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Aerobiology and Spread of Microbial Diseases

Ake Bovallius and Roger Roffey
National Defence Research Institute, S-901 82 Umea, Sweden

ABSTRACT

The different steps in spread of micro-organisms through the atmosphere causing diseases have been detailed. Case studies of a variety of disease outbursts have been correlated with the source and spread of causative agents. These micro-organisms also flourish in certain work environments causing an occupational hazard.

1. INTRODUCTION

Aerobiology as a term was coined in the 1930's to describe a multi-disciplinary research area with the aim of increasing the knowledge of how biological particles in the atmosphere are transported, become airborne, deposited and their biological effects. The biological particles considered in aerobiology covers both micro-organisms, for example bacteria, fungi, algae and viruses, as well as spores, pollen, seeds, insects and other biological material. This review mainly deals with micro-organisms, their transmission in the atmosphere and their negative effects on humans, animals and plants. Reports and reviews that cover other aerobiological particles are numerous and will only briefly be dealt with here.[1-5]

2. AEROBIOLOGICAL PROCESS

The aerobiological process consists of a series of successive steps which are dependent on each other and also on outer environmental factors. These steps are source, release, dispersion, impaction and biological effect.

2.1 Sources and Release

The sources of airborne biological particles can be of many types. Large quantities of micro-organisms are found in most environments.[6] Humans and animals carry large

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quantities of microbes, for example on the skin and in the digestive systems. From these sources they can be released by passive release (external forces) or by active release.

Unicellular organisms like virus and bacteria have no possibilities to become airborne by their own force. On a small scale this can be a human sneezing, coughing or talking. For instance one single sneeze can produce millions of droplets with a speed of 40 m/s to a distance of up to 100 cm from the mouth. Both humans and animals release small skin fragments from the body containing different bacterial species. Humans can release up to 5,000 bacteria per minute to the surrounding air.[5] This release can be a problem in operation rooms or industrial activities where a sterile environment is essential. Dental drilling, air humidifiers, and microbiological laboratory work are other examples of small scale local take-off processes. In these cases there is practically no outdoor aerosol dispersal of any significance although the initial concentration can be considerable.[5,7] In somewhat larger scale active take-off can be found in connection with different industrial or agricultural activities like sewage treatment, waste water irrigation and animal rendering.[8-14]

For large scale passive take-off of micro-organisms water, soil, dust and decaying material are commonly said to be the main sources.[4] For fungal spore liberation the wind is the dominating take-off factor. A similar behaviour is possible for bacteria. Other possible take-off mechanisms are rain splash and sea-spray which has been shown for such species as *Pseudomonas, Xanthomonas* and *Sterptomyces*.[4] Many fungi and plants also have a number of active release mechanisms.

2.2 Dispersion

The airborne particles released from its substrate or environment in different ways are transported up in the atmosphere due to turbulance and air currents. The concentration of particles in a volume of air above the ground depends on the amount of particles released from the source per unit time, on the meteorological conditions in the air mass and also on the characteristics of the particles like mass and form..

Many of the biological particles that by different means are transported up in the air are large or attached to large particles or consist of aggregates. Particles over 10-20 μm will, due to their weight, soon be deposited again on the ground. The number of airborne particles will be high during a short time. Due to rapid evaporation (a 0.1 mm droplet will evaporate in only 1 second) they have a better chance to stay airborne especially if the relative humidity is low.

It has been found that intramural aerosols have a size range that is smaller than in outdoor air. In many work environments very high total counts have been found which are much higher than usually found in outdoor environments. Examples of counts that can be found in different working environments are summarized in Table 1. It is mainly bacteria and spores of fungi that, due to the risk for spread of infectious diseases and various occupational allergies, have been monitored in indoor environments.

Table 1. Concentration of viable micro-organisms in the air of different occupational environments

Working place	Bacteria x $10^3/m^3$	Fungi x $10^3/m^3$	Ref
Sewage treatment	10–300		15
Do	100		16
Cotton mill	17	6	17
Do	10	–	18
Do	1200	200	19
Chicken rendering plant	23	2	20
Storage house	18	1	20
Poultry slaughter house	20		15
Swine house	170	–	21
Mushroom green-house	200	10	22
Saw mill	10	–	15
Garbage treatment	30	10	15
Compost	5	–	23
Bakery		6	24
Underground in London	4		25

The composition and concentration of the airborne flora shows great variations depending on geographical locality, meteorological situation, time of day and sampling techniques used.[4] For example it can be mentioned that the concentration of airborne viable bacteria[26,27] over sea lies around 10 bacteria/m,[3] somewhat higher over agricultural[28-30] land around 10-100 bacteria/m,[3] and in a city still higher[28,30-36] around 100-100000 bacteria/m.[3] The same type of results were found as early as 1900 by Miquel.[37] The biological particles also show a seasonal[28] and diurnal variation in concentration. As one example the highest concentration for bacteria was found in the afternoon in a city,[30] while for the fungi *Cladosporium* it was found before noon.[38]

Attempts have been made to characterize the collected bacteria taxonomically but this has shown a very complex pattern and studies using numerical taxonomy have shown that hundreds of different species may be present.[4,35,39,40] Common types of bacteria have been found to be *Micrococcus Bacillus* species and gram negative rods. Similar studies have been carried out for fungi where *Cladosporium* and *Alternaria* species have been found to be common.[4,38,41,42]

When a micro-organism becomes airborne the immediate fate of the particle largely depends on the local meteorological conditions. A cloud of particles follows the movement of the wind. Depending on the turbulance of the air, the cloud will become more or less dispersed. The turbulance is dependent on the ground topography, the temperature in the air mass and the wind speed. Near small sources of biological aerosols, especially those that produce an aerosol under a short period of time like coughing and sneezing, the concentration of aerosol particles will rapidly decrease as the distance increases. Only a few metres from the source it will be difficult to detect an increased concentration.

