Review

Influence of climate change on plant disease infections and epidemics caused by viruses and bacteria

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Abstract

This review is motivated by (i) the magnitude of the threat to world food security and diversity of natural vegetation posed by viral and bacterial pathogens of plants at a time of accelerating climate change; and (ii) the inadequate attention given to this subject by earlier reviews on climate change and plant disease. It starts by providing background information on current climate change predictions, the increasing worldwide importance of viral and bacterial diseases, critical features of their pathosystems and the general influence of environmental factors upon them. It then develops comprehensive climatic and biological frameworks and uses them to determine the likely influences of direct and indirect climate change parameters on the many different host, vector and pathogen parameters that represent the diversity of viral and bacterial pathosystems. This approach proved a powerful way to identify the relevant international research data available and many information gaps where research is needed in the future. The analysis suggested that climate change is likely to modify many critical viral and bacterial epidemic components in different ways, often resulting in epidemic enhancement but sometimes having the opposite effect, depending on the type of pathosystem and circumstances. With vector-borne pathosystems and new encounter scenarios, the complication of having to consider the effects climate change parameters on diverse types of vectors and the emergence of previously unknown pathogens added important additional variables. The increasing difficulties in controlling damaging plant viral and bacterial epidemics predicted to arise from future climate instability warrants considerable research effort to safeguard world food security and biodiversity.

Keywords: Global warming, Temperature, Rainfall, Wind, Greenhouse gasses, Extreme weather events, Viral, bacterial and phytoplasmal pathogens, Vectors, New encounters, Computer simulations, Disease epidemics, Integrated disease management

Review Methodology: We searched the following databases: existing reviews and reports on the subject; textbooks; CAB Abstracts; Web of Science; Google Scholar; Google; and The University of Western Australia electronic journals collection. Search terms used included climate change, global warming, temperature, rainfall patterns, drought, flood, wind, CO_2, epidemiology, virus, bacteria, phytoplasma, vector. We also used references cited by the articles obtained by these methods to search for relevant additional material.

Introduction

Understanding what alterations climate change is likely to cause to the prevalence of diseases of cultivated and wild plants, and the damage they cause in different parts of the world, is of great importance. This is because of the need for food security as the world's population increases at a time when its capacity to increase production in many populous middle- and lower-latitude regions is projected to decline and climate insecurity challenges Man's ability to manage plant diseases effectively. It is also because of the increasing threat plant diseases pose to plant

biodiversity and the likelihood of mass species extinctions arising from the combined influences of climate change and Man's activities (e.g. [1–10]).

Identifying and quantifying future effects of climate change on plant pathogen epidemics constitutes a complex undertaking [11–14]. A number of non-linear relationships influence the interplay between host, pathogen, vector and environment. These depend on the pathosystem, extent of plant damage, cultivation system involved and socio-economic context [15, 16]. Furthermore, the influences of climate change on plant pathogen epidemics are often difficult to unravel from those caused by the current rapid expansion in human activity, such as the impacts of agricultural extensification, intensification and diversification, global population increase, increasing volume and speed of trade in plants and plant products, and increasing fragmentation of natural vegetation [4, 17, 18].

The anticipated influences of individual parameters associated with climate change on diverse aspects of fungal pathogens and the diseases they cause have been investigated in depth in different types of cultivated plants. The aspects covered include their life cycles, inoculum levels, interactions with hosts, expression of host resistances and epidemiologies. Many reviews have been written on this subject [3, 5, 11, 12, 19–42]. In some of these reviews, brief mention was also made of the influences of climate change parameters on diseases caused by plant viral or bacterial pathogens (e.g. [3, 38, 42]), and one covered North American bacterial pathogens to a somewhat greater extent [20]. Only two focused solely on diseases caused by viruses [4, 43]. However, neither was comprehensive as they did not include many aspects of viral pathosystems potentially vulnerable to climate change. Also, only one review focused solely on diseases caused by bacteria, but this concerned central Europe alone and did not include phloem-limited vector-borne bacteria or mollicutes [44]. This review therefore provides comprehensive coverage of currently available international research data that illustrate the influences of climate change on infections and epidemics caused by viruses and bacteria in cultivated plants or native plant communities in different regions of the world. Changes in management tactics required to manage these effects are also emphasized. Mention of fungal pathogens is limited to a few examples that illustrate climate change scenarios not yet illustrated by the other two types of pathogen.

