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Review

Roles of sunlight and natural ventilation for controlling infection: historical and current perspectives

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SUMMARY

Background: Infections caught in buildings are a major global cause of sickness and mortality. Understanding how infections spread is pivotal to public health yet current knowledge of indoor transmission remains poor.
Aim: To review the roles of natural ventilation and sunlight for controlling infection within healthcare environments.
Methods: Comprehensive literature search was performed, using electronic and library databases to retrieve English language papers combining infection; risk; pathogen; and mention of ventilation; fresh air; and sunlight. Foreign language articles with English translation were included, with no limit imposed on publication date.
Findings: In the past, hospitals were designed with south-facing glazing, cross-ventilation and high ceilings because fresh air and sunlight were thought to reduce infection risk. Historical and recent studies suggest that natural ventilation offers protection from transmission of airborne pathogens. Particle size, dispersal characteristics and transmission risk require more work to justify infection control practices concerning airborne pathogens. Sunlight boosts resistance to infection, with older studies suggesting potential roles for surface decontamination.
Conclusions: Current knowledge of indoor transmission of pathogens is inadequate, partly due to lack of agreed definitions for particle types and mechanisms of spread. There is recent evidence to support historical data on the effects of natural ventilation but virtually none for sunlight. Modern practice of designing healthcare buildings for comfort favours pathogen persistence. As the number of effective antimicrobial agents declines, further work is required to clarify absolute risks from airborne pathogens along with any potential benefits from additional fresh air and sunlight.

We shape our buildings, and afterwards our buildings shape us.

Winston Churchill

Introduction

There is evidence that fresh air and sunlight penetration in buildings can influence the transmission of airborne pathogens.[1,2] Before the discovery of antibiotics, both ventilation and sunlight were considered important safeguards against infection.[3] Now that microbial resistance is increasing, it may be helpful to review preventive strategies utilized during the

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pre-antibiotic era.[4] Bioterrorism poses a further potential threat to public health indoors.[5–7]

Before considering the evidence for natural ventilation, there is a need to establish the range of pathogens that could potentially spread through the air along with their relative transmission risks. There is some confusion over types of airborne particles, modes of dispersal and risk of infection.[8] Particles of different sizes pose different risks but current evidence does not provide assurance that infection control practices are appropriate for preventing transmission in hospitals.[9] Furthermore, there is a paucity of work to support the relaxation of airborne precautions for convalescent patients with specific infections.

This article examines historical and current evidence supporting the effects of natural ventilation and sunlight in reducing the risk of infection in healthcare and home environments. Fresh air may provide additional microbiocidal and/or attenuating effects other than physical dilution or displacement of airborne pathogens. We examine current confusion over definitions of particle size, transmission characteristics and associated infection risk. Given the threat from escalating microbial resistance, it is timely to consider whether buildings could be designed or adapted to limit the risk from airborne pathogens, including those released with criminal intent.

Search strategy and selection criteria

An extensive literature search was performed, using electronic and library databases and personal files to retrieve English language papers combining any infection, pathogen, infection risk and mention of ventilation, fresh air and sunlight. Foreign language articles with English translation were also included. No limit was imposed on date.

Role of air in transmitting pathogens

Historical background

In 1864, Britain's Chief Medical Officer John Simon underlined the importance of natural ventilation in wards.[10] Like Florence Nightingale, Simon supported oblong wards with sash windows reaching to the top along the two long sides, with sufficient space for one bed between each window.[3] At the time, it was believed that smallpox and other infections were contracted following the inhalation of airborne material.[11] The Victorians and Edwardians were preoccupied with ventilating their homes to stop the accumulation of 'foul air'.[12] Building codes and standards therefore set high ventilation rates in order to minimize the risk of airborne contagion.

In 1914, the British Admiralty recommended that the air change rate on the Royal Navy's ships should be 3000 cubic feet (85 m^3) per man per hour.[13] This was also recommended for British housing.[14] By the 1920s, scientific opinion had turned against the airborne transmission of respiratory diseases. Aseptic surgery and barrier nursing had shown the importance of contact in hospital infection. It was thought that respiratory infections were transmitted by large droplets over short distances or through contact with freshly contaminated surfaces; not via the air, or dust.[11] Natural cross-ventilation remained popular in hospitals, however, with open-air management of tuberculosis patients having a direct influence on hospital design.[15]

Following improvements in living conditions and anti-infective strategies, diseases such as tuberculosis, smallpox and others became less of a threat to public health. This eroded the importance of ventilation as an infection control strategy. Rather than construct buildings to prevent infection, the aim was to create comfortable conditions and remove odours produced by building occupants. Standards based on comfort remained in place until the 1973 Oil Embargo, when energy efficiency became a priority.[16] There was increased use of recirculated air, particularly in commercial buildings. Air change rates were reduced to save fuel and money. The latter may have contributed towards so-called 'sick building syndrome', characterized by a range of symptoms including headache, fatigue, dry eyes and throat, nasal congestion and dry skin.[16,17]