The most studied examples of small scale sources outdoors is the spread of micro-organisms from sewage treatment plants. The concentration near such aerosolizing[9,12,43,44] sites may be in the range of 10^4-10^5 bacteria/m.[3] Among potential pathogenic bacteria that have been isolated from downwind aerosols are *Escherichia, Aerobacter, Klebsiella, Salmonella, Streptococcus, Proteus* and *Pseudomonas.*[10,12] There are no verified cases of increases in disease near such activities and only very few reports give an indication that this might be the cause. In one investigation a 50 per cent higher incidence of influenza and a 20 per cent increase in common colds, but no increase in pneumonia, have been found among workers in waste water treatment plants in contrast to workers in water works.[45] Waste water spray irrigation have in one case been shown to increase the enteric diseases in the near surroundings. The increase of shigellosis, salmonellosis, typhoid fever and infectious hepatitis was two to four times higher in communities practising waste water irrigation.[46]

For the long range transmission of micro-organisms the size of the source needs to be large. Examples of such sources can be densely populated areas, wind erosion of soil and sea-spray where the turbulent airflow cause micro-organisms to become airborne. Dispersal of bacteria from these sources can be expected but there are few systematic investigations concerning this. Sampling from aeroplanes in a land air mass as it traversed on ocean showed a consistent pattern of decrease. At 152 m above the sea level the number of bacteria decreased from 600 bacteria/m^3 at the coast to 40 bacteria/m^3 640 km from the coast.[47] In connection with an air sampling study in Sweden it was possible to prove that airborne bacterial spores collected originated from the Black Sea area, a distance[48] of 1800 km.

Observations in another study showed in one case that the percentage of spore-forming bacteria is larger (57 per cent) in air masses that have travelled across the sea than in air masses originating over land[49] (7 per cent).

One important factor for the transmission of micro-organisms over long distances is their stability against external forces so that they will remain viable. In the aerosol state there are many factors that will determine the survival time for the organism why it is very difficult to distinguish the effect of each separate factor. The knowledge concerning survival in the natural outdoor environment is very limited and most data have been collected under simulated laboratory conditions. There are however, one series of experiments to simulate exposure of bacteria in the airborne state under natural conditions the organisms were attached to spider webs, and the effects of different meteorological conditions, gases and traces of chemicals were examined. A bactericidal effect due to unstable "open air factor(s)" not present in clean air was found. Motor traffic and especially unburned olefins from engine exhaust that may become bactericidal when mixed with ozone, was suggested as at least one explanation.[50-54]

There are many theories which try to explain the reversible or lethal injuries on micro-organisms in the aerosol state. Some claim that the water transport in the micro-organism is the most important factor for survival while others claim that oxygen is the most important factor.[4] The decimal reduction rate shows a wide range of values for different organisms and a minimum of 1–1500 minutes and a maximum of 50 - >19,000 minutes can be found in the literature.[55] This gives an indication of the magnitudes that can be found and the great variation found in experimental results.

2.2.1 Vertical Transmission

Miquel found that the levels of bacteria were less on the roof of pathenon in Paris than at the street[37] (5 per cent less on the roof). Using balloons Flemming in the beginning of 1900 found micro-organisms at a height[56] of 4,000 meters. In USA using aircraft pollen and spores (*Alternaria, Puccinia* and *Cladosporium*) were found at 3,300 meters height.[57] During 1950 micro-organisms were collected from 6,000 meters height over the North Pole.[58] In Sweden studies have also been carried out of bacteria and fungi at different heights.[6,59] Bacteria and spores of fungi have been isolated from material collected[60] from 20 km. Using rockets,Russian scientists claim that they have collected micro-organisms like *Mycobacterium, Micrococcus, Aspergillus* and *Pencillium* from 48 to 77 km in the atmosphere.[61]

2.2.2 Theoretical Models of Dispersal

In order to gain a better understanding of the aerobiological process and its different phases mathematical models have been constructed that simulate the natural conditions. The aim of these models is to forecast the spread of an aerosol or to trace the source of release. Most such models however, do not include biological important factors but a few that add deposition and viability has been described.[62-65]

These models discussed in the literature are said to be approximately valid up to a distance[64,65] of 1-10 km. Concerning estimates of long-distance downwind concentrations from large scale sources different types of air pollution models may be used.[66,67]

2.3 Sampling Techniques in Aerobiology

There are a great variety of airborne biological particles and various reasons for sampling them. Therefore a sampling device or method should be selected only after the purpose of sampling has been established.[2,4,68]

In general it can be stated that present air sampling apparatus can be divided according to sampling principle into impaction, filtration and deposition samplers. For impaction an air current is blown or sucked by a pump through a slit or a hole towards a surface where particles are impacted. The surface can be a solid, or consist of a liquid. Impaction on solid surfaces gives a measure of the number of micro-organisms particles present. It can, though, be difficult to estimate if a particle consists of one or many small particles. If the impaction occurs on a liquid surface, particle aggregates will be broken up and an estimate can be obtained of the total number of organisms in the sample.

Filtration is the most common method for removing particles from air drawn into an entrance by suction. The air passes through a fibrous or porous medium that impacts or sieves the particles.

Liquid impingers and bubblers operate by drawing a stream of air into the bottom of a container of water or other liquid and allowing it to rise through the liquid as buoyant bubbles. During the process particles are transferred to the liquid and retained. Bubblers have been recommended for sampling delicate organisms such as algae.[69]

Many sampling instruments operating on the principles described above have been used for collecting biological particles. Most are useful only over a limited range of particle sizes but some are being used for particles that they cannot sample efficiently or representatively. In Table 2 a number of different samplers for airborne particles are listed.

Table 2. Sampling principles for airborne particles and micro-organisms

Sampling principle	Type of apparatus	Equipment
Impaction		
– Solid surface	Slit samplers	BIAP, Bourdillon, Casella
	Cascade impactors	Andersen, May impactor
	Centrifuges	Wells aircentrifuge, Reutercentrifugal sampler
	Spore traps	Hirst spore trap, Burkard spore trap
	Precipitation	Thermal, Lovelace, Electrostatic TSI 3200
– Liquid	Cyclones	Porton, FOA-cyclone
	Impingers	Allglass, Midget, GreenBurg Smith, Marx, Multistage, Bublers
Filtration	Fiberfilters	Glassfiber
	Membranefilters	Millipore, Sartorius

2.3 Deposition

Two main processes are responsible for the deposition of aerosol particles, wet and dry deposition. Sedimentation dominates for dry deposition for large particles with a radius over 20 μm.

Comparative small spores of fungi and bacteria (for example *Penicillium* spores 2-7 μm) have an insignificant sedimentation rate. They will instead be caught by turbulent impaction or electrostatic deposition, due to different electrical charges and surfaces.

With rain airborne particles can be washed out and transported to the ground where they are deposited, is called wet deposition. In snow and rain both bacteria and fungi have been found.[4]

The fate of biological particles after landing will depend on many factors like type of surface, chemical composition and environmental factors etc. If the micro-organisms have survived the aerial transport they can grow and reproduce in the new environment. Sometimes the particles will again become airborne and be spread further to new environments. If the airborne particles are inhaled by a living organism their fate will depend on many factors. Large particles, i.e. larger than 5 μm diameter, easily adhere to the mucus membranes of the upper respiratory tracts. Continuously moving cilia guide the particles to the throat, where they are removed by coughing or swallowing. Particles smaller than 5 μm are inhaled into the deeper parts of the lungs. Many bacteria and virus are thus well adapted by their size to reach the alveoli.