Current climate change predictions

The world is now experiencing one of its regular climate change cycles [3, 45–48]. However, this cycle is occurring more rapidly than earlier ones as the rate of temperature change is accelerated by human activities, especially those arising from greenhouse gas emissions, such as carbon dioxide (CO_2), methane and nitrous oxide [49–51]. The greenhouse effect is the process by which absorption and emission of infrared radiation by gases in the atmosphere warms the planet's lower surface. Evidence for global warming includes observed increases in global average air and ocean temperatures, widespread melting of snow and ice, and rising global sea levels. The amount of CO_2 in the atmosphere has been increasing over the last 50 years (1956–2006) at $0.25 \pm 0.21\%$/year [52], and may double during the current century [53, 54].

The most recent global climate change predictions are summarized in the 2007 Fourth Assessment Reports of the Intergovernmental Panel on Climate Change [55–58]. During the 21st century, the planets' surface temperature is likely to rise by 2.1–2.9 or 2.4–6.4°C for lowest and highest emissions scenarios, respectively. These varying estimates arise from models with differing sensitivities to greenhouse gas concentrations. Warming is expected to vary across regions around the planet (Figure 1) with most occurring at high latitudes (e.g. [59]). Increased global temperature is expected to cause sea levels to rise further, change the amount and pattern of rainfall precipitation and cause expansion of subtropical deserts. Increases in the amount of precipitation are projected for high latitudes, but decreases in precipitation in most subtropical land regions (by as much as about 20% in 2100). More frequent extreme weather events including strong winds, heatwaves, droughts and episodes of torrential rain are predicted. Future tropical cyclones are expected to become more intense, with larger peak wind speeds and more heavy precipitation associated with ongoing increases in tropical sea-surface temperatures. Extra-tropical storm tracks are projected to move poleward, with consequent changes in wind, precipitation and temperature patterns [56, 58].

Over the course of this century, net carbon uptake by terrestrial ecosystems is likely to peak before mid-century and then weaken or even reverse, thus amplifying climate change. The resilience of many ecosystems is likely to be exceeded by an unprecedented combination of climate change, associated disturbances (e.g. flooding, drought, wildfire, insect pests, plant disease, ocean acidification) and other global change drivers (e.g. land use change, pollution, fragmentation of natural systems and over-exploitation of resources). Approximately 20–30% of plant species are at increased risk of extinction if increases in global average temperature exceed 1.5–2.5°C. Major changes in ecosystem structure and function, species' ecological interactions and shifts in species' geographical ranges are projected, with predominantly negative consequences for biodiversity [57, 58, 60, 61]. Natural ecosystems and biodiversity are expected to come under increasing stress from pathogen disease epidemics in all latitudes owing to the combination of climate change and increased human activities [1].

The temporal and spatial shifts in climate arising from climate change are likely to influence world food production considerably. It is projected to increase slightly at high latitudes for local mean temperature increases of up

