**A TERM PAPER ON**

**WATER RESOURCES MANAGEMENT AND ENGINEERING STRATEGIES FOR HANDLING COVID-19 PANDEMIC FOR ENVIRONMENTAL HEALTH AND ECONOMIC SUSTAINABILITY**

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ABSTRACT

Human coronaviruses, first characterized in the 1960s, are responsible for a substantial proportion of upper respiratory tract infections in children. Since 2003, at least 5 new human coronaviruses have been identified, including the severe acute respiratory syndrome coronavirus, which caused significant morbidity and mortality. NL63, representing a group of newly identified group I coronaviruses that includes NL and the New Haven coronavirus, has been identified worldwide. These viruses are associated with both upper and lower respiratory tract disease and are likely common human pathogens. The global distribution of a newly identified group II coronavirus, HKU1, has not yet been established. Coronavirology has advanced significantly in the past few years. The SARS epidemic put the animal coronaviruses in the spotlight. The background and history relative to this important and expanding research area are reviewed here.

# INTRODUCTION

Water, as a social service is not only important for the proper functioning of the human system

but for overall economic development process. Pure water has no substitute being perhaps the

most versatile servant of man. Water has a very high status in the domestic and economic life

of both rural and urban dweller. It remains one of the key social amenities without which life

will be at a standstill in any environment, settlement or society (Tebbut, 1993).

In spite of its extreme importance, potable water supply in Nigeria is facing serious challenges.

Water supply in Abia State is problematic, particularly potable water. The general picture

about potable water supply in Abia State is one of either total absence or gross inadequacy of

the existing system. Successive governments have been pursuing with vigour aggressive water

supply programme. Despite these efforts in water related infrastructure, the public are still

disenchanted because access to potable water and the quality of services in this sector remains

poor.

The responsibility of providing potable water in Abia State rests solely on the shoulders of the

State Water Board. But all it has to show for this is an array of abandoned projects and obsolete

equipments that have become monuments, a situation which has resulted in one of either total

absence or gross inadequacy of potable water supply. This state of affair in which most existing

water supply projects are abandoned is very disturbing and unacceptable because water

security is not only for human consumption but provides a take-off ladder for the economic

development of any region.

It is an established fact that the demand for water is perfectly inelastic. Therefore, there is no

substitute for water. Water can be put into its best use when the water is of very good quality.

The development of adequate water supply is a function of availability of water sources;

method and efficiency of exploiting the source; and the effectiveness of the distribution system

(Chima, 1994). Hence, the availability of water sources without proper method of exploiting

them and subsequent distribution to the final consumers may result in potable water supply

shortages in relation to demand.

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[According to](https://www.who.int/health-topics/coronavirus) the World Health Organization (WHO), coronaviruses are a family of viruses that cause illnesses ranging from the common cold to more severe diseases such as severe acute respiratory syndrome (SARS) and the Middle East respiratory syndrome (MERS). These viruses were originally transmitted from animals to people. SARS, for instance, was transmitted from civet cats to humans while MERS moved to humans from a type of camel. Several known coronaviruses are circulating in animals that have not yet infected humans. The name coronavirus comes from the Latin word corona, meaning crown or halo. Under an electron microscope, the looks like it is surrounded by a solar corona. The novel coronavirus, identified by Chinese authorities on January 7 and since named SARS-CoV-2, is a new strain that had not been previously identified in humans. Little is known about it, although human-to-human transmission [has been confirmed](https://www.aljazeera.com/news/2020/01/china-confirms-human-human-transmission-coronavirus-200120162507948.html). According to the WHO, signs of infection include fever, cough, shortness of breath and breathing difficulties. In more severe cases, it can lead to pneumonia, multiple organ failure and even death. Current estimates of the incubation period - the time between infection and the onset of symptoms - range from one to 14 days. Most infected people show symptoms within five to six days. However, infected patients can also be asymptomatic, meaning they do not display any symptoms despite having the virus in their systems.

The number of fatalities from the new coronavirus has overwhelmingly surpassed the toll of the 2002-2003 SARS outbreak, which also originated in China. SARS killed about 9 percent of those it infected - nearly 800 people worldwide and more than 300 in China alone. MERS, which did not spread as widely, was more deadly, killing one-third of those infected. While the new coronavirus is more widespread than SARS in terms of case numbers, the mortality rate remains considerably lower at approximately 3.4 percent, according to the WHO. According to the Centers for Disease Control and Prevention (CDC), older people are at higher risk for severe illness from COVID-19 which may result in increased stress during a crisis. People who have severe underlying medical conditions like heart or lung disease or diabetes also seem to be at high risk for developing more serious complications from COVID-19 illness.