Retention of the invading within the lung is a necessary, but by no means sufficient condition for the infection of the host. Invasions and infections due to retention of micro-organisms are fortunately uncommon. This is due in part to the inefficiency of natural processes in generating finely dispersed aerosols, the low survival of many airborne pathogens as well as the host defence e.g. immunity and phagocytosis. Many respiratory viruses are also able to penetrate the mucus blanket and thus infect the upper parts of the respiratory system.

3. BIOLOGICAL EFFECTS

Micro-organisms can after dispersion be deposited in different living organisms where they in some cases can cause harmful effects on humans, animals or plants. On humans these effects can be divided into the well-known infectious diseases and the less studied effects whereby micro-organisms and biological matter can cause occupational hazards. The classical effects of allergenic biological particles, like pollen or fungi etc, on humans are beyond the scope of this paper.

3.1 Human Diseases

Historically airborne contagion have been regarded as the predominant route of infection concerning most infectious diseases. With increasing awareness of transmission by water and food as well as by insects and direct contacts many cases of disease were removed from the airborne category.[70] From the beginning of the 20th century there have been a pendulum – like change in opinion of the importance of the airborne route of infection. During the 10s or 20s an almost total denial of airborne transmission was predominant.[71] In a series of papers Wells brought new insights on the transmission of contagious diseases through the air.[72-74] During the 30s and 40s many studies were performed with the aim to prove the importance of the airborne route. The results of these studies were not conclusive[75] and the belief in airborne contagion, thus, became less in the early 50s.

Direct evidences, for airborne transmission instead of indirect, started to appear in the 50s much due to the expanded controlled experimental research in airborne infection that developed e.g. military medicine.

This extensive research mostly with different bacterial species led to a far greater understanding of the mode of spread of contagious diseases. A large conference in

1960 provided a substantive base of information of the status of knowledge of airborne infections to that date.[76] Since then controlled experimental work (then also with virus aerosols) have continued through the 60s and early 70s and in a decreased volume also during the last decade. This work has been reported in a series of conferences.[5,70,77-80]

Concurrently, with laboratory experimental approaches there have been a number of careful epidemiological studies of localized outbreaks where the existence of an airborne mode of spread seems to have been well established. Thus, in aeromicrobiology today, a number of human diseases are generally recognized as at least partly airborne and several more or less critical tabulations of infectious diseases transmitted in the airborne state exist. The hazard due to the spread of infectious diseases is mainly associated with indoor environments including laboratory and hospital-acquired infections. It has also been stated that the importance of human disease transmission by biological aerosols has been in part a function of urbanization.[81] Evidence for an outdoor long range transmission of pathogenic bacteria from infected humans or animals or their surroundings are very few and the discussed distances are in the range of up to a few km. *Coxiella burnetti, Chlamydia psicatti* and *Bacillus anthracis* spores are examples of organisms that have been discussed.[5,82] In the following some examples of possible airborne transmission of infectious diseases are described with an emphasis on *Legionella spp* being a recently discovered disease.

3.1.1 Legionnaires' Disease

Legionnaires' disease was first described as the cause of an outbreak of severe pneumonia in Philadelphia[83] in 1976. The pattern of illness suggested airborne spread in a hotel but the source of spread was not identified. Since then, *Legionella pneumophila* has been identified as the cause of the disease.[84] Airborne transmission is likely to be the most common way of human infection. Several investigators have shown that *L. pneumophila* can be found in numerous environmental niches, including natural fresh-water, potable water, and cooling-tower water.[85-89] Although the members of genus *Legionella* are essentially intracellular organisms during their growth in humans they are also capable of growth *in vitro* provided very special conditions exist. To explain the high quantities of *Legionella* necessary to cause the numerous airborne outbreaks, some sort of multiplication in nature seems probable. In the laboratory it has now been shown that the viable bacterial count can be increased inside the amaeba *A. castellani*. Whether interactions of this type actually occur in *L. pneumophila* containing environments is still unknown.[49]

Outbreaks are generally associated with some human activities allowing the production of an infectious aerosol. Experimental studies have confirmed the airborne transmission.[90-93] Below a number of cases where airborne spread has been shown will be presented in more detail.

Airborne spread from sites of soil excavation was suggested as the cause of an outbreak of Legionnaires' disease in 1965 at a psychiatric hospital.[94] In that outbreak, 81 cases were identified, with 14 deaths. The epidemic curve showed two peaks, each occuring five days after the filling in of excavation sites, suggesting that in the process of closing the excavation, *L. pneumophila* may have become airborne and infected patients nearby.

Another outbreak of pneumonia occurred among golfers at a country club in Atlanta. A study showed the golfers who caught pneumonia had played considerably more golf in the period before the outbreak. Further investigations indicated that these persons had an increased exposure to the horizontal airconditioner exhaust from the wall of the club house. The bacteria was also isolated from the condenser.[95]

Airborne spread in an outbreak of Legionnaires' disease was clearly demonstrated in an outbreak in Memphis.[87] The cooling towers that usually served a large hospital was inactivated so an auxiliary cooling tower was put into service. It was the first time it had been used in two years. 39 cases of Legionnaires' disease occurred. Cases ceased to occur 9 days after the use of the auxiliary system was discontinued. *L. pneumophila* was isolated from 2 samples of water taken from the cooling tower.

3.1.2 Tuberculosis

Pulmonary tuberculosis is perhaps the most well-known example of an airborne infection, and several studies have shown that small sized aerosol particles can result in infection. The pathogenesis of the infection was studied in rabbits exposed to a cloud of *Mycobacterium tuberculosis.*[96] The primary sites of deposition were the peripheral alveoli of the lungs. This work was confirmed by Nyka[97] who exposed mice to a cloud of *M. tuberculosis* and found single bacilli in their lungs by microscopic studies.

In another study[98] the air from a series of rooms with single beds, in which patients known to have *M. tuberculosis* were nursed, was drawn through a chamber in which guineapigs were housed. Some of the animals were infected with the same strain of the bacteria.

That tuberculose aerosols can cause infections on a naval ship has clearly been shown.[82] It has also been shown that dried sedimented particles containing tuberculose could be resuspended in the air and cause infection,[99,100] though it is clear that this is an uncommon event.