In 1976, an outbreak of *Legionella pneumophila* revived interest in indoor air quality in buildings, with the source traced to the ventilation and humidification system of a hotel.[18] A decade later, New York City and other urban centres in the USA witnessed resurgence in tuberculosis, including multiple-drug-resistant strains.[19,20] Then in 2003, a new virus emerged, termed severe acute respiratory syndrome (SARS). The SARS pandemic illustrated the lack of scientific evidence underpinning minimum ventilation rate guidelines.[21,22]

Current British guidance recommends passive natural ventilation rather than air-conditioning or mechanical ventilation in hospitals. This aims to control energy use and reduce carbon emissions, but it prioritizes patient comfort and containment of costs rather than infection control.[23,24] Whereas the risk of airborne transmission is recognized in specialist facilities such as operating theatres and isolation rooms, there is growing evidence that the airborne transmission of pathogens elsewhere has been underestimated.[25–27] If this is true, the potential for ward ventilation to control infection needs to be reassessed, particularly with respect to increasing antimicrobial resistance, novel airborne pathogens and existing pathogens for whom an airborne route of transmission may only just be evident.

Mechanisms for airborne transmission of pathogens

Surprisingly little progress has been made in understanding how pathogens pass from one host to the next.[28] Since the 1930s, four mechanisms of transmission have been described. These are: contact; dust; 'respiratory droplets' and 'droplet nuclei'.[11,29] There is confusion in the literature regarding the definition of these particles and their mechanisms of spread. For example, 'contact' may be used to indicate inhalation of large droplets from contagious individuals when they cough or sneeze, i.e. droplet transmission, but 'contact' may also refer to infectious particles transmitted directly from contaminated surfaces.[29] Aside from particle size, the potential for transmission depends upon dynamic factors, such as number of particles produced; velocity at which they are produced; number of micro-organisms contained within the spectrum of droplet sizes; infectious longevity of those microbes; and proximity of a susceptible target.[9,30]

Large respiratory droplets fall on to horizontal surfaces, including the ground. They contribute towards the many and varied components of dust. Dust particles can be suspended and resuspended by activities such as dressing, sweeping, or bed-making.[29] The range of these droplets is supposedly no more than 1 m, which suggests that anyone standing more than this distance from an infected person does not require

protection.[31] Resuspended dust and fibres from fabric, however, can easily be seen floating in air currents illuminated by beams of light during various energetic activities. Smaller respiratory droplets quickly evaporate, leaving residues which, in turn, become minute suspended particles or 'droplet nuclei'.[32] By contrast with larger droplets, these smaller particles can stay airborne for minutes to hours depending on size and density. Their size means that they could potentially penetrate deeper into lung tissues than droplets.[9] Droplet nuclei are also exhaled during normal breathing and talking, although the number of particles exhaled during breathing and conversation are many orders of magnitude fewer than the aerosols produced by coughing or sneezing.[9,33]

The importance and mode of airborne transmission has been a matter of dispute for decades and remains controversial.[8,34] The relative importance of droplets, droplet nuclei initially in air and raised again as a dust component, in the spread of respiratory infections remains unknown. All three modes of spread probably occur.[35] Furthermore, there is no agreed classification of airborne particles.[36] The cut-off diameter at which transmission changes from exclusively droplet to airborne droplet nuclei, or vice versa, has never been universally accepted, and indeed, may be impossible to determine. Nevertheless, current infection control procedures make a clear distinction between different types of droplet and the precautions required to prevent transmission according to presumed category of dispersal.[37] The definitive test should be efficacy of a given infection control practice.

The survival of airborne pathogens depends partially on ambient environmental factors such as temperature and humidity (relative or absolute), as well as ultraviolet (UV) radiation and atmospheric pollutants.[38] In addition, the movement of airborne droplets is affected by various other environmental factors, such as local ventilation airflows (including presence of vertical shafts), door opening, and movement of people, their clothes, and thermal gradients produced by electrical equipment.[39–41]

In healthcare premises, patients with accepted airborne infections, such as *Mycobacterium tuberculosis*, require the use of negative pressure isolation rooms. Anyone who enters an isolation room must wear a high filtration respirator, not just a surgical face mask.[42] These precautions do not necessarily apply to diseases thought to be spread by droplet transmission such as influenza virus, rhinoviruses, adenoviruses and respiratory syncytial virus.[43] If activities such as breathing, coughing, sneezing, and talking generate different sizes of particles, however, both droplets and droplet nuclei could be produced simultaneously. A recent literature review concluded that infection control precautions should be revised, so that airborne risk is considered whenever potentially infectious aerosols are generated, whatever the size of droplets.[9]

The following sections summarize current knowledge on transmission and control of a variety of pathogens, all of which may be dispersed into the air as infectious particles in one form or another.

