did not play a part in any of 200 deaths occurring during 1957-1958.  The same trend was observed with tuberculous and staphylococcal infections.

An inactivated RS virus vaccine has been developed and tested in children. Kapikian et al. (1969), Fulginiti et al. (1969), and Chin et al. (1969) reported on the results of field trials. They all concluded that the vaccine not only failed to offer protection, but also induced an exaggerated, altered clinical response to naturally occurring RS virus infection in the younger vaccinees, as *nine (69%) of 13 vaccinated and only 4 (9%) of nonvaccinated Harrison Cottage residents 6-23 months of age developed pneumonia*.  The paradoxical effect of vaccination suggests that serum antibody may play an active role in the pathogenesis of RS virus disease (Kapikian et al. 1969).  According to Fulginiti et al. (1969), three injections of an aquaeous trivalent parainfluenza vaccine failed to provide significant protection against natural disease caused by the parainfluenza viruses, while *with exposure to natural infection, an unexpected increase in the incidence of RS virus illness requiring hospitalisation among vaccines was observed as compared to control groups*.  This difference was most significant in the 6-11 month old group (13.7% were hospitalised with RSV illness as compared to only 0.86% of an age-matched unvaccinated control group).  They ascribed this to delayed hypersensitivity caused by the vaccine.

Chin et al. (1969) reported very high attack rates of parainfluenza virus, types 1 and 3 and RS virus during the study period of infants and children in both vaccine groups.  A protective effect was not demonstrated for either vaccine.  *Infants who received the RS virus vaccine and who subsequently became infected with RS virus tended to have a more severe clinical illness than infants who did not receive the vaccine.*

Additionally, Forsyth (1969) described a development of delayed dermal hypersensitivity in Guinea Pigs vaccinated with inactivated RSV vaccines.  As with alum containing DPT vaccine (in contrast to aequeous vaccines), animals vaccinated with bovine kidney parainfluenza vaccine developed delayed dermal hypersensitivity when skin tested with bovine kidney RS vaccine. Partially purified RS antigen A and B did not elicit delayed dermal hypersensitivity.

Going back to Rogers (1959): there was an impressive increase in the number of enterobacterial infections of life-threatening concern observed during the latter period of the study (1957-1958).  Only 9 patients dying during 1938-1940 had

important enterobacterial infections. In contrast, 22 patients with infections due to gram-negative bacilli were observed during the post-antimicrobial period, and indeed such infections represented the most common microbial cause of death on the medical service during this period. There was also a shift in the place where infection was acquired. *During the preantimicrobial era, most infections were acquired before admission to hospital, while in the postantimicrobial era the vast majority of infections arose in hospital.*

Mycotic infections, were also more frequent in patients hospitalised in 1957-1958, compared with the postantimicrobial era; infections produced by strains of Candida albicans becoming a major problem. Unusual generalised clostridial infections arose as serious illness. In contrast to this, apparently, deaths due to infections produced by pneumococci, streptococci and tubercle bacilli diminished in the postantimicrobial era.

In summary, Rogers (1959) concluded that his figures support the impression that *antimicrobials have not dramatically altered the risk of, or mortality resulting from, endogenous infections arising in sick, hospitalised patients*. Microbial disease remains a significant medical problem. What is puzzling to me is the total disregard for these findings by medical system and the continued mass use, and the development of many more, antibiotics since 1959 to this day by the drug companies.

Right through the seventies, eighties, nineties and to this day, a great number of papers have been published dealing with the pandemic of bronchiolitis caused by respiratory syncytial virus in infants and young children. Some started in the present, without mentioning the original virus name chimpanzee coryza virus, others started from the original published research and mentioned chimpanzee coryza virus. They all agreed, though, that *RSV became the most prevalent cause of bronchiolitis in very young children, with many cases serious and even fatal.* All represent jigsaw pieces in a tell-tale story. One of such publications is that by Levy et al. (1997) who wrote that "Respiratory syncytial virus (RSV) is the most common cause of lower repiratory tract infection (LRTI) in infants and young children. Infection with RSV is a major health problem during early childhood and primary RSV infection occurs most often between the ages of 6 weeks and 2 years. *Approximately one half of all infants become infected with RSV during the first year of life and nearly all by the end of their second year of life."  This is very strongly indicative of vaccines being the source of RSV infection*; in the US, the first vaccine is given at 6 weeks. They also wrote that in the US each year, approximately 100,000 children are hospitalised at an estimated cost of $300

million. More than half of those admitted for RSV bronchiolitis are between 1 and 3 months of age.  A real tell-tale story incriminating vaccination.