3.1.3 Q-fever

Coxiella burnetti has since long been known to cause laboratory infections. This and experimental aerosol work with laboratory animals have clearly shown the potential airborne route of this organism.[101,102] The recently recognized sporeform gave further support to the possibility for natural airborne outbreaks.

A study of a Q-fever outbreak showed that almost all of the cases resided in a narrow valley. At the head of the valley in line with the almost constant wind direction was a rendering plant where sheep and goats and occasionally the placentas from these animals were processed. This was probably the source from which the infective aerosol travelled several miles down wind. Just how and when the infective aerosols were created was not certain.[103]

3.1.4 Brucellosis

Laboratory experiments clearly show that different *Brucella spp* can be transmitted by aerosols.[104] It has also been widely recognized that *Brucella spp* is a potential hazard in laboratories.[105-107] The natural route of infection has however traditionally been considered to be by consumption of pasteurized milk products or by direct contact.

However, from the 60s and onwards evidence has been gathered that natural airborne outbreaks can occur. One example is an outbreak in a large slaughterhouse where over 1000 workers were infected. Most of the men had been in direct contact with the animals, but some cases apparently involved airborne transmission.[108] Six outbreaks in abattoires have carefully been examined by Kaufmann[109] and found to be airborne.

3.1.5 Anthrax

The pulmonary form of anthrax-infection has been excessively rare in modern times.[80] Extensive laboratory studies have been performed and also in a field study monkeys were exposed to a naturally occuring aerosol of the bacterial spores.[110]

An outbreak of five cases of inhalation anthrax, four of them fatal, occurred among workers in a goat-hair processing mill. The contamination of anthrax spores in the air, measured with an Andersen sampler some months after the epidemic, clearly indicated the airborne nature of the infection. 25-30 per cent of spores were found to be less than 5 microns in diameter. 18 cases of inhalation anthrax by industrial contacts in USA since 1900 have been reviewed by Brachman.[111,112]

3.1.6 Pest

The epidemiology of the pulmonary form in nature is not fully understood.[113] Pulmonary infections with Yersinia pestis have been studied in the laboratory[114] and it has been shown that 1 micron particles initiate pneumonia in guineapigs, while inhalation of larger particles leads to septicaemia, presumably from invasion through the upper respiratory tract.

3.1.7 Psittacosis

Small outbreaks of psittacosis often follows the same route, suggesting an airborne infection. Birds in cages have been shown to cause spread of infectious aerosols giving rise to infections in humans. More extensive evidence of airborne psittacosis is found in the repeated outbreaks among employees of turkey and chicken processing plants.[115] The airborne character of these outbreaks seems clear.

3.1.8 Tularemia

Francicella tularensis was frequently studied as a possible biological warfare agents in the post-war period, and also laboratory accidents have been reported.[116-118] However, in nature, few evidences of airborne infection have clearly been shown. One example may be a tularemia epidemic in northern Finland where respiratory symptoms were observed in 72 per cent of the 50 patients. They acquired the infection during common farming activities.

3.1.9 Measles

Many investigations have shown that measles often can be airborne, but it is far from clear whether measles is solely an airborne infection, or whether contact and airborne routes both play roles under different circumstances. It is clear however that airborne infection is sufficiently common and important to be a determining factor in the continuance of measles at the present time.

From case studies there are clear evidences that people can be infected not being in the same room where the infected person has stayed. Other investigations has shown that the virus can survive and remain infective indoors over at least one hour.[119-121]

3.1.10 Smallpox

The main route of the now eradicated since a decade variola infection was by direct contact.[122] A few cases of proven airborne transmission have however been described. In an outbreak in Germany 17 cases with three deaths, occurred in an hospital with one infected patient. This was said to be the first demonstration that smallpox could be transmitted by air currents.[123]

3.1.11 Influenza

Transmission of influenza virus has been generally accepted to occur predominantly from contact–or direct droplet infection from coughing or sneezing. However, the rapidity with which influenza epidemics move through the community suggests that small particle aerosols cannot be an uncommon mode of spread of this infection. Influenza has also been studied experimentally to verify the airborne transmission.[124]

The perhaps most striking example of airborne influenza outbreak is the epidemic concerning persons on one commercial aircraft.[125] After 3 hours exposure to one infected person through the recirculated air in the aeroplane 37 of the 52 persons became ill within 4 days. Contact and direct dropleet-infection could only explain a few of the cases. A true airborne transmission must in the future be regarded as a possible way of transmission and animal models for better understanding have been developed.[126]

3.1.12 Common Cold

Experimental attempts to transmit rhinoviruses by small particle aerosols have not been very successful.[127,128] Whether these findings are representative of what happens under natural conditions is not clear and large volume air sampling has not been shown to collect rhinoviruses,[10] but on the other hand the periods of aerosol production is short and the sampling and cultivation techniques are insensitive.

Coxackie virus A 21 has with better results been collected from coughs and sneezes of infected persons and infection has been shown to be transmitted between persons with no direct contact.[129]

Also adenovirus type 4 appear to be possible to transmit in a natural way in small particle aerosols.[130]

3.2 Hazards Due to Micro-organisms in Working Environments

During the 60s and 70s it became apparent that certain types of workers' ill health were connected with airborne biological particles in the working environment, and that micro-organisms could be an occupational hazard.[10]

The symptoms are mainly of two kinds. The most common was connected with dry, dusty environments and resulted in acute symptoms including chills, fever, breathlessness, and after exposure for a longer period of time, chronic asthmatic coughs and a sense of tightness in the chest. This acute and chronic disease, allergic alveolitis, is caused by an immunological reaction of specific fungal spores and spores of the thread forming type of bacteria *Actinomycetes.* The problems are greatest in the area of agriculture, but also occur in the lumber, textile, sawmill and printing industries among others[131] (Table 3).