Influenza

Influenza continues to have a major impact on global health, but knowledge of its transmission and control remains poor.[44–46] Infection control is based on the assumption that influenza is transmitted by large droplets but it is possible that aerosol transmission is responsible for the most severe cases of influenza involving the lower respiratory tract.[47] A report from 1979 describes an event involving transmission of influenza aboard an airliner, the particular circumstances of which seem to support airborne transmission of the virus over distances greater than a few metres.[48]

The possibility of aerosol transmission assumes greater importance when considering recent H5N1 avian influenza, which has demonstrated high virulence and lethality.[49] Until recently, this form of influenza could only pass between humans via physical contact.[50] Most of the reported cases were traced to direct contact with diseased poultry and other birds.[51] However, recent work suggests that H5N1 can be transmitted through the air following modification. Strains have been experimentally spread between mammals without recombination in an intermediate host.[52] This illustrates the potential for human pandemic influenza and underlines the need to revise current infection control practices in hospitals.[46]

Smallpox

In the past, smallpox was more widely believed to be airborne than any other disease.[11] When the World Health Organization launched its campaign to eradicate smallpox in the 1960s it did so on the basis that the virus spread by face-to-face contact.[53] This assumption was challenged following a smallpox outbreak at a German hospital in 1970.[54] The pattern of spread suggested airborne transmission of the virus, later supported by smoke tests. Another incident occurred in 1971 when a naval scientist contracted smallpox while offshore from a smallpox testing site in Kazakhstan.[55] The virus travelled downwind over a distance of at least 15 km to reach the ship. This raised concerns that scientists in the former Soviet Union not only 'weaponized' smallpox, but succeeded in aerosolizing it. Further evidence for airborne transmission of smallpox came in 1978 when a medical photographer contracted the disease and died.[56] The virus is thought to have dispersed via air currents up a service duct from a laboratory to the room where the victim worked.

Severe acute respiratory syndrome

The official investigation into the 2003 SARS epidemic assumed that direct contact, not air, was the main transmission route.[21] Viruses such as SARS are shed in large numbers and survive for long periods on surfaces or objects commonly found indoors.[57] Healthcare workers who avoided face-to-face contact with SARS patients were certainly at less risk of contracting the virus. This may have been due to decreased exposure to infected droplets.[58] However, the Amoy Gardens outbreak in Hong Kong suggests that airborne transmission was also involved.[59] It is possible that virus-laden aerosol from sewage passed through faulty floor drains into bathrooms, where further exposures occurred. Later investigation implied that contaminated air then rose up a ventilating shaft and prevailing winds carried it to adjacent buildings.[59,60]

Further examples include the spread of SARS at a Hong Kong hospital, thought to be due to faulty ventilation, and transmission on aircraft, suggesting that airborne droplets may have infected passengers during two separate flights.[61–63] Before SARS emerged, the dynamics of virus-laden aerosols had attracted little research, so little was known about control and prevention.[28] The SARS epidemic confirmed that the

mechanisms of respiratory disease transmission are still poorly understood.[63]

Hantavirus

Ten years before the SARS pandemic, hantavirus demonstrated transmission between animals and humans. It was widely accepted that this viral pathogen was airborne and could travel over distance. Hantavirus spreads to humans through the inhalation of aerosolized excreta and saliva from wild rodents.[64] In 1993–2007 there were 2500 cases of hantavirus with an overall mortality of 30%. This is comparable to the mortality rate for SARS during the 2003 epidemic.[65] A key preventive measure is ventilating rooms or buildings showing signs of rodent infestation, with staff advised to use disposable respirators and gloves during cleaning after the ventilation process.[64,66]

Norovirus

Norovirus causes a significant amount of gastrointestinal illness in the developed world.[67] This virus is easily transmitted between individuals, via contact with contaminated surfaces, food, and/or through aerosol spread.[68] A recent review of hospital outbreaks concluded that 18.5% were caused by person-to-person transmission; 3.7% were foodborne; and infection route for the remaining 77.8% was unknown.[69] The latter may be due to aerosolized particles from vomiting or liquid diarrhoea. A sick person can produce 10^7 virus particles per millilitre of vomit whilst faecal material contains up to 10^{12} viruses per gram.[27] Projectile vomiting is likely to be a major source of cross-infection and droplets generated by flushing toilets can be inhaled or deposited on surfaces.[27,70] The high attack rates seen during norovirus outbreaks could be due to rapid dispersal via aerosols.[71,72]