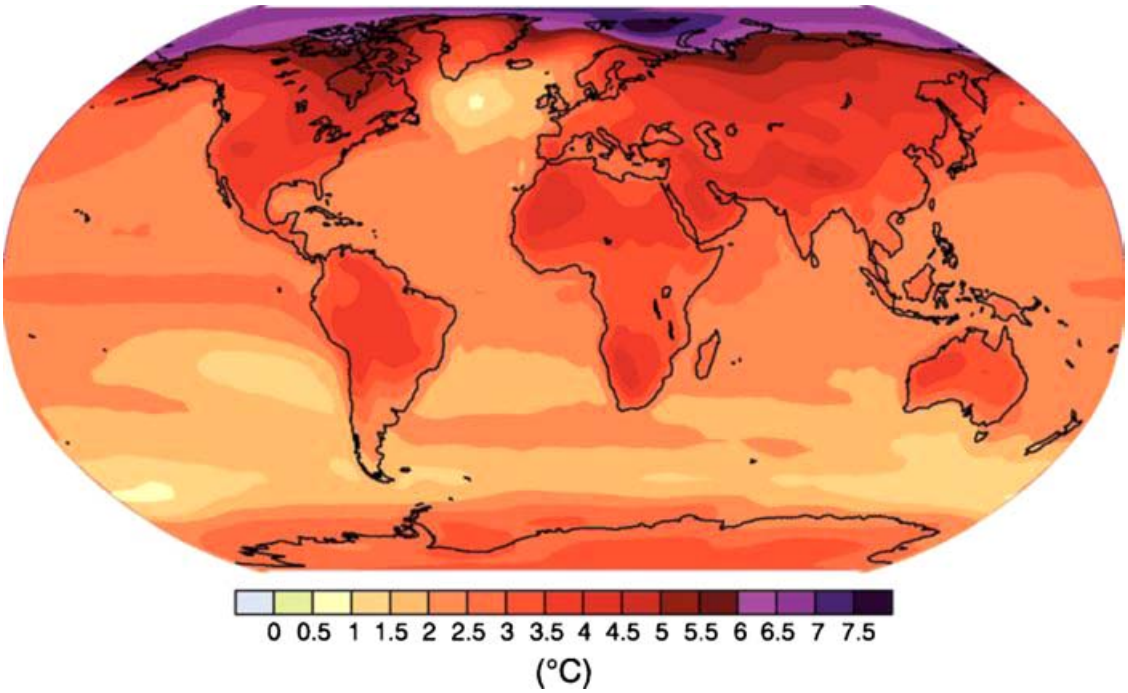

Figure 1 World map showing the projected geographical pattern of surface warming for the world in the late 21st century (2090–2099). Temperatures are relative to the period 1980–1999. From the Fourth Assessment Report of the Intergovernmental Panel on Climate change (Pachauri and Reisinger [56]).

to 1–3°C depending on the crop, and then decrease beyond that in some regions. At other latitudes, especially in seasonally dry and tropical regions, crop productivity is projected to decrease from even small local temperature increases (1–2°C), which would increase the risk of hunger. Temperate higher-latitude regions in Canada, Northeast Asia, southern South America, and northern USA, Europe and Russia, as well as cool mountainous regions in otherwise warmer areas, are potential beneficiaries. In contrast, higher temperatures and less rainfall are projected to cause adverse effects on cultivated ecosystems in middle-latitude regions, including Southern Australia, semi-arid and subtropical parts of Asia, India, Southern and Northern Africa, Southern Europe, Northeast Brazil and the Central American lowlands. Ranges of crops and the areas able to be cropped productively should increase in regions with temperate climates, but decline in arid or semi-arid climatic zones, which are likely to suffer frequent droughts. Predictions for food insecure areas are for increasingly serious shifts towards less food production, especially after 2050 [8, 48, 55, 62]. In developing countries, overall agricultural production may decline by 10–25% by the 2080s [2]. These climatic risks to food security are coincident with rapidly expanding human populations and the diversion of large tracts of land for biofuel production (e.g. [4, 63]).

Elevated CO_2 increases the growth and yield of most crop species [64–66], and this increase is mainly caused by increased rates of photosynthesis and/or increased water use efficiency [67–69]. However, the potential for greater crop yields arising from increasing ambient CO_2 levels and temperature in temperate higher-latitude regions, and cool mountainous regions in otherwise warmer areas, is unlikely to be realized fully. This is because the impact of CO_2 fertilization may be limited by deficiencies in other nutrients, increased respiratory C demand associated with higher temperatures, diminished water supply or the increased likelihood of pathogen disease epidemics and pest outbreaks.