Prober and Sullender (1999) wrote that respiratory syncytial virus, "first isolated in 1955 [sic] from adolescent monkeys with severe coryza illness, was initially called chimpanzee coryza agent.  Soon thereafter, the virus was isolated from children with bronchiolitis and pneumonia and was recognized as the most common cause of annual winter epidemics of lower respiratory tract infections throughout the world. Because the virus had a proclivity for the respiratory tract and formed multinucleated giant cell syncytia in tissue cultures, it was renamed respiratory syncytial virus in 1957." They also wrote that RSV is an enveloped RNA virus in the family Paramyxoviridae.  There are two major antigenic groups of RSV, designated A and B.

Shann et al. (1999) wrote that acute respiratory tract infections kill 4 million children every year in developing countries.

Simoes (1999) wrote that "since it was identified as the agent that causes chimpanzee coryza in 1956, and after its subsequent isolation from children with pulmonary disease in Baltimore, USA, respiratory syncytial virus (RSV) has been described as the single most important virus causing acute respiratory-tract infections in children  The WHO estimates that of the 12.2 million annual deaths in children  under 5 years, a third are due to acute infections of the lower respiratory tract.  Streptococcus pneumoniae, Haemophilus influenzae, and RSV are the predominant pathogens.  Then he, surprisingly, wrote that *a formalin-inactivated RSV vaccine, tested in the 1960s, was immunogenic, with high-rates of seroconversion.  Despite this immunogenicity, vaccinated children were not protected from subsequent RSV infection.  Furthermore, RSV-naïve infants who received formalin-inactivated RSV vaccine, and who were naturally infected with RSV later, developed more severe disease in the lower respiratory tract than a control group immunized with a trivalent parainfluenza vaccine.*  He philosophised that although the age distribution of RSV infection in children in developing countries is similar to that in developed countries, older children are more severely affected in developing countries, perhaps reflecting the (unidentified) crowding, indoor smoke pollution, and malnutrition may play a part in the development of a more severe disease. The WHO-sponsored field studies were done in Indonesia, Ethiopia, Guinea Bissau, Mozambique, Nigeria, and South Africa.  *Data from ten developing countries (Argentina, Colombia, Guatemala, Kenya, Nigeria, Pakistan, Papua New Guinea, the Philippines, Thailand, and Uruguay), showed that the most frequent cause of LRT infection was RSV (70% of all cases).  Needless to say,*

*these are also the countries with intense polio vaccination. It is no secret that the source of RSV are the polio vaccines in both the developing and developed countries.*

Bent et al. (1999) performed a meta-analysis of randomised controlled trials to estimate the effectiveness of antibiotics in the treatment of acute bronchiolitis. They concluded that their meta-analysis suggests a small benefit from the use of the antibiotics erythromycin, doxycycline, or trimethoprim/sulfamethoxazole in the treatment of acute bronchitis in otherwise healthy patients. As this small benefit must be weighed against the risk of side effects and the societal cost of increased antibiotic resistance, we believe that the use of antibiotics is not justified in these patients.

Nystad et al. (1999) wrote that children who attend day care have an increased risk of asthma with early infections as a mediator risk. They have also detected a dose-response relationship between type of day care and infection. Their study was also supported by a study among Finnish children, which revealed that care in daycare centres was a determinant of acute respiratory infections in children under 2 years of age. Family daycare did not essentially increase the risk. What they did not mention is that children attending daycare centers are more likely to have received full vaccination.

Waris (1991) wrote that RSV, a member of the Paramyxoviridae family, genus Pneumovirus, is considered the major pathogen causing severe lower respiratory tract infections among infants and children. Yearly epidemics of RSV have been occurring world wide with seasonal regularity. In the Scandinavian countries a major outbreak alternates with a minor one every second year, a phenomenon, apparently, not reported elsewhere. In at least some parts of the United States, an alternation in timing but not in the size of the epidemics are evident. It has been suggested that the variations on the epidemiological peaks are caused by interference between RSV, parainfluenza viruses, and influenza virus. Time-resolved fluoroimmunoessay with monoclonal antibodies distinguishing between respiratory syncytial virus (RSV) group A and B strains were used to analyze their prevalence in Finland during 1981-1990 among 3285 patients with laboratory diagnosis of RS, most of them hospitalized. The group typing of antigens in 680 RSV-positive nasopharyngeal aspirates showed a regular alternation of group prevalence, following the cycle occurrence of the virus. Group A predominated in 73%-90% of specimens from 1981-1982, 1985-1986, and 1989-1990, whereas group b predominated in 70%-100% of specimens from 1983-1984 and 1987-1988. This indicated that children more than 6 months of age during the first infection

were more resistant to severe reinfection with the homologous than with the heterologous group virus. The study shows that group antigenic variation of RSV has a significant effect on the epidemiology of the virus.