Common cold

Adults suffer between two and five colds each year, usually caused by rhinoviruses.[27,73] Despite years of study, transmission between individuals remains controversial. There is evidence that sharing office space increases the risk of catching a cold.[74] A study in mechanically ventilated office buildings found additional evidence for aerosol transmission of rhinovirus, when a low outdoor air supply resulted in an increased risk of inhaling infectious droplets.[75] A more recent study found that students accommodated in six-person rooms were twice as likely to have colds as students in three-person rooms.[76] Some 35% of students caught more than six colds each year when the ventilation rate was 1 L/s/person. If this rate was increased to 5 L/s/person, the number of students catching colds six times or more fell to 5%, suggesting that there was an association between colds and outdoor-to-indoor air flow rates; this reflects the multifactorial nature of the spread of such respiratory infections. There is evidence that hand-to-surface contacts are of greater significance, at least for respiratory syncytial virus.[77] Nevertheless, ventilation may still be important in diluting and dispersing virus-laden droplets than is currently appreciated.[78]

Tuberculosis

The transmission of tuberculosis, like human viral infections, is assumed to be an indoor event. Improved housing is one of a number of factors thought to explain the decline of tuberculosis in Britain from the mid-19th century onwards.[79] Since tuberculosis is mainly contracted through airborne droplets, it follows that transmission of *M. tuberculosis* to a non-infected person is more likely if there is overcrowding and poor ventilation.[80] Anyone who lives and sleeps in the same household as an infected person is at risk. Similarly, there is greater risk of contracting the disease in confined environments such as prisons, homeless shelters and long-term care facilities.[20] Studies on ventilation issues and transmission risk of tuberculosis in private houses have received little attention, in company with most other respiratory infections.[81] Statistics from the USA in 2002 put the healthcare costs of building-influenced communicable respiratory infections, including tuberculosis, at US$10 billion.[78]

Staphylococcus aureus

Hospital patients continue to be at risk from meticillin-resistant *Staphylococcus aureus* (MRSA).[82] Hands are presumed to be the main vehicle for transmission, but airborne contribution has been suspected since the 1960s.[83,84] This may be linked to daily shedding of 10^6–10^7 skin cells from colonized or infected patients.[41] A staphylococcal carrier with a cold also facilitates spread and has been termed the so-called 'cloud adult'.[85] Nasal cavities of susceptible adults become colonized with *S. aureus* by inhaling particles from the air.[86,87] This type of transmission is more likely when there is increased people-traffic or activities such as bed-making that precipitate air turbulence.[25,88] Recent experimental work shows that MRSA bioaerosols can deposit throughout a room with no clear correlation between relative surface concentration and distance from source.[89] Burns patients are particularly susceptible to staphylococcal infection, with airborne transmission implicated in outbreaks in hospital burns units.[90–92]

More recently, community strains have emerged that infect healthy young people who have not had prior hospital exposure.[93] It is known that persons with MRSA infection and/or carriage will readily disperse their strain throughout the home environment.[94] Contamination of household surfaces could play a major role in community-acquired MRSA, but the health effects associated with exposure to airborne *S. aureus* in housing have not yet been investigated.[95]

Aerial spread of other pathogens

Traditional airborne pathogens, such as aspergillus and *Bacillus* spp., are found throughout hospitals, particularly during hot, dry weather. These have also been associated with construction or renovation.[96,97] Other pathogens have demonstrated aerial spread in healthcare facilities, including *Escherichia coli*, klebsiella, acinetobacter, pseudomonas and *Clostridium difficile*.[98–102] Patients infected with *C. difficile* shed large numbers of spores in faeces, contaminating skin, bedding and nearby surfaces. Symptomatic patients increase the risk of airborne spores, which fall to the ground at a rate dependent on spore size.[102] Another potential source of spread is the lidless toilet frequently installed in hospitals. When a toilet is flushed without a closed lid, aerosol production may contaminate the surrounding environment with *C. difficile*.[103] The potential for airborne transmission of *C. difficile* is not adequately addressed by current control measures.[104]

Preventing infection through ventilation

The open air factor

The majority of microbes that cause airborne infections cannot tolerate sunlight, oxidants or the temperature extremes that occur outdoors. Both the Nightingale ward and tuberculosis sanatorium were designed to make conditions indoors as close as possible to those outdoors.[3,105] There may be an additional protective effect other than dilution of pathogens from high ventilation rates. In 1894, it was noticed that exposure to fresh air appeared to reduce the virulence of the tubercle bacillus. Whereas this effect persisted in well-ventilated dark spaces, tubercles retained their infectivity for long periods in the absence of fresh air or confined air.[106] In the 1960s, the lethality of outside air to micro-organisms was rediscovered and the term 'open air factor' (OAF) was introduced.[107,108] Further research showed that this ephemeral property of outdoor air had an adverse effect on viability and virulence of airborne micro-organisms, including influenza and the Category IV pathogen, *Francisella tularensis*.[109,110] These natural disinfection characteristics of outdoor air were then ignored for more than two decades.[36]