Worldwide importance of viral and bacterial diseases

The effects of viral diseases in causing losses in yield of cultivated plants and quality of plant produce, and the economic and social consequences of this are well documented (e.g. [7, 17, 70–88]). These losses occur worldwide and range from minor effects to complete failure and devastation. They occur in all types of cultivated plants, including world human food staples, such as cereals, grain legumes, root and tuber crops, vegetables and fruits, and also occur in ornamentals, fibre crops and medicinal plants (e.g. [4, 70, 83–87, 89]). They are particularly severe in the tropics and subtropics (e.g. [83, 85, 86, 89]) and in protected or organic cropping systems (e.g. [77, 90]). They also damage wild plants and threaten native plant biodiversity (e.g. [4, 91–93]). Virus-infected plants are often stunted or deformed with localized or extensive chlorotic areas. Mosaic or ringspot symptoms

often develop on leaves or fruits, and a diverse range of other symptoms often appear (e.g. [95]). In their review 'Plant disease: a threat to global food security' Strange and Scott [96] highlighted the debilitating effect caused by *Barley yellow dwarf virus* (BYDV) infection worldwide on the staple cereal crops wheat, barley, oats, rice and maize, and the devastating pandemic of *Cassava mosaic virus* (CaMV) in East and Central Africa, which forced many farmers to stop growing their staple food source, cassava. A recent example from tropical and subtropical Australia is the disease of cucurbit crops caused by *Zucchini yellow mosaic virus*, which is forcing many farmers to stop growing cucurbits [72, 97]. Recent Symposia of the International Plant Virus Epidemiology Group of the International Society for Plant Pathology have highlighted many other virus diseases threatening global food security, including those caused by *Rice yellow mottle virus* (RYMV) in rice, *Maize streak virus* in maize, *Cocoa swollen shoot virus* (CSV) in cocoa, *Groundnut rosette virus* in peanut, and *Banana bunchy top virus* in banana in sub Saharan Africa; *Faba bean necrotic yellows virus* and *Bean leaf roll virus* in grain legumes in West and Central Asia, and North Africa; rice tungro viruses in South and Southeast Asia; *Tomato yellow leaf curl virus* in tomato in most continents; potyviruses of cucurbit crops and soil-borne viruses of cereals in Europe; potyviruses in diverse food crops in Australasia; and begomoviruses and tospoviruses in diverse tropical and subtropical food crops in most continents (e.g. [4, 78, 85, 86, 89, 90, 98–105]). *Poytviruses*, *Tospoviruses* and *Begomoviruses* are the names of three large groups of plant viruses that differ in various ways, including particle shape and size and type of vector, and contain many economically important well established or emerging viruses (e.g. [4, 72, 86, 88, 90, 97, 99, 100, 103, 104, 106, 107]). Moreover, viruses appear to cause a greater proportion of emerging infectious diseases of plants than fungi, bacteria or nematodes put together, and Anderson *et al.* [17] calculated that viruses cause nearly half (47%) of such diseases. In addition to being used to describe newly recognized or newly evolved pathogens that cause damaging epidemics, the term emerging is often applied broadly to pathogens that have increased their host ranges or geographical distributions, or changed their pathogenesis [4, 17, 88, 106].

Losses from virus epidemics in annual and perennial grass and legume forage crops and pasture production result from decreased nitrogen fixation, and herbage and seed production. The competitive ability of infected forage or pasture plants is also diminished such that stands deteriorate over time, leading to unproductive predominantly weedy forage crops and pastures (e.g. [74, 75, 77, 81, 108–110]). Moreover, production of undesirable chemicals, such as oestrogens, can be stimulated by virus infection of pasture plants [111]. Viruses also cause considerable losses in fibre production, e.g. *Cotton leaf curl virus* causes a devastating disease of cotton in the Indian sub-continent [85, 88].

Financial losses attributed to plant virus disease include the following examples: CSV is estimated to cause an annual loss of 50 000 tons of cocoa beans in Africa with an estimated value of $28 million. In Southeast Asia, infection of rice with rice tungro viruses leads to an estimated annual economic loss of $1.5 billion annually [112]. The estimated annual losses worldwide associated with *Tomato spotted wilt virus* (TSWV) infection of a wide variety of plants including tomato, peanuts and tobacco were estimated to be $1 billion [112]. In 1996, diminished feed for dairy cattle caused by widespread infection with *Subterranean clover mottle virus* and *Bean yellow mosaic virus* (BYMV) in annual clover pasture was estimated to cause economic losses of $31 million per year to the Australian dairy industry [75]. Moreover, these two viruses cause greater annual losses to the much larger Australian wool and meat industries [74, 75, 81, 109].