Singleton et al. (1995) characterised the epidemiology of Alaska native children hospitalised for RSV infections. They reviewed records of hospitalisations during the winter season of 1991 to 1992 and 1992 to 1993 at a hospital in Anchorage and a rural hospital in the Yukon Kuskokwim Delta (YKD) region of southwestern Alaska. The median age of hospitalisation for RSV infection was 2 months of age for YKD residents and 4.5 months for Anchorage residents. Sixteen percent of the hospitalised YKD children were less than 1 month of age, whereas the same was true for only 3% of the Anchorage children. Eight percent of the YKD patients required mechanical ventilation, whereas none of the Anchorage patients required ventilation. The median hospital stay was 4.8 days for YKD patients and 3.2 days of Anchorage patients. The extremely high hospitalisation rate especially among very young infants in the rural YKD region point to a need for early preventative efforts.

Among the reasons mentioned by the authors for this difference, was overcrowding (with 3-4 children sleeping in the same bed), lack of flush toilets and town water, indoor smoking and younger age at infection (under one month of age). *In my opinion, this facilitated early RSV infection from older siblings already infected with RSV from their vaccines*. Very few, if any, of the native children are unvaccinated due to lack of knowledge of vaccine dangers and poor educational level.

Translated into absolute figurers, we are talking about thousands of children affected by RSV LRTIs. The authors list 91,000 hospitalisations with 2000 death annually in the US, in the peak age between 2 and 5 months.

Hadi et al. (1999) studied the effect of vitamin A supplementation on growth with varied results. They analysed data from a randomized, double-marked, placebo-controlled trial to examine the role of infections and diarrhea in modifying the growth response to vitamin A supplementation. A single high dose of vitamin A or placebo was given every 4 months to 1405 children aged 6-48 months, and 4430 child treatment cycles were used in their analysis. Their results showed that vitamin A supplementation improved the linear growth of children who have a low intake of vitamin A but this effect was muted by respiratory infections.

Shay et al. (2001) studied bronchiolitis-associated mortality and estimates of Respiratory Syncytial virus-associated deaths among US children under 5 years of age. From 1980 through 1996, the RSV proportion of LRD hospitalisations increased from 22% to 47%. They revised the original estimate of 4500 deaths due to RSVI down to 519 RSV-associated deaths annually during the study period.

Langley et al. (2003) evaluated temporal trends in hospitalisation for bronchiolitis found among Canadian children for 1980-2000. The rate of hospitalisations increased in all provinces over the 2 decades for all age groups but was highest in those aged less than 6 months. Because a concurrent increase in other respiratory diagnostic codes was not seen, it is unlikely that physician practice variation could explain this consistent trend over almost 2 decades, which may indicate a change in disease prevalence or severity.

Hoebee et al. (2003) examined the association of variants of genes encoding interleukin (IL)-4 and the IL-4 receptor alpha chain (IL-4Ralpha) with respiratory syncytial virus bronchiolitis in hospitalised infants. Polymorphisms in Il-4 (C-590T0 and IL-4Ralpha (150V and Q551R) were genotyped by restriction fragment-length polymorphism analysis. Control subjects included parents of hospitalised children (for the transmission/disequilibrium test), and a random population sample (for the case-control study). Results were analysed in a combination of these two tests, using Fisher's method. The IL-4 590T was found more frequently among children hospitalised with RSV than expected in the case-control and combination tests. Higher frequencies of both the IL-4 590T and the IL-4Ralpha allele were found in children who were more than 6 months old when hospitalised for RSV infection compared with the control group or with the younger than 6 months olds. These results indicate that gain-of-function variants of T helper type 2 cytokine genes may play a role in increasing the severity of RSV disease, which appears more pronounced after the first half-year of life.

Such result is not surprising considering that by 6 months of age most infants are given three doses of multiple vaccines which substantially suppress and damage their immune system.

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References

Morris JA, Blount RE, and Savage RE. 1956. Recovery of cytopathogenic agent from chimpanzee with coryza. Proc Soc Exp Biol & Med; 92:544-549.

Chanock RM. 1956.  Association of a new type cytopathogenic myxovirus with infantile croup.  J Exp Med; 104: 555-576.

Chanock R, Roizman B, and Myers R.  1957.  Recovery from infants with respiratory illness of virus related to chimpanzee coryza agent (CCA). I. Isolation properties and characterisation. Am J Hyg; 66: 281-290.

Chanock R, and Finberg L.  1957.  Recovery from infants with respiratory illness of virus related to chimpanzee coryza gent (CCA) II. Epidemiologic aspects of infection in infants and young children.  Am J Hyg; 66: 291-300.

Lewis FA, Rae MI, Lehmann NI, and Ferris AA. 1961.  A syncytial virus associated with epidemic disease of the lower respiratory tract ion infants and young children.  Med J Australia; December 9: 932-933.

Rogers DE.  1959.  The changing pattern of life-threatening microbial disease. NEJM; 261 (14): 678-683.