Since March 16, more cases were registered outside mainland China than inside, marking a new milestone in the spread of the global pandemic. The virus has spread from China all around the world, prompting the WHO to label the COVID-19 outbreak a pandemic. Human-to-human transmissions became evident after cases were recorded with no apparent link to China.

Scientists around the globe are racing to develop a vaccine but have warned it is not likely one will be available for mass distribution before 2021. Meanwhile, a growing number of countries have introduced a series of sweeping measures to slow the spread of the coronavirus, including nationwide lockdowns, bans on gatherings, closure of schools, restaurants, bars and sports clubs, as well as issuing mandatory work-from-home decrees. International airlines have cancelled flights the world over. Some countries have banned non-citizens from entering their territories, and several more have evacuated their citizens from abroad.

Chinese health authorities are still trying to determine the origin of the virus, which they say likely came from a seafood market in Wuhan, China where wildlife was also traded illegally. On February 7, Chinese researchers said the virus could have spread from an infected animal species to humans through illegally-trafficked pangolins, which are prized in Asia for food and medicine. Scientists have pointed to either bats or snakes as possible sources of the virus.

Smoking can make people more susceptible to serious complications from a coronavirus infection, the European Union agency for disease control said. In its updated assessment of the risks caused by the coronavirus, the European Centre for Disease Control and Prevention (ECDC) included smokers among those potentially most vulnerable to COVID-19. Smokers have also appeared to be more susceptible to breathing complications caused by the disease, and the ECDC said it was advisable to identify them as a potential vulnerable group, confirming an earlier assessment.

# LITERATURE REVIEW

The history of human coronaviruses began in 1965 when Tyrrell and Bynoe found that they could passage a virus named B814. It was found in human embryonic tracheal organ cultures obtained from the respiratory tract of an adult with a common cold. The presence of an infectious agent was demonstrated by inoculating the medium from these cultures intranasally in human volunteers; colds were produced in a significant proportion of subjects, but Tyrrell and Bynoe were unable to grow the agent in tissue culture at that time. At about the same time, Hamre and Procknow were able to grow a virus with unusual properties in tissue culture from samples obtained from medical students with colds. Both B814 and Hamre's virus, which she called 229E, were ether-sensitive and therefore presumably required a lipid-containing coat for infectivity, but these 2 viruses were not related to any known myxo- or paramyxoviruses. While working in the laboratory of Robert Chanock at the National Institutes of Health, McIntosh et al3 reported the recovery of multiple strains of ether-sensitive agents from the human respiratory tract by using a technique similar to that of Tyrrell and Bynoe. These viruses were termed “OC” to designate that they were grown in organ cultures. Within the same time frame, Almeida and Tyrrell4 performed electron microscopy on fluids from organ cultures infected with B814 and found particles that resembled the infectious bronchitis virus of chickens. The particles were medium sized (80–150 nm), pleomorphic, membrane-coated, and covered with widely spaced club-shaped surface projections. The 229E agent identified by Hamre and Procknow2 and the previous OC viruses identified by McIntosh et al had a similar morphology

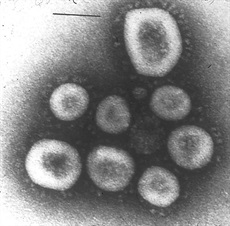


Figure ‑ Coronavirus OC16. Reprinted with permission from Proc Natl Acad Sci USA. 1967;57;933–940.

In the late 1960s, Tyrrell was leading a group of virologists working with the human strains and a number of animal viruses. These included infectious bronchitis virus, mouse hepatitis virus and transmissible gastroenteritis virus of swine, all of which had been demonstrated to be morphologically the same as seen through electron microscopy. This new group of viruses was named coronavirus (*corona* denoting the crown-like appearance of the surface projections) and was later officially accepted as a new genus of viruses.

Ongoing research using serologic techniques has resulted in a considerable amount of information regarding the epidemiology of the human respiratory coronaviruses. It was found that in temperate climates, respiratory coronavirus infections occur more often in the winter and spring than in the summer and fall. Data revealed that coronavirus infections contribute as much as 35% of the total respiratory viral activity during epidemics. Overall, he proportion of adult colds produced by coronaviruses was estimated at 15%.