Table 3. Examples of fungi and actinomycetes associated with respiratory infection causing extrinsic allergic alveolitis

Disease	Source	Organism
Farmer's lung	Mouldy hay	*Micropolyspora faeni* *Thermcactinomyces vulgaris*
Bagassosis	Mouldy sugar cane	*T. vulgaris*
New Guinea lung	Mouldy roofing thatch	*Streptomyces olivaceous*
Maple bark pneumenitis	Mouldy maple bark	*Cryptostroma corticale*
Malt worker's lung	Mouldy malt	*Aspergillus fumigatus*
Mushroom worker's lung	Mushroom compost	*M. taeni, T. vulgaris*
Sequoiosis	Mouldy redwood sawdust	*Graphium and Pullalaria spp*

Farmer's lung is a common variety of allergic alveolitis and is caused by spores of *Micropolyspora* faeni and *Thermoaetinomyces vulgaris*. High incidence rate have been reported from parts of Scotland.[132] The spore concentration may reach 1.7×10^9 per m^3 when mouldy hay is shaken[133] or 2.9×10^9 per m^3 when grain silos are unloaded.[134]

The other type of symptom include acute fever, eye inflammation, and diarrhoea. The most typical environments for this type of illness are sewage plants, factories and offices with badly cleaned air humidifiers.[135] "Humidifiers fever" is a hypersensitivity reaction of the lungs that occurs in environments where indoor air has been humidified with apparatuses which use recirculating water.[136] Symptoms of humidifier fever are usually worse when affected persons after some interval return to the exposure but are relieved when the exposure continues ("Monday sickness"). Endotoxin is probably responsible for the disease.[137]

Recently, a new health hazard has been identified in very well insulated buildings which have been built during the last ten years to save energy costs. The symptoms include irritation of the eyes, nose and throat, headache, nausea and dizziness. The symptoms may be partly due to various organic substances in the indoor air, but some of the symptoms may also be allergic from bacteria and fungal spores.

3.3 Animal and Plant Diseases

There is a vast literature that covers the spread of plant and animal diseases. In many cases it can be questioned if these really have been transmitted by air. In the following a number of examples of possible airborne transmissions will be given but it does not aim at covering the whole of this area of aerobiology.

Airborne transmission of some viral animal diseases are well-known. Newcastle disease of poultry and foot and mouth disease of cattle[2,48,138,139] can cause severe economic losses for the food producing industry.

As one example of an airborne transmitted animal disease a major outbreak of FMD occurred in England in 1967-68 which became the subject of several extensive investigations.[140-145] Meteorological records were examined covering periods of previous FMD outbreaks in England and the data suggested that some of these outbreaks occurred during a period which would favour a windborne transport of infection from the continent. Snow and rain were also factors contributing to the spread of FMD.

Of the plant diseases which are caused by micro-organisms, a large majority are caused by fungi. These can form spores which are resistant to drying and can be easily spread by wind.

Only few bacteria have been described to be transmitted by air.[2] Among these, black arm of cotton, caused by *Xantomonas malvacearum* is said to be dispersed over many kilometers.[5] The spread of *Erwinia amylovara* that causes fire blight on apple and pear trees have been extensively studied since the last century and it has often been noted that fire blight seems to spread in the direction of prevailing winds.[146,147] More recently, aerosol dissemination of these plant pathogenic bacteria have been convincingly demonstrated.[148,149] However for real long distance transportation birds or insects are suggested as carriers.[147]

Evidence for the long-range dispersal of plant viruses have been discussed.[150-152] However, it remains unclear whether long-range dispersal is likely to occur only in exceptional circumstances or whether it is a regular feature in the epidemiology of plant viruses.

Evidences, that plant virus aerosols can be produced in the laboratory and that at least TMV aerosols can exist naturally in the field has however been clearly demonstrated.[153,154]

REFERENCES

Brown, R.M., Larson, D.A. & Bold, H.C., *Science*, 43 (1964), 583-585.

2. Edmonds, R.L., Aerobiology–The Eecological System Approach, (Dowden

Hutchinson and Ross, Stroudsburg, Pennsylvania), 1979

3. Faegri, K. & Iversen, J., Textbook of Pollen Analysis, (Munksgaard, Copenhagen), 1975.
4. Gregory, P.H., The Microbiology of the Atmosphere, (Leonard Hill Books, Alvesbury Bucks), 1973.
5. Hers, J.F. & Winkler, K.C., (Eds), Airborne transmission and airborne infection. Concepts and methods, presented at the 6th International Symposium on Aerobiology held at the Technical University at Enschede, the Netherlands (Oosthoek Publishing Company, Utrechdt), 1973.
6. Bovallius, A., Lihnell, D., Nilsson, S., Pegelow, K.-O., Tragardh, C. & Wiktelius, S., Biology of air. Signum, Lund (In Swedish), 1985.
7. Dimmick, R.L. & Akers, A.B., (Eds), An Introduction to Experimental Aerobiology, (Wiley, New York), 1969.
8. Adams, A.P. & Spendlove, J.C., *Science,* **169** (1970), 1218-1220.
9. Andersson, R., Bergstrom, B. & Bucht, B., Air·dispersal of bacteria from a sewage treatment plant (In Swedish). *Vatten,* **2** (1973), 117-123.
10. Dart, R.K. & Stretton, R.J., Air pollution and micro-organisms. *In* Microbiological Aspects of Pollution Control (Elsevier, New York), 1977, pp. 29-51.
11. Goff, G.D., Spendlove, J.C., Adams, A.P. & Nicholes, P.S., *Health Serv. Rep.,* **88** (1973), 640-652.
12. Hickey, J.L.S. & Reist, P.C., *J. Water. Pollut. Control Fed.,* **47** (1975), 2758-2773.
13. Napolitano, P.J. & Rowe, D.R., Microbial, content of air near sewage treatment plants. Water Sewage Works, Dec. 1966, pp. 480-483.
14. Spendlove, J.C., *Public Health Rep. (U.S.),* **72** (1957), 176-180.
15. Bovallius, A. & Anas, P., FOA investigations concerning airborne micro-organisms in different workroom environments, VAV, M28, (In Swedish), 16-20 July, 1979.
16. Bergstrom, B., Bucht, B. & Hallberg, B.O., A comparison of the bacterial concentration in air in some sewage treatment works. FOA report C1536-B4. (In Swedish), 1973.
17 Cinkotai, F.F., Lockwood, M.G. & Rylander, R., *Am. Ind. Hyg. Assoc. J.,* **38** (1977), 554-559.
18. Wells, W.F. & Wells, M.W., *JAMA,* **21** (1936), 1698-1703.
19. Lacey, J., *Lancet,* **2** (1977), 455-456.
20. Bhavani Shankar, K., Shanmugasundaram, S., Viswanathan, R.S. & Thiagarajan, M., *Cheiron,* **9** (1980), 219-221.

21. Curtis, S.E., Balsbaugh, R.K. & Drummond, J.G., *Appl. Environ. Microbiol.*, **35** (1978), 208-209.

22. Kleyn, J.G., Johnson, E.M. & Wetzer, T.F., *Appl. Environ. Microbiol*, **41** (1981), 1454-1460.

23. Hovsenius, G., Hygiene study of composting domestic waste and sludge, SNV, Final report project 76/192, (In Swedish), 1978.