Whereas the OAF disappears rapidly in any form of enclosure, it is possible to ventilate experimental containers at rates that enable the germicidal properties of open air to be fully retained.[107] More recently, an automated air disinfection system has been shown to reduce both airborne microbial counts and environmental contamination in the healthcare environment.[111] The system produces hydroxyl radicals from a reaction of ozone and water vapour catalysed by an olefin (D-limonene).[112] Hydroxyl radicals may be present in open air, with the main source of hydroxyl being the photolysis of ozone — this could relate to the 'open air factor'. Hydroxyl radicals have been shown to possess disinfection characteristics.[111] Despite widespread acceptance of the germicidal effect of fresh air over a century ago, it remains unknown whether maintaining high natural ventilation rates in buildings would preserve the OAF and its effects on indoor pathogen counts.[106]

Open windows

Natural ventilation has a number of advantages, including relatively low cost and low maintenance. Another advantage of open doors and windows is that air can enter a building by more than one route, whereas air must enter through a specified intake with mechanical systems. This is arguably more relevant regarding malicious dispersal of toxic chemicals or biological agents. Following the 2001 anthrax attacks, it was recommended that air intakes should be relocated to publicly inaccessible locations, with intakes on large buildings placed on secure roofs or high sidewalls.[113] Mere presence of doors and windows, however, is no guarantee that they will be regularly utilized to encourage air movement. In addition, hospitals cannot necessarily keep windows open during unfavourable climatic conditions.[114]

Further disadvantages of using open windows include safety issues and entry of unfiltered air containing outdoor air contaminants such as fungal spores.[115,116] Whether originating from occupant or outside air, the quantity of potential pathogens in window-ventilated rooms is not necessarily higher than those in mechanically ventilated rooms.[117] In British hospitals, a maximum opening of 100 mm is recommended for windows within reach of patients.[118] The risk of unsupervised patients injuring themselves has to be weighed against cross-infection due to inadequate ventilation but window design could encompass both ventilation and patient safety. Designing buildings for human comfort rather than health may influence the ecology of indoor microbes.[117]

Natural versus mechanical ventilation

There is some evidence that natural ventilation can be more effective than mechanical systems for preventing transmission. During the 1918 influenza pandemic, sick patients who were accommodated in the open air survived in greater numbers than those in hospital wards.[105,119,120] Eighty years later, during Operation Desert Shield, respiratory tract infections were more frequent in military personnel in air-conditioned barracks than among those housed in tents.[121] A 1971 Scottish study reported that patients transferred from an old Nightingale-type ward to a mechanically ventilated ward had significantly fewer staphylococcal infections.[122] This did not necessarily confirm superiority of mechanical over natural ventilation for reducing infection because respective ventilation rates for both wards were not included. Nightingale wards were designed for cross-ventilation via open windows but smoke tests showed that the old ward had no set ventilation pattern, other than 'wild' air streams from sporadically opened windows.[122] There may also have been less staphylococcal debris in dust in a new ward.

In 2007, researchers measured natural ventilation in 70 different rooms containing tuberculosis patients.[123] Air exchange rates in wards and outpatient areas were compared with those in mechanically ventilated negative-pressure isolation rooms, with an airborne infection model used to predict the effect on tuberculosis transmission. The highest risk of infection occurred in mechanically ventilated rooms with sealed windows, despite being ventilated at recommended rates. By contrast, clinical rooms with high ceilings and multiple large windows in pre-1950 hospitals gave the greatest protection, even if the windows were only partially open.[123]

Naturally ventilated wards can achieve higher air change rates than those with mechanical systems.[124] Measurements on general wards found higher rates than the 12 air changes per hour specified for isolation rooms. In a ward with open doors and windows, cross-ventilation and a strong breeze, the maximum rate was 69 air changes per hour. Even without cross-ventilation, rates were between 14 and 31.6 air changes per hour.[124] Case studies from China show that cross-ventilation is useful for controlling SARS transmission in hospitals.[125] In addition, isolation wards with a high proportion of operable windows were more effective in preventing outbreaks of SARS among healthcare workers.[126] A study from Thailand reported that air change rates in naturally ventilated hospital rooms were mostly higher than recommended standards. By contrast, ventilation rates in work areas with air-conditioning were often inadequate. This was most pronounced where there was a high risk for nosocomial tuberculosis infection such as radiological and emergency room departments.[127]

Ventilation systems in hospitals may be compromised by poor design, construction or inadequate maintenance, and these failings have been implicated in outbreaks of tuberculosis.[123] Contaminated hospital ventilation systems have also been blamed for other types of outbreak.[128,129] The source of a