In the 3 decades after discovery, human strains OC43 and 229E were studied exclusively, largely because they were the easiest ones to work with. OC43, adapted to growth in suckling mouse brain and subsequently to tissue culture, was found to be closely related to mouse hepatitis virus. Strain 229E was grown in tissue culture directly from clinical samples. The 2 viruses demonstrated periodicity, with large epidemics occurring at 2- to 3-year intervals. Strain 229E tended to be epidemic throughout the United States, whereas strain OC43 was more predisposed to localized outbreaks. As with many other respiratory viruses, reinfection was common. Infection could occur at any age, but it was most common in children.

Despite the extensive focus placed exclusively on strains 229E and OC43, it was clear that there were other coronavirus strains as well. As shown by Bradburne,coronavirus strain B814 was not serologically identical with either OC43 or 229E. Contributing to the various strain differences in the family of coronaviruses, McIntosh et al found that 3 of the 6 strains previously identified were only distantly related to OC43 or 229E.

Epidemiologic and volunteer inoculation studies found that respiratory coronaviruses were associated with a variety of respiratory illnesses; however, their pathogenicity was considered to be low. The predominant illness associated with infections was an upper respiratory infection with occasional cases of pneumonia in infants and young adults. These viruses were also shown to be able to produce asthma exacerbations in children as well as chronic bronchitis in adults and the elderly.

While research was proceeding to explore the pathogenicity and epidemiology of the human coronaviruses, the number and importance of animal coronaviruses were growing rapidly. Coronaviruses were described that caused disease in multiple animal species, including rats, mice, chickens, turkeys, calves, dogs, cats, rabbits and pigs. Animal studies included, but were not limited to, research that focused on respiratory disorders. Study focus included disorders such as gastroenteritis, hepatitis and encephalitis in mice; pneumonitis and sialo dacryoadenitis in rats; and infectious peritonitis in cats. Interest peaked particularly regarding areas of encephalitis produced by mouse hepatitis virus and peritonitis produced by infectious peritonitis virus in cats. Pathogenesis of these disease states was various and complex, demonstrating that the genus as a whole was capable of a wide variety of disease mechanisms. Human and animal coronaviruses were segregated into 3 broad groups based on their antigenic and genetic makeup. Group I contained virus 229E and other viruses, group II contained virus OC43 and group III was made up of avian infectious bronchitis virus and a number of related avian viruses.

## CORONAVIRUS GENOME AND STRUCTURE

Coronaviruses are medium-sized RNA viruses with a very characteristic appearance in electron micrographs of negatively stained preparations. The nucleic acid is about 30 kb long, positive in sense, single stranded and polyadenylated. The RNA is the largest known viral RNA and codes for a large polyprotein. This polyprotein is cleaved by viral-encoded proteases to form the following: an RNA-dependent RNA polymerase and an ATPase helicase; a surface hemagglutinin-esterase protein present on OC43 and several other group II coronaviruses; the large surface glycoprotein (S protein) that forms the petal-shaped surface projections; a small envelope protein (E protein); a membrane glycoprotein (M protein); and a nucleocapsid protein (N protein) that forms a complex with the RNA. The coding functions of several other ORFs are not clear. The strategy of replication of coronaviruses involves a nested set of messenger RNAs with common polyadenylated 3-ends. Only the unique portion of the 5-end is translated.21 Mutations are common in nature. In addition, coronaviruses are capable of genetic recombination if 2 viruses infect the same cell at the same time.

All coronaviruses develop in the cytoplasm of infected cells. budding into cytoplasmic vesicles from the endoplasmic reticulum. These vesicles are either extruded or released from the cell within the same time frame, and then the cell is destroyed.

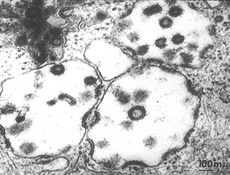


Figure ‑ Strain 229E in WI-38 cells. Reprinted with permission from J Virol. 1967;1:1019–1027.

All group I coronaviruses, including 229E, use human aminopeptidase N as their cellular receptor. Mouse hepatitis virus, a group II coronavirus, uses a member of the carcinoembryonic antigen family as its receptor. The receptor for OC43 is not known, but it may be 1 of several cell surface molecules, including 9-O-acetylated neuraminic acid and the HLA-I molecule. The SARS coronavirus uses angiotensin-converting enzyme II as its cellular receptor.