24. Gemeinhardt, H. & Bergmann, I., *Zentralbl. Bakteriol.* Abt. II. Bd., **132** (1977), 44-54.

25. Forbes, J.G., *J. Hyg.*, **22** (1924), 124-155.

26. Pady, S.M. & Kelly, C.D., *Can. J. Bot.*, **32** (1954), 202-212.

27. Rittenberg, S.C., *J. Mar. Res.*, **2** (1939), 208-217.

28. Bovallius, A., Bucht, B., Roffey, R. & Anas, P., *Appl. Environ. Microbiol.*, **35** (1978), 847-852.

29. Kelly, C.D. & Pady, S.M., *Can. J. Bot.*,**31** (1953), 90-106.

30. Roffey, R., Bovallius, A., Anas, P. & Konberg, E., *Grana.*, **16** (1977), 171-177.

31. Buller, A.H.R. & Lowe, C.W., *Trans. R. Soc. Can.*, Sect., IV, **4** (1910), 41-58.

32. Fulton, J.D. & Mitchell, R.B., *Appl. Microbiol.*, **14** (1966), 232-236.

33. Kelly, C.D. Pady, S.M., *Can. J. Bot.*, **32** (1954), 591-600.

34. Lee, R., Harris, K. & Akland, G., *Am. Ind. Hyg. Assoc. J.*, **34** (1973), 164-170.

35. Mancinelli, R.L. & Shulls, W.A., *Appl. Environ. Microbiol.*, **35** (1978), 1095-1101.

36. Wright, T.J., Greene, V.W. & Paulus, H.J., *J. Air. Pollut. Control. Assoc.*, **19** (1969), 337-341.

37. Miquel, M.P., Les organismes vivantes de l'atmosphere, (Gauthier-Villars, Paris), 1861.

38. Makinen, T. & Ollikainen, P., *Bull. Ecol. Res. Comm. NFR.*, **18** (1973), 143-152.

39. Diab, A., Batram, F.G. & Zaidan, A., *Zentralbl. Bakteriol. Abt. II Bd.* **131** (1976), 535-544.

40. Anas, P. & Bovallius, A., *Proc. Ist. Int. Conf. Aerobiol.*, (1978), 455

41. Larsen, L.S., *Allergy*, **36** (1981), 15-22.

42. Riess, J., *Zentralbl. Bakteriol. Abt. II Bd.*, **130** (1975), 157-160.

43. Sorber, C.A., Baausum, H.T., Schaub, S.A. & Small, M.J., *J. Wadter Pollut. Control Fed.*, **48** (1976), 2367-2379.

44. Wanner, H.U., *Zentralbl. Bakteriol. Hyg. I Abt. Orig. B.*, **161** (1975), 46-53.

45. Ledbetter, J.O. & Randall, C.W., *Ind. Med. Surg.*, **34** (1965), 130-133.

46. Katzenelson, E. & Teltch, B., *J. Water Pollut. Control Fed.*, **48** (1976), 710-716.

47. Wolf, H.W., Skaliy, P., Hall, L.B., Harris, M.M., Decker, H.M., Buchanan, L.M. & Dahlgren, C.M., Sampling Microbial Aerosols, (Public Health Service, Washington), 1964.

48. Gloster, J., Sellers, R.F. & Donaldson, A.I., *Vet. Rec.*, **110** (1982), 47-52.

49. Holden, E.P., Wood, D.O., Winkler, H. & Leinbach, E.D., Intracellular growth of legionella pneumophila within freshwater amobae.

50. Benbough, J.E. & Hood, A.M., *J. Hyg. Camb.*, **69** (1971), 619-626.

51. Dark, F.A. & Nash, T., *J. Hyg., Camb.*, **68** (1970), 245-252.

52. Hood, A.M., *J. Hyg., Camb.*, **72** (1974), 53-60.

53. May, K.R. & Druett, H.A., *J. Gen. Microbiol.*, **51** (1968), 353-366.

54. May, K.R., Druett, H.A. & Packman, L.P., *Nature*, **221** (1969), 1146-1147.

55. Henningson, E. & Roffey, R., Survival of Micro-organisms in Aerosols–a Review, FOA report A 40053-B1 (In Swedish), 1986.

56. Flemming., *Z. Hyg. InfecktKr.*, **58** (1908), 345-385.

57. Stakman, E.C., Henry, A.W., Curran, G.C. & Christopher, W.N., *J. Agric. Res.*, **24** (1923), 599-606.

58. Polunin, N. & Kelly, C.D., *Nature*, **170** (1952), 314-316.

59. Tradgard, C., *Grana*, **16** (1977), 139-143.

60. Greene, V.W., Pederson, P.D., Lundgren, D.A. & Hagberg, C.A., Microbiological exploration of stratosphere; results of six experimental flights, Proc. Atmospheric Biology Conf., Tsuchiya, H.M., and Brown, A.H., (Eds), (University of Minnesota, Minneapolis), April 1964, pp. 199-211.

61 Imshenetsky, A.A., Lysenko, S.V. & Kazakov, G.A., *Appl. Environ. Microbiol.*, **35** (1978), 1-5.

62. Bovallius, A., Roffey, R. & Henningson, E., *Ann. N.Y. Acad. Sci.*, **353** (1980), 186-200.

63. Lighthart, B. & Frisch, A.S., *Appl. Environ. Microbiol.*, **31** (1976), 700-704.

64 Peterson, N. & Lighthart, B., *Microb. Ecol.*, **170** (1952), 314-316.

65. Anas, P. & Holmberg, C., A Stocastic Model to Evaluate the Dispersal of Aerosols in the Air (In Swedish) National Defence Research Institute, Sweden, FOA report C 400090-B1, 1978.