The impacts of introduced viruses on native plants that have not encountered them before can be severe [4, 91]. Virus epidemics in native vegetation are influenced by its fragmentation into small pockets surrounded by crops or urban areas, disturbance by grazing animals and human activities such as woodcutting or flower collection. This is particularly noticeable when introduced viruses invade, e.g. when BYMV infects *Kennedia prostrata* (scarlet runner) at the margins of bushland or growing in disturbed bushland remnants in south west Australia [4, 92]. However, when undisturbed communities of native wild plants are infected by indigenous viruses that co-evolved with them, viruses are generally considered not to be overtly damaging to such hosts. This is because natural control measures operate to limit virus epidemics in native plant communities (e.g. [4, 113]).

Plant-pathogenic bacteria are most destructive in the tropics, but under warm or moist climatic conditions can be destructive anywhere. They affect all kinds of plants, often causing major losses in yield and quality of plant products. They are also very important causes of post-harvest damage to plant produce. The symptoms they cause include rots, leaf spotting and yellowing, galls, cankers, and plant wilting or stunting (e.g. [20, 114–116]). In their review of plant diseases threatening global food security, Strange and Scott [96] highlighted bacterial blight in rice in tropical Asia caused by *Xanthomonas oryzae* pv. *oryzae,* which severely constrains rice production in countries such as India; a severe wilt disease of banana in East Africa induced by *Xanthomonas campestris* pv. *musacearum*, which also causes plant dieback and fruit rotting; and the devastating disease bacterial wilt of potato, banana and a wide range of other crops in many warmer countries caused by *Ralstonia solanacearum*. In their textbook 'Essential Plant Pathology', Schumann and D'Arcy [115] highlighted six well-known, destructive and economically important plant bacterial diseases among their 'Disease Classics'. These were bacterial spot of pepper and tomato caused by *X. campestris* pv. *vesicatoria*, citrus canker caused by *Xanthomonas axonopodis* pv. *citri*, crown

gall of pome and stone fruits, vines and canes, nuts and ornamentals caused by *Agrobacterium tumefaciens*, fire blight of apple and pear caused by *Erwinia amylovora*, soft rot of potato, vegetables, fleshy fruit and ornamentals caused by *Erwinia carotovora*, and Stewarts wilt of maize caused by *Erwinia stewartii.* Schaad [117] emphasized the emergence of three heat-loving plant pathogenic bacteria now causing serious problems worldwide, *Acidovorax avenae* subsp. *avenae*, *Burkholderia glumae* and *R. solanacearum.*

Critical features of plant pathogenic viral and bacterial pathosystems

A virus is an infectious, sub-microscopic, intracellular agent, composed minimally of nucleic acid (RNA or DNA) and protein, which directs its own replication in living host cells [115]. All viruses are obligate parasites that cannot reproduce on their own but replicate using host cell components, such as amino acids, nucleotides and ribosomes for this purpose. Most plant viruses have a nucleic acid genome and a protective protein coat. Outside the host, they form particles that have different shapes and sizes, including spheres, rigid rods, and flexuous filaments. Other shapes include baciliform or bipartite particles. These shapes and sizes help distinguish the different virus groups. Each virus consists of variants called strains that arise mainly through mutation or recombination.