Serratia marcescens outbreak in a baby unit of a hospital in United Arab Emirates was traced to an air conditioner duct.[130] Faulty and/or contaminated ventilation systems have also been linked with outbreaks of MRSA.[131–133]

Ventilation in the home

Data from Germany, Canada, and the USA show that people spend most of their time indoors in private homes.[134–136] According to one estimate, more than half of the body's intake of air during a lifetime is inhaled indoors.[137] New homes in Britain and other countries make increasing use of mechanical ventilation. There is no evidence that mechanical ventilation has facilitated the spread of infection in houses, although a wide range of micro-organisms, including pathogens, may be found indoors.[138] In 2012, a study of 299 mechanically ventilated Dutch homes identified major problems with the installation, operation and maintenance of ventilation systems in homes.[139] Researchers found dust and dirt in the air supply ducts in 67% of houses fitted with mechanical ventilation heat recovery (MVHR). Air filters were dirty in almost half, with insufficient ventilation in at least one room, and exhaust air being recirculated in more than half of houses with MVHR. Most occupants did not control ventilation systems as recommended, nor use the highest ventilation settings due to noise levels.[139]

Preventing infection by sunlight

Historical background

In 1877, Downes and Blunt reported that sunlight inhibited the growth of bacteria from behind glass.[140] Later studies showed that sunlight could kill a range of bacteria, including those causing tetanus, typhoid, anthrax and tuberculosis.[141] In 1890, Koch reported that direct sunlight could kill the bacillus in a few minutes, or several hours, through glass. The time depended on the thickness of the layer of bacteria exposed. Furthermore, ordinary diffuse daylight, such as is found near windows in houses, could kill the bacterium in five to seven days.[142] Even before this, it was recognized that tuberculosis transmission was less likely to occur in clean, well-lit, well-ventilated houses or hospitals.[106]

During the 1920s, manufacturers produced glass that transmitted a greater proportion of UV radiation than achieved by ordinary glass. Solar radiation was popular at this time, so encouraging a greater proportion of sunlight into buildings was a logical development.[143] In 1930, the bactericidal power of direct sunlight, sunlight through plate glass, and sunlight through a commercial glass product (Vitaglass) was tested against *S. aureus*.[144] Exposure times ranged from 30 min in good weather to 3 h in variable conditions. Nearly three-quarters of *S. aureus* were killed by unfiltered sunlight, half died when Vitaglass was used and one-quarter died following sunlight exposure through ordinary glass.[144] These experiments were performed nearly a century ago, remain unverified and have not been recently explored.

The World Health Organization refers to sunlight in guidance on preventing hospital infections, although reasons for this are not made clear.[145] For airborne infections such as tuberculosis, one document recommends that patients should be placed in single rooms with sunlight, negative air pressure and six to 12 air changes per hour. Guidelines on healthy housing state that natural lighting should be provided for toilets, preferably using special glass that transmits a higher proportion of UV rays. If ordinary window glass is fitted, windows should be left open in warm weather for at least 3 h in order to allow penetration by shorter wavelength UV radiation, presumably to exert some bactericidal effect.[146]

Mycobacteria and sunlight

Direct sunlight kills *M. tuberculosis* but diffuse light is less effective.[147] A study from 1942 investigated the risk of tuberculosis infection among staff and patients in a Californian sanatorium. Patients were treated according to the 'open air' regimen where they were exposed to the elements day and night.[148] Environmental cultures from hand-touch sites, dust and air samples in the ward were all negative. Further investigations suggested that tubercle bacilli exposed through an unglazed north window died within four or five days depending upon original inoculum. Bacilli survived for two or three months in a drawer in the same room, for six months in a refrigerator and for longer in winter than in summer.[148] These studies have not been verified in recent times.

Streptococci and sunlight

Hospital dust near patients' beds was found to contain large numbers of streptococci during a 1944 outbreak of scarlet fever.[149] Environmental screening demonstrated the absence of viable streptococci in dust at sites near windows. Previous work had already confirmed the lethal effect of sunlight on *Streptococcus pyogenes*.[150,151] Studies showed that streptococci could survive for long periods indoors with undiminished virulence, but only about 5 min in the sun compared with more than an hour in diffuse daylight.[151] Garrod commented,

> Although good lighting is universally recognised as desirable, it has never, so far as I am aware, been insisted on as a prime necessity in wards for septic surgical cases … preoccupation with the ultraviolet part of the spectrum has led to a common belief that only direct sunlight is usefully bactericidal; it must now be recognized that ordinary diffuse daylight, even on a cloudy day and even in winter in England, can be lethal to bacteria, and that glass is no absolute bar to this effect.[149]

Diffuse daylight is more rapidly lethal for the pneumococcus than for *S. pyogenes*. A 1905 study showed that exposing dried sputum to sunlight inactivates *S. pneumoniae* within half an hour.[152] This illustrates the difficulty in separating out the effects of desiccation from that of light. Later, it was found that pneumococci in dried sputum exposed to diffuse daylight remained viable and retained virulence for extended periods of time.[153] Pneumococci can survive in sputum, with airborne transmission thought to be possible.[150,154] There are no recent studies on pneumococcal survival in the environment and no additional evidence for airborne transmission.