66 Draxler, R.R. & Elliott, W.P *Atmos. Environ* **11** (1977), 35-40

67 Scriven, R.A. & Fischer, B.E.A., *Atmos. Environ.*, **9** (1975), 49-58.

68 Henningson, E., Roffey, R. & Bovallius, A., *Grana*, **20** (1981), 155-159

69. Schlichting, H.E., *Air. Pollut. Cont. J.*, **19** (1969), 946-951.
70. Kundsin, R.B. (Ed), Airborne Contagion, *Ann. N.Y. Acad. Sci.*, 1980.
71. Chapin, C.V., Sources and Modes of Infection, (Wiley, N.Y.), 1912.
72. Wells, W.F., *Am. J. Hygiene.*, **20** (1934), 611-618.
73. Wells, W.F. & Fair, G.M., *Science,* **82** (1935), 280-281.
74. Wells, W.F., Wells, M.W. & Wilder, T.S., *Am. J. Hygiene*, **35** (1942), 97-121.
75. Subcommittee for the evaluation of methods of control airborne infections, Present status of the control of airborne infections, *Am. J. Public Health,* **37** (1947), 13-22.
76. Conference on Airborne Infections, Dec. 7-10, 1960, Division of medical sciences. National Academy of Sciences, Miami Beach, Florida., *Bacteriol. Rev.*, **25** (1961), 173-382.
77. Dimmick, R.L. (Ed), First International Symposium on Aerobiology, Naval Biol. Lab., Naval Supply Centr., Oakland Calif., 1963, p. 405.
78. Lepper, M.H. & Wolfe, E.K., Second International Conference on Aerobkiology Naval Biol. Lab., Oakland., *Bakt. Rev.*, **30** (1966), 485-698.
79. Silver, I.H. (Ed), •Aerobiology Proc. Third International Symposium on Aerobiology, (Academic Press, London), 1970.
80. Williams, R.E., Spread of airborne bacteria pathogenic for man. *In* Airborne Microbes, 7th Symp. Soc. Gen. Microbiol. (Cambridge University Press), 1967.
81. Finkelstein, H., Preliminary air pollution survey of biological aerosols: A literature review. APTD 69-30, US-DHEW/PHS, Natl. Air Pollut. Control Adm., Raleigh, N.C., 1969, pp. 94.
82. Honk, V.N., *Ann. N.Y. Acad. Sci.,* **353** (1980), 10-24.
83. Fraser, D.W., Tsai, T.F., Orenstein, W., Parkin, W.E., Beecham, H.J., Sharrar, R.G., Harris, J., Mallison, G.F., Martin, S.M., McDade, J.E., Shepard, C.C. & Brachman, P.S., and Field Investigation Team, *New Engl. J. Med.,* **297** (1977), 1189-1197.
84. Morris, G.K., Patton, C.M., Feelay, J.C., Johnson, S.E., Gorman, G., Martin, W.T., Skaliy, P., Mallison, G.F., Politi, B.D. & Mackel, D.C., *Ann. Intern. Med.,* **90** (1979), 664-666.
85. Band, J.D., LaVenture, M., David, J.P., Mallison, G.F., Skaliy, P., Hayes, P.S., Schell, W.L., Weiss, H.L., Greenberg, D.J. & Fraser, D.W., *J. Am. Med. Assoc.,* **245** (1981), 2404-2407.
86. Cordes, L.G., Wiesenthal, A.M., Gorman, G.W., Phair, J.P., Sommers, H.M., Brown, A., Yu, V.L., Magnussen, M.H., Meyer, R.P., Wolf, J.S., Shands, K.N. & Fraser, D.W., *Ann. Intern. Med.,* **94** (1981), 194-199.
87. Dondero Jr, T.J., Rendtorff, R.C., Mallison, G.F., Weeks, R.M., Levy, J.S., Wong, E.W. & Schaffner, W., *New. Engl. J. Med.,* **302** (1980), 365-370.

88 Fliermans, C.B., Cherry, W.B., Orrison, L.H., Smith, S.J., Tison, D.L. & Pope, D.H., *Appl. Environ. Microbiol.*, **41** (1981), 9-16.

89. Wadowsky, R.M., Yee, R.B., Mezmar, L., Wing, E.J. & Dowling, J.N., *Appl. Environ. Microbiol.*, **43** (1982), 1104-1110.

90. Baskerville, A., Fitzgeorge, R.B. & Broster, M., *Lancet,* **2** (1981), 1389-1390.

91. Fitzgeorge, R.B., Baskerville, A., Broster, M., Hambleton, P. & Dennis, P.J., *J. Hyg.*, **90** (1983), 81-89.

92. Hampleton, P., Broster, M.G., Dennis, P.J., Nenstridge, R., Fitzgeorge, R.B. & Conlan, J.W., *J. Hyg.*, **90** (1983), 451-460.

93. Meenhorst, P.L., Reingold, AlL., Gorman, G.W., Feeley, J.C., Van Cronenburg, B.J., Meyer, C.L. & Van Furth, R., *J. Infect. Dis.*, **147** (1983), 129-132.

94. Thacker, S.B., Bennett, J.V., Tsai, T.F., Fraser, D.W., McDade, J.E., Shepard, C.C., Williams Jr, K.H., Stuart, W.H., Dull, H.B. & Eickhoff, T.C., *J. Infect. Dis.*, **138** (1978), 512-519.

95. Cordes, L.G., Fraser, D.W., Skaliy, P., Perlino, C.A., Elsea, W.R., Mallison, G.F. & Hayes, P.S., *Am. J. Epidemiol.*, **111** (1980), 425-431.

96. Ratcliffe, H.L., *Am. J. Hyg.*, **55** (1952), 36.

97. Nyka, W., *Am Rev. Resp. Dis.*, **85** (1962), 33.

98. Riley, R.L., Mills, C.C., O'Grady, F., Sultan, L.U., Wittstadt, F. & Shivpuri, D.N., *Am. Rev. Resp. Dis.*, **85** (1962), 511.

99. Walter, C.W. & Kundsin, R.B., *Surg. Gynecol. Obstet.*, **111** (1960), 412-422.

100. Ochs, C.W., *J. Am. Med. Assoc.*, **179** (1962), 247-252.

101. Hall, W.C., White, J.D., Kishimoto, R.A. & Whitmire, R.E., *Vet. Pathol.*, **18** (1981), 672-683.

102. Tigertt, W.D., Benenson, A.S. & Gochenour, W.S., *Bact. Rev.*, **25** (1961), 285-293.

103. Irons, J.V., Denley, M.L. & Sullivan, T.D., Psittacosis in turkeys and fowls as source of human infection *In* Psittacosis, Diagnosis, Epidemiology and Control, Beaudette, F.R., (Ed), (Rutgers Univ. Press, New Brunswick, N.J.), 1955, pp 44-65.

104. Harper, G.J., *J. Exp. Pat.*, **36** (1955), 60-70.

105. Dalrymple-Champneys, W., Brucellosis Infection and Undulant Fever in Man (Oxford University Press, London), 1960.

106. Meyer, K.F. & Eddie, B., *J. Infect. Dis.*, **68** (1941), 24-32.

107. Trever, R.W., Cluff, L.E.I., Peeler, R.N. & Bennett, I.L., *Am. Med. Assoc. Arch. Int. Med.*, **103** (1959), 381-397.