Levy BT, and Graber MA.  1997.  Respiratory syncytial virus infection in infants and young children.  J Family Practice; 45 (6): 473-481.

Prober CG, and Sullender WM.  1999.  Advances in prevention of respiratory syncytial virus.  J Pediatr; 135: 546-558.

Shann F, Woolcock A, Black R, Cripps A, Foy H, Harris M, and D'Souza R.  1999. International Conference on acute respiratory infections.  Clin Infect Diseases; 28: 189-191.

Simoes EAF.  1999.  Respiratory syncytial virus infection.  Lancet; 354: 847-852.

Bent S, Saint S, Vittinghoff E, and Grady D.  1999.  Antibiotics in acute bronchiolitis: a meta-analysis.  Am J Med; 107: 62-67.

Nystad W, Skrondal A, and Magnus P.  1999.  Day care attendance, recurrent repiratory tract infections and asthma.  Intern J Epidemiology;  28: 882-887.

Waris M.  1991.  Pattern of respiratory syncytial virus epidemics in Finland: two-year cycles with alternating prevalence of group A and B.  J Infect Diseases; 163: 464-469.

Singleton RJ, Petersen KM, Berner JE, Schulte E, Kit Chiu, Lilly CM et al,. 1995.  Hospitalizations for respiratory syncytial virus infection in Alaska native children.  Pediatr Infec Dis J; 14: 26-130.

Hadi H, Stolzfus RJ, Moulton JH, Dibley MJ, and West KP.  1999.  Respiratory infections reduce the growth response to vitamin A supplementation in a radndomized controlled trial.  Intern J Epidemiology; 28: 874-881.

Shay DK, Holman RC, Rooseve;t GE, Clarke MJ, and Anderson LJ.  2001.  Bronchiolitis - associated mortality and estimated of respiratory syncytial virus-associated deaths among US children, 1975-1997.  J Infect Diseases; 183: 16-22.

Langley JM, LeBlanc JC, Smith B, and Wang EEL.  2003.  Increasing incidence of hospitalization for brochiolitis among Canadian children, 1980-2000.  J Infect Diseases; 188: 1764-1767.

Hoebee B, Rietveld E, Bont L, an Oesten M, Hodermaekers HM, et al. 2003.  Association of severe respiratory syncytial virus bronchiolitis with Interlukin-4 and Interlukin-4 receptor alpha polymorphisms.  J Infect Diseases; 187: 2-11.

Kapikian AZ, Mitchell RH, Chanock RM, Shvedoff RA, and Stewart AE.  1969.  An epidemiologic study of altered clinical reactivity to respiratory syncytial (RS) virus infection in children previously vaccinated with an inactivated RS virus vaccine.  Am J Epidemiology; 80 (4): 405-421.

Hyun Wha Kim, Canchola JG. Brandt CD, Pyles G, Chanock RM, Jensen K, and Parrott RH.  1969.  Respiratory syncytial virus disease in infants despite prior administration of antigenic inactivated vaccine.  Am J Epidemiology; 80 (4): 422-434.

Fulginiti VA, Eller JJ, Sieber OF, Joyner JW, Minamitani M, and Meijklejohn G.  1969.  Respiratory virus immunizations.  I.  A field trial of two inactivated respiratory virus vaccines: an aqueous trivalent parainfluenza virus vaccine and an

alum-precipitated respiratory syncytial virus vaccine.  Am J Epidemiology; 80 (4): 435-448.

Chin J, Magoffin RL, Shearer LA, Schieble JGH, and Lennette EH.  1969.  Field evaluation of a respiratory syncytial virus vaccine and a trivalent parainfluenza virus vaccine in a pediatric population.  Am J Epidemiology; 80 (4): 449-463.

Beem M, Wright FH, Hamre D, Egerer R, and Oehme M.  1960. Association of the chimpanzee coryza agent with acute respiratory disease in children.  NEJM; 263 (11): 523-530.

Kravetz HM.  Knight V, Chanock RM, Morris JA. Et al. 1961.  III. Production of illness and clinical observations in adult volunteers.  JAMA; 176 (8): 657-663.

Lewis FA, Rae ML, Lehman NI, and Ferris AA.  1961.  A syncytial virus associated with epidemic disease of the lower repiratory tract in infants and young children.  Med J Australia; Dec 9: 322-323.

Forbes JA, Bennett N. McK, and Gray NJ.  1961.  Epidemic of bronchiolitis caused by a respiratory syncytial virus: clinical aspects.  Med J Australia; Dec 9: 323-325.

Johnson KM, Bloom HH, Mufson MA, and Chanock RM.  1962.  Natural reinfection of adults by repiratory syncytial virus. Possible relation to mild upper respiratory disease.  NEJM; 267 (2): 68-72.