Meningococci and sunlight

It is assumed that the meningococcus cannot withstand desiccation. In 1944, two linked studies showed that virulent

organisms were recovered from a range of surfaces one week or more after drying and storing in a cupboard.[155,156] Meningococci were then exposed to different intensities of natural light. Direct sunlight passing through an ordinary window killed the organisms within a few hours, with less effect from diffuse daylight through a north-facing window. During cloudy weather, meningococci died more rapidly near a window than 12 feet away. Tests with coloured filters showed that whereas red light had little impact on meningococcal viability, blue light was highly bactericidal. Lethality of light transmitted by coloured filters of orange, green and yellow was proportional to the amount of blue light each filter transmitted.[156] None of this work has since been repeated.

Staphylococci and sunlight

As noted above, sunlight also kills staphylococci. Cultures were exposed to sunlight for 45 min shielded by photocopier paper, window glass and Perspex.[157] The bactericidal effect of sunlight was marginally reduced by the glass filter, whereas Perspex partially protected the staphylococci and photocopying paper inhibited the killing effect. Lethality of sunlight against staphylococci is due to radiation at 300–380 nm, with further effects apparent at shorter and longer wavelengths. In addition to bactericidal effect, solar radiation is mutagenic. It has long been believed that exposing clothing and bed linen to sunshine reduces the risk of staphylococcal contamination.[157]

Later tests show that unfiltered sunlight kills staphylococci within 70 min of exposure.[158] Partial filtering of the UV-B component of sunlight (280–315 nm) using Perspex marginally reduces this effect. Cells took longer to die when UV-B was blocked in order to allow exposure to UV-A (315–400 nm) and visible radiation. Ordinary window glass absorbs solar radiation at $<$300 nm, which permits entry of solar UV-A and small amounts of UV-B. This would be lethal to staphylococci over time.[158]

Disinfection with artificial light

There are some older studies demonstrating the bactericidal effect of artificial light.[159–161] UV wavelengths inactivate micro-organisms by causing cross-links between constituent nucleic acids. The absorption of UV can result in the formation of intra-strand cyclobutyl-pyrimidine dimers within DNA, which lead to mutations and/or cell death. This lethal effect of UV radiation is primarily due to the structural defects caused when thymine dimers form but secondary damage is also produced by cytosine dimers. There are other types of photoproducts that can contribute towards cell death.[162] High-intensity narrow-spectrum light (HINS-light) is a modern light-based disinfection method that can inactivate a wide range of bacterial pathogens.[163] The HINS-light method utilizes a narrow bandwidth of high-intensity visible violet light with peak output at 405 nm. Inactivation of bacteria by exposure to high-intensity light is thought to be associated with photo-excitation of certain molecules (porphyrins) within the bacteria. This results in the production of highly reactive compounds such as singlet oxygen which are strongly bactericidal.[164] HINS-light has already been assessed for its efficacy in decontaminating the clinical environment but has not yet been linked to correspondingly lower rates of hospital infection.[165]

Sunlight and resistance to infection

Direct sunlight may enhance well-being and resistance to infection for those who receive it; even from behind glass. Bright-light therapy is used to treat a range of psychiatric conditions including seasonal and major depression.[166] Hospital patients in sunlit wards recover better from depressive illness and other conditions, with sunlight having a positive effect on the length of stay, mortality rate, perceived stress and pain.[167–169] Bright light also has benefits beyond relieving the symptoms of depression, since increasing light levels slows down the rate of cognitive decline in dementia patients.[170] Without proper time-cues from the sun, or other sources, underlying body rhythms may become disturbed to cause a range of health problems, including depression, diabetes, obesity, and breast and prostate cancer.[171] Regulating biological rhythms in patients may enhance immunological activity but it is not known whether habitual exposure to sunlight behind glass would help patients withstand infection.[172]

Solar infra-red radiation and infection

Infra-red radiation may also have a health impact on patients indoors. Roman villas and baths used to have a glazed sun-room called a 'heliocaminus' or solar furnace.[173] In Scandinavian countries, saunas have been popular for hundreds of years, with evidence of health benefits.[174] Passive solar design provides building occupants with radiant heat, which acts on the surface of the body as well as deeper-lying tissues. Infra-red radiation encourages wound-healing and relieves pain.[175–177]