# METHODOLOGY

## Strategies for handling covid-19 pandemic for environmental

1. COVID-19 has and is likely to affect people from many countries, in many geographical locations. When referring to people with COVID-19, do not attach the disease to any particular ethnicity or nationality. Be empathetic to all those who are affected, in and from any country. People who are affected by COVID-19 have not done anything wrong, and they deserve our support, compassion and kindness.
2. Do not refer to people with the disease as “COVID-19 cases”, “victims” “COVID-19 families” or “the diseased”. They are “people who have COVID-19”, “people who are being treated for COVID-19”, or “people who are recovering from COVID-19”, and after recovering from COVID-19 their life will go on with their jobs, families and loved ones. It is important to separate a person from having an identity defined by COVID-19, in order to reduce stigma.
3. Minimize watching, reading or listening to news about COVID-19 that causes you to feel anxious or distressed; seek information only from trusted sources and mainly so that you can take practical steps to prepare your plans and protect yourself and loved ones. Seek information updates at specific times during the day, once or twice. The sudden and near-constant stream of news reports about an outbreak can cause anyone to feel worried. Get the facts; not rumours and misinformation. Gather information at regular intervals from the WHO website and local health authority platforms in order to help you distinguish facts from rumours. Facts can help to minimize fears. -2-
4. Protect yourself and be supportive to others. Assisting others in their time of need can benefit both the person receiving support and the helper. For example, check by telephone on neighbours or people in your community who may need some extra assistance. Working together as one community can help to create solidarity in addressing COVID-19 together.
5. Find opportunities to amplify positive and hopeful stories and positive images of local people who have experienced COVID-19. For example, stories of people who have recovered or who have supported a loved one and are willing to share their experience.
6. Honour carers and healthc-are workers supporting people affected with COVID-19 in your community. Acknowledge the role they play in saving lives and keeping your loved ones safe. Messages for healthcare workers
7. Feeling under pressure is a likely experience for you and many of your colleagues. It is quite normal to be feeling this way in the current situation. Stress and the feelings associated with it are by no means a reflection that you cannot do your job or that you are weak. Managing your mental health and psychosocial well-being during this time is as important as managing your physical health.
8. Take care of yourself at this time. Try and use helpful coping strategies such as ensuring sufficient rest and respite during work or between shifts, eat sufficient and healthy food, engage in physical activity, and stay in contact with family and friends. Avoid using unhelpful coping strategies such as use of tobacco, alcohol or other drugs. In the long term, these can worsen your mental and physical well-being. The COVID-19 outbreak is a unique and unprecedented scenario for many workers, particularly if they have not been involved in similar responses. Even so, using strategies that have worked for you in the past to manage times of stress can benefit you now. You are the person most likely to know how you can de-stress and you should not be hesitant in keeping yourself psychologically well. This is not a sprint; it’s a marathon.
9. Some healthcare workers may unfortunately experience avoidance by their family or community owing to stigma or fear. This can make an already challenging situation far more difficult. If possible, staying connected with your loved ones, including through digital methods, is one way to maintain contact. Turn to your colleagues, your manager or other trusted persons for social support – your colleagues may be having similar experiences to you.
10. Use understandable ways to share messages with people with intellectual, cognitive and psychosocial disabilities. Where possible, include forms of communication that do not rely solely on written information.
11. Know how to provide support to people who are affected by COVID-19 and know how to link them with available resources. This is especially important for those who require mental health and psychosocial support. The stigma associated with mental health problems may cause reluctance to seek support for both COVID-19 and mental health conditions. The mhGAP Humanitarian Intervention Guide includes clinical guidance for addressing priority mental health conditions and is designed for use by general healthcare workers.

# DISCUSSION

 Coronaviruses are common, and they are generally related to the upper respiratory tract family of disorders. They also trigger asthma in children and adults and severe respiratory disease in the elderly. Under the bell-shaped curve of respiratory infection, they probably cause pneumonia and bronchiolitis infections in the infant and child population. The clinical impact of coronaviruses has not yet been fully determined because much still remains to be discovered, despite recent research advances.