Viruses have no survival structures comparable to fungal spores when outside a living cell; so for a virus to spread within a plant population there must be transmission both from plant-to-plant and from one generation to the next. Plant-to-plant spread occurs through the activity of specific insect, mite, nematode or fungal vectors, or contact transmission where mechanical damage occurs. They enter the cells of healthy plants through tiny wounds in their cell walls created by vector activity or damage caused in other ways. Spread from one generation to the next does not always require wounding. It can occur through sowing infected seed as about 10% of plant viruses are seed-borne [118], in pollen or by vegetative propagation from infected planting material, such as cuttings, root stocks, tubers, bulbs and corms. It can also occur in infected volunteer crop plants and weeds that persist between growing seasons (e.g. [95]). Viruses can be generalists that have broad host ranges but are often poorly adapted to individual hosts, or specialists that have narrow host ranges but are well adapted to the hosts they infect. Generalists are better adapted than specialists to alterations in host range under climate change scenarios (e.g. [4, 113]). This parallels wild plants where generalist, fast-growing species, such as weeds, are best adapted to survive climate change (e.g. 119, 120). Viruses have different temperature optima for multiplication within their host plants, some being adapted to warmer regions and others to cooler ones. Such optima are highest in viruses adapted to invade hosts growing in lowland tropical environments, such as CaMV [104], and lowest in viruses adapted to cold climates such as those in cool temperate zones or at high altitude in mountainous regions in the tropics, such as *Potato mop-top virus* (PMTV) [121–123].

Insects are the most important virus vectors, and the most important types of insect vectors are aphids, whiteflies and thrips, but other insects, such as leafhoppers, plant hoppers, mealybugs and beetles, also transmit some viruses. Aphid-borne viruses (e.g. potyviruses) are the most widespread and damaging viruses of cultivated plants in temperate regions, but whitefly-borne viruses (e.g. begomoviruses) and thrips-borne viruses (e.g. tospoviruses) are often the most important in regions with tropical and subtropical climates. Aphid, whitefly and thrips vector are all important in protected cropping. Some viruses multiply within their insect vectors in addition to multiplying in their plant hosts. Infection with some viruses may alter host physiology or morphology, making infected plants more attractive to insect vectors. Some economically important viruses are transmitted by tiny mites that are wingless and wind-blown (e.g. [95, 112]). Soil-borne viruses are transmitted from roots of infected plants to healthy plant roots by ectoparasitic nematodes or fungus-like organisms (oomycetes and protists) that are obligate root parasites. Soils infested with virus-infected resting spores can be dispersed by flooding or human activities, and some viruses are waterborne, being spread over long distances by flooding or in irrigation and drainage channels (e.g. [124–126]).

Contact-transmitted viruses are generally ones that reach very high concentrations in the plant and have particles that are relatively stable outside the infected cell. They do not require a vector for spread as they infect readily through wounds caused when leaves of infected plants rub against leaves of healthy ones, machinery or large animals move through partially infected crops, or via grazing and trampling by stock or mowing in pastures (e.g. [77, 95, 115, 127, 128]).

As mentioned above, most economically important emerging viruses are members of the poytviruses, tospoviruses and begomoviruses. These types of viruses adapt rapidly to changing conditions, and a complex chain of events gives rise to the novel virus–vector–plant–environment interactions that lead to emergence. The evolutionary process involved is influenced at the macro level by selection pressures arising from major agricultural changes that cause significant alterations in the biology of viruses and vectors. At the micro level, alterations to virus genomes result when this selection pressure drives molecular alterations in viral nucleic acid. Although plant virus evolution rates increase generally when viruses are exposed to a strong selective pressure, members of these three virus groups are particularly responsive to such selection pressures (e.g. [4, 17, 89, 90, 106, 107, 129–131]).

Mixed infections with more than one virus often cause more severe disease symptoms than either virus alone.

This occurs, for example, when *Potato virus X* (PVX) infects potato plants in combination with *Potato virus* A causing 'crinkle' disease, *Potato virus Y* (PVY) causing 'rugose mosaic' disease or *Potato virus S* causing obvious mosaic and rugosity (e.g. [9, 95, 132]), and in co-infection of tomato plants with PVX and *Tobacco mosaic virus* (TMV), which causes a severe 'streak' disease [95]. In some cases, the synergistic effects between two viruses can be extreme, e.g. with the devastating sweet potato virus disease commonly caused relating mixed infection with *Sweet potato feathery mottle virus* and *Sweet potato chlorotic stunt virus* both of which cause relatively mild symptoms when present alone (e.g. [133]). When viral and fungal pathogens occur together in the same plants, increased susceptibility to fungal disease often results. However, presence of the fungal pathogen can either increase or decrease susceptibility to viruses [95].