108. Hendricks, S.L., Borts, I.H., Heeren, R.H., Hausler, W.H. & Held, J., Brucellosis outbreak in an Iowa packinghouse. I. Epidemiology, Ann. Conf. Am. Public. Health Assoc., San Francisco, 1960 (in press).

109. Kaufmann, A.F., *Ann. N.Y. Acad. Sci.*, **353** (1980), 105-114.

110. Dahlgren, C.M., Buchanan, L.M., Decker, H.M., Freed, S.W., Phillips, C.R. & Brachman, P.S., *Am. J. Hyg.*, **72** (1960), 24-31.

Brachman, P.S., *Ann. N.Y. Acad. Sci.*, **353** (1980), 83-93.

112. Brachman, P.S., Kaufmann, A.F. & Dalldorf, F.G., *Bacteriol. Rev.* **30** (1966), 646-657.

Meyer, K.F., *Pneumatic Plague Bact. Rev.*, **25** (1961), 249.

114 Druett, H.A., Robinson, J.M., Henderson, D.W., Packman, L. & Peacock, S., *J. Hyg. Camb.*, **54** (1956), 37.

Wellock, C.E. & Parker, M.F., An urban outbreak of Q-fever in northern California. Ann. Conf. Am. Public Health Assoc., San Francisco, 1960.

116. Day, W.C. & Berendt, R.F., *Infect. Immun.*, **5** (1972), 77-82.

Hood, A.M., *J. Hyg.*, **59** (1984), 497-504.

18. Schricker, R.L., Eigelsbach, H.T., Mitten, J.Q. & Hall, W.C., *Infect. Immun.*, **5** (1972), 734-744.

119. Bloch, A.B., Orenstein, W.A., Weing, W.M., *Pediatrics,* **75** (1985), 676-683.

120. Remington, P.L., Hall, W.W., Davis, I.H., Herald, A. & Gunn, R.A., *J. Am. Med. Assoc.*, **253** (1985), 1574-1577.

21 Riley, E.C., Murphy, G. & Riley, R.L., *Am. J. Epidem.*, **107** (1978), 421-432.

122. Foege, W.H., Millar, J.D. & Henderson, D.A., *Bull. World Health Organisation.*, **52** (1975), 209-222.

123. Wehrle, P.F., Posch, J., Richter, K.H. & Henderson, D.A., *Bull. World Health Organisation.*, **42** (1970), 669-679.

Noack, K., Bergmann, K.C. & Eckert, H., *Z. Erkr. Atmundsorgane.*, **159** (1982), 155-165.

125 Moser, M.R., Bender, T.R., Margolis, H.S., Noble, G.R., Kendal, A.P. & Ritter, D.G., *Am. J. Epidem.*, **110** (1979), 1-6.

126. Fairchild, G.A. & Roan, J., *Arch. Environ. Health,* **25** (1972), 51-59.

127. D'Alessio, D.J., Dick, C.R. & Dick, E.C., Transmission of rhinovirus type 55 in human volunteers. *In* Second International Congress for Virology. Melnick, J.L. (Ed), Basel, 1972 .

128. Gwaltney Jr, J.M., Moskalski, P.B. & Hendley, J.O., *Ann. Int. Med.*, **88** (1978), 463.

129. Couch, R.B., Douglas, R.G., Lindgren, K.M., Gerone, P.J. & Knight, V., *Am. J. Epidem.*, **91** (1970), 78-86.

130. Rowe, W.P., Seal, J.R., Huebner, R.J., Whiteside, J.E., Woolridge, R.L. & Turner, H.C., *Am. J. Hyg.*, **64** (1956), 211-219.

131. Pepys, J., Hypersensitivity diseases of the lungs due to fungi and organic dusts. Monographs in Allergy, Vol 4, (S.Karger, Basel), 1969.

132. Grant, I.W.B., Blyth, W., Wardrop, V.E., Gordon, K.L.M., Pearson, J.C.G. & Mair, A., *Br. Med. J.*, **1** (1972), 530.

133. Lacey, J., & Lacey, M.E., *Trans. Br. Mycol. Soc.*, **47** (1964), 547.

134. Lacey, J., *Ann. Appl. Biol.*, **69** (1971), 187.

135. Parrott, W.F. & Blyth, W., *J. Soc. Occup. Med.*, **30** (1980), 63-68.

136. MRC Symposium Humidifier Fever, *Thorax,* **32** (1977), 653-663.

137. Rylander, R., Haglind, P., Lundholm, M., Mattsby, I. & Stenqvist, K., *Clin. Allergy.*, **8** (1978), 511-516.

138. Donaldson, A.I., *Philos. Trans. R. Soc. Lond. B. Bio. Sci.*, **302** (1983), 529-534.

139. Wisniewski, J., *Medycyna Wterynayina.*, **34** (1978), 407-411.

140. Davies, W.K.D., Levis, G.B. & Randall, H.A., *Nature,* **219** (1968), 121-125.

141. Donaldson, A.I., Herniman, K.A., Parker, J. & Sellers, R.F., *J. Hyg. Camb.*, **68** (1970), 557-564.

142. Henderson, R.J., *J. Hyg. Camb.*, **67** (1969), 21-23.

143. Hugh-Jones, M.E. & Wright, P.B., *J. Hyg.*, **68** (1970), 253-271.

144. Sellers, R.F. & Parker, J., *J. Hyg. Camb.*, **67** (1969), 671-677.

145. Smith, L.P. & Hugh-Jones, M.E., *Nature,* **223** (1969), 712-715.

146. Stevens, F.L., Ruth, W.A. & Spooner, C.S., *Science,* **48** (1918), 449-450.

147. Van Der Zwet, T., *FAO Plant Prot. Bull.*, **18** (1970), 83-88.

148. Graham, D.C. & Harrison, M.D., *Phytopathol,* **65** (1975), 739-741.

149. Perombelon, M.C.U., Fox, R.A. & Lowe, R., *Phytopath. Z.*, **94** (1979), 249-260.

150. Banttari, E.E. & Venette, J.R., *Ann. N.Y. Acad. Sci.*, **353** (1980), 167-173.

151. Johnson, C.G., *Neth. J.P. Path.*, **73** (suppl), (1967), 21-43.

152 Johnson, C.G., Migration and Dispersal of Insects by Flight. (Methuen, London), 1969.

153. Banttari, E.E., Formation, dispersal, and detection of plant virus aerosols. Fourth International Cong. for Virology; 153, Centre for Agric. Pub. and Doc. Wageningen, Netherlands., 1978.

154. Banttari, E.E. & Venette, J.R., *Proc. of the American Phytopathol. Soc.*, **2** (1975), 51.