Although light treatment in winter may alleviate seasonal depression, summertime usually brings spontaneous remission.[178] It is possible that seasonal variations in the infra-red component of solar radiation may affect mood states. Like the visible spectrum, infra-red may also have an antidepressant effect, which could itself influence resistance to infection.[179] Direct evidence of this in humans is lacking, although infra-red light has been shown to enhance immune response and wound-healing in mice with meticillin-susceptible *Staphylococcus aureus* (MRSA) skin infections.[175,180] In humans, infra-red irradiation inactivates fungal and other pathogens, as well as MRSA in the nasal passages of carriers.[181]

Sunlight through glass

During the 1920s, it was found that children develop rickets if they stay out of the sun.[182] Ordinary window glass filters out the UV radiation that produces vitamin D. A study in 1933 found that exposing rachitic rats to sunlight through window glass improved resistance to infection without alleviating rickets.[183] It is now accepted that vitamin D is involved in the functioning of the immune system but the immunological effect of sunlight through window glass has not been further explored.[184] Sunrooms used to be a standard feature of hospitals.[185] Whereas there is renewed appreciation that patients could benefit from sunlight, solar gains have to be minimized to prevent overheating.[23] This might reduce the need for air-conditioning, but the exclusion of sunlight in hospitals, and indeed, other buildings, increases risk of infection, depression and other health problems. Greater sunlight exposure might encourage faster recovery for patients.[186]

Conclusion

Before the advent of antibiotics, ventilation and natural light were considered to be important safeguards against infection. Nowadays, there is less emphasis on fresh air and light in buildings. Many of the studies in this review were conducted a long time ago, which makes it difficult to assess the evidence in relation to modern hospitals. There is some justification in reviewing current lighting or ventilation arrangements for better protection of patients and staff from airborne contagion.

Despite more than a century of infection study, detailed specifications needed to prevent airborne dispersal of pathogens are still unknown, with ongoing controversy over the importance of airborne spread for certain pathogens.[8,187,188] There is lack of consensus on particle size, transmission characteristics and associated infection risk. Scientists disagree over the number of changes required to dilute airborne pathogens such as SARS or avian influenza, as well as efficacy of natural cross-ventilation systems for control purposes. This means that infection control staff do not know how to manage isolation or droplet precautions for patients with airborne infections. There are also insufficient data to advise on ventilation requirements for non-hospital buildings.[1,114]

Bioterrorism poses a further potential threat to public health indoors.[5] Recent anthrax attacks exposed the vulnerability of building occupants to airborne pathogens and there is a concern that other aerosolized agents could be used for bioterrorism.[6] It is therefore timely to re-examine ventilation requirements for infection control along with those for overall comfort.[8] The same may be true for sunlight and its components, despite unverified science.[189] Examples of older hospitals and other buildings structurally compatible for both comfort and health might offer a starting point.

Nowadays, building codes and regulations specify highly insulated sealed structures to meet government 'zero carbon' targets. As insulation levels have risen, so have the risks of overheating and poor indoor air quality.[190] Typically, the heating in new buildings is provided by warm air, whereas heating in older ones was often from a radiant source.[191] One advantage of radiant heating is that air temperatures can be kept lower than with convective systems. Since radiant sources heat internal surfaces rather than air, comfortable conditions can be maintained at lower air temperatures.[192] The potential for energy savings and the health benefits of radiant heating have been overlooked in recent years.[191] When Florence Nightingale set out to create healthy indoor environments, she insisted on radiant heat in sick rooms because she felt that air heated by metal surfaces was unhealthy. As she put it: 'To shut your patients tight in artificially warmed air is to bake them in a slow oven.'[3] She believed that combining warm-air heating and mechanical ventilation was especially harmful because it prevented or delayed recovery.

Nightingale was so opposed to warm-air heating that she called for it to be removed from hospitals along with mechanical ventilation. Any increase in fuel costs would be offset by the reduction in treatment time for patients.[3] Later, during the 1920s, the British physiologist Sir Leonard Hill reported that the human body requires the stimulus of a constantly changing environment; and that monotonous over-warm indoor conditions are harmful. In common with Nightingale, he recommended sunlight, fresh air and radiant heating.[193] Detailed research on indoor air quality and other health factors in today's energy-efficient buildings is lacking.[194] Present-day buildings may perform better in energy terms than older designs, but the exclusion of sunlight and fresh air could encourage poor health and associated costs.

In conclusion, designing buildings to allow increased exposure to sunlight and outdoor air may discourage survival and spread of infectious agents with consequential health benefits for occupants.[2,3] Most of the evidence in this review comes from the pre-antibiotic era and much of it has since been overlooked. Given the continuing decline in antimicrobial agents, and risk of future pathogens or modified pathogens, potential benefits from sunlight penetration and natural ventilation merit further investigation.

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