Interestingly enough SARS did not seem to be as much of a threat to infants and children. The infection appeared to be less severe in babies, and babies were also less infectious. This was evident by looking at the trend of secondary cases that developed. This is in marked contrast to the age-related severity of most respiratory viral infections. These data have provoked considerable interest and discussion, but no good explanation has surfaced. My own theory relates to the fact that almost all respiratory viral infections in adults are reinfections, and these occur on a background of partial immunity. Theoretically, if you took a virus like RSV or parainfluenza and introduced it for the first time into the human population, adults, who are infected and have no pre-existing immunity, might develop more severe disease than babies. However, until further research can verify this, it can only be seen as a theory.

# CONCLUSION AND RECOMMENDATIONS

## CONCLUSION

The field of coronavirology has advanced significantly in recent years. The SARS epidemic was a dramatic reminder that animal coronaviruses are potential threats to the human population, although the exact mechanism of species-to-species spread of the SARS coronavirus remains obscure. NL63 has been identified in many countries. This virus and the related viruses NL and HCoV-NH are likely the cause of a substantial proportion of respiratory tract disease in infants and children. The impact of HKU1 is not yet known. It seems clear that the coronaviruses infecting humans and causing respiratory disease are heterogeneous and quite widely distributed among groups I and II. It may be that some of the newer coronaviruses represent strains similar to the original B814 and OC strains that could not be further characterized in the 1960s. Additional human coronavirus strains will very likely be discovered, which stresses the need for further investigation into the virology and etiology of these infectious organisms.

## RECOMMENDATIONS

1. Stay home if you are sick or have symptoms of a respiratory infection.
2. Remember hand hygiene: wash your hands with soap and water and do not touch your nose/mouth/eyes with your hands. Cough and sneeze into the crook of your arm.
3. Keep up to date via Krisinformation.se and the Public Health Agency of Sweden web pages.
4. Don’t shake hands.
5. ​Cut back on your social contacts.

# REFRENCE

Almeida JD, Tyrrell DA. The morphology of three previously uncharacterized human respiratory viruses that grow in organ culture. *J Gen Virol*. 1967;1:175–178.

Bradburne AF. Antigenic relationships amongst coronaviruses. *Archiv Gesamte Virusforsch*. 1970;31:352–364.

Bradburne AF, Bynoe ML, Tyrrell DA. Effects of a “new” human respiratory virus in volunteers. *Br Med J*. 1967;3:767–769.

Bradburne AF, Somerset BA. Coronative antibody tires in sera of healthy adults and experimentally infected volunteers. *J Hyg (Lond)*. 1972;70:235–244

Callow KA, Parry HF, Sergeant M, Tyrrell DA. The time course of the immune response to experimental coronavirus infection of man. *Epidemiol Infect*. 1990;105:435–446.

Centers for Disease Control and Prevention. Available at <http://www.cdc.gov/ncidod/sars/index.htm>.

Collins AR. Human coronavirus OC43 interacts with major histocompatibility complex class I molecules at the cell surface to establish infection. *Immunol Invest*. 1994;23:313–321.

McIntosh K, Dees JH, Becker WB, Kapikian AZ, Chanock RM. Recovery in tracheal organ cultures of novel viruses from patients with respiratory disease. *Proc Natl Acad Sci USA*. 1967;57:933–940.

Hamre D, Procknow JJ. A new virus isolated from the human respiratory tract. *Proc Soc Exp Biol Med*. 1966;121:190–193.

McIntosh K, Becker WB, Chanock RM. Growth in suckling-mouse brain of “IBV-like” viruses from patients with upper respiratory tract disease. *Proc Natl Acad Sci USA*. 1967;58:2268–2273.

Witte KH, Tajima M, Easterday BC. Morphologic characteristics and nucleic acid type of transmissible gastroenteritis virus of pigs. *Arch Gesamte Virusforsch*. 1968;23:53–70.

McIntosh K, Kapikian AZ, Turner HC, Hartley JW, Parrott RH, Chanock RM. Seroepidemiologic studies of coronavirus infection in adults and children. *Am J Epidemiol*. 1970;91:585–592.

Monto AS. Medical reviews: coronaviruses. *Yale J Biol Med*. 1974;47:234–251.

McIntosh K, Kapikian AZ, Hardison KA, Hartley JW, Chanock RM. Antigenic relationships among the coronaviruses of man and between human and animal coronaviruses. *J Immunol*. 1969;102:1109–1118.

.

McIntosh K, Chao RK, Krause HE, Wasil R, Mocega HE. Coronavirus infection in acute lower respiratory tract disease of infants [see comment]. *J Infect Dis*. 1974;130:502–507.

Wenzel RP, Hendley JO, Davies JA, Gwaltney JM Jr., Mufson MA. Coronavirus infections in military recruits. Three-year study with coronavirus strains OC43 and 229E. *Am Rev Respir Dis*. 1974;109:621–624.

McIntosh K, Ellis EF, Hoffman LS, Lybass TG, Eller JJ, Fulginiti VA. Association of viral and bacterial respiratory infection with exacerbations of wheezing in young asthmatic children. *Chest*. 1973;63(suppl):43S.

Falsey AR, McCann RM, Hall WJ, et al. The “common cold” in frail older persons: impact of rhinovirus and coronavirus in a senior daycare center. *J Am Geriatr Soc*. 1997;45:706–711.

Falsey AR, Walsh EE, Hayden FG, et al. Rhinovirus and coronavirus infection-associated hospitalizations among older adults. *J Infect Dis*. 2002;185:1338–1341.

Haring J, Pearlman S. Mouse hepatitis virus. *Curr Opin Microbiol*. 2001;4:462–466.

Lai MM, Holmes KV. Coronaviridae: the viruses and their replication. In: Knipe DM, Howley PM, eds. *Fields Virology*. Philadelphia, PA: Lippincott-Raven, 2001.

Drosten C, Gunther S, Preiser W, et al. Identification of a novel coronavirus in patients with severe acute respiratory syndrome [see comment]. *N Engl J Med*. 2003;348:1967–1976.

Ksiazek TG, Erdman D, Goldsmith CS, et al. A novel coronavirus associated with severe acute respiratory syndrome [see comment]. *N Engl J Med*. 2003;348:1953–1966.

Peiris JS, Lai St, Poon, LL et al. Coronavirus as a possible cause of severe acute respiratory syndrome [see comment]. *Lancet.* 2003;361:1319–1325.

Guan Y, Zheng BJ, He YQ, et al. Isolation and characterization of viruses related to the SARS coronavirus from animals in southern China. *Science*. 2003;302:276–278.

Yeager CL, Ashmun RA, Williams RK, et al. Human aminopeptidase N is a receptor for human coronavirus 229E. *Nature*. 1992;357:420–422.

Williams RK, Jiang GS, Holmes KV. Receptor for mouse hepatitis virus is a member of the carcinoembryonic antigen family of glycoproteins. *Proc Natl Acad Sci USA*. 1991;88:5533–5536

Li W, Moore MJ, Vasilieva N, et al. Angiotensin-converting enzyme 2 is a functional receptor for the SARS coronavirus. *Nature*. 2003;426:450–454.

Li W, Zhang C, Sui J, et al. Receptor and viral determinants of SARS-coronavirus adaptation to human ACE2. *EMBO J*. 2005;24:1634–1643.

van der Hoek L, Pyrc K, Jebbink MF, et al. Identification of a new human coronavirus. *Nat Med*. 2004;10:368–373.

Fouchier RA, Hartwig NG, Bestebroer TM, et al. A previously undescribed coronavirus associated with respiratory disease in humans. *Proc Natl Acad Sci USA*. 2004;101:6212–6216.

Esper F, Martinello RA, Boucher P, et al. Evidence of a novel human coronavirus that is associated with respiratory tract disease in infants and young children. *J Infect Dis*. 2005;191:492–498.

Woo PC, Lau SK, Chu CM, et al. Characterization and complete genome sequence of a novel coronavirus, coronavirus HKU1, from patients with pneumonia. *J Virol*. 2005;79:884–895.

Esper F, Shapiro ED, Weibel C, Ferguson D, Landry ML, Kahn JS. Association between a novel human coronavirus and Kawasaki disease [see comment]. *J Infect Dis*. 2005;191:499–502.

Graf JD. Identification of peptide epitopes recognized by antibodies in untreated acute Kawasaki disease. Presented at the Eighth International Kawasaki Disease Symposium, San Diego, CA, 2005.

Tyrrell DA, Bynoe ML. Cultivation of viruses from a high proportion of patients with colds. *Lancet*. 1966;1:76–77.

Tyrrell DA, Almeida JD, Cunningham CH, et al. Coronaviridae. *Intervirology*. 1975;5:76–82.