Bacteria are tiny, single-celled, prokaryotic organisms that reproduce by binary fission and evolve mainly through mutation. Most plant-pathogenic bacteria have cell walls and are bacilliform, but mollicutes (phytoplasma are spiroplasma) lack cell walls; phytoplasma are variably shaped and spiroplasma are partially helical. Most bacteria are facultative parasites that adapt readily to different environments and are relatively easy to culture. However, fastidious bacteria are ones that invade the xylem or phloem and have never been cultured successfully or can only be cultured on complex, specialized media. Fastidious bacteria have no life-cycle stage that is separate from the plant host or insect vector, and depend entirely on vectors for plant-to-plant spread. Non-fastidious bacteria invade plants through natural openings, such as stomata, lenticels or hydathodes, or *via* wounds. Some are also transmitted non-specifically when sticky bacteria contaminate an insect and sites of insect feeding or egg laying provide wounds for bacterial infection. Non-fastidious bacteria live and move in the intercellular spaces, but fastidious bacteria live inside dead xylem cells or living phloem cells. Fastidious plant-pathogenic bacteria that invade phloem cells include phytoplasma, spiroplasma and some walled bacteria (e.g. *Liberibacter*). Non-fastidious bacterial infections cause many of the same kinds of symptoms in plants that fungi cause, including leaf spots and blights, cankers, galls, leaf spots and rots. Fastidious bacteria that invade the xylem (e.g. *Xylella fastidiosa*) commonly cause vascular wilts, but infected plants may also be yellowed and stunted. Fastidious bacteria that invade the phloem often cause yellowing, stunting, phyllody or witches' broom symptoms. Since they are transmitted by insect vectors and frequently induce 'virus-like symptoms' (yellowing and stunting) in plants, they were originally thought to be caused by viruses. Fastidious bacteria are transmitted mainly by insects with piercing-sucking mouthparts, and many (e.g. phytoplasma and spiroplasma) multiply in their vectors [114, 115].

Most plant-pathogenic bacteria do not produce survival structures like fungal spores. Therefore, when not protected by an insect vector or host plant, their ability to survive adverse environmental conditions, especially desiccation and ultraviolet radiation, is limited. However, a few non-fastidious bacteria can survive as epiphytes on plant surfaces or as saprophytes in the soil, which decreases their vulnerability to environmental stresses. Under adverse environmental conditions, persistence of plant-pathogenic bacteria that depend entirely on their vectors for plant-to-plant spread relies entirely on survival of their hosts and vectors. Non-fastidious plant-pathogenic bacteria are spread to their hosts mainly by water, usually in the form of rain splash. In humid, wet conditions abundant moisture increases their multiplication in infected tissues, inducing them to exude and ooze masses of bacteria. This promotes their spread. Heat-loving non-fastidious plant pathogenic bacteria have optimum growth temperatures of 32–36°C, but others grow better at lower temperatures. Several bacterial plant pathogens have strong pectolytic activities that enable them to cause economically important soft rots in plant tissues of a wide range of plant species both in the field and after harvest during transit or storage, e.g. *Bacillus spp*. and *Erwinia chrysanthemi* in warm climates, and *E. carotovora* subsp. *atroseptica* and *Clostridium* in temperate climates [134]. Cold loving plant pathogenic pseudomonads have minimum growth temperatures of 4–5°C, while more themophylic xanthomonads have minimum growth temperatures of 7–9°C [44, 135]. Ice-nucleation-active pseudomonads serve as nuclei for ice formation and therefore cause frost injury to plants when temperatures fall below freezing [136]. Non-fastidious bacteria multiply very rapidly and their significance as plant pathogens stems primarily from the fact that they can produce tremendous numbers of cells in a short space of time [114].

Plants of otherwise susceptible species resist infection with viruses or bacteria in different ways. Single resistance genes confer extreme resistance (immunity) or hypersensitive resistance (HR) to infection. Extreme resistance genes are generally effective against all variants (or strains) of a virus or bacterium, but HR genes normally operate against some variants but not others. HR genes usually induce a local necrotic response, but in some cases a virus then moves systemically, killing the whole plant, thereby removing it as a source of infection for spread to healthy plants. Single resistance genes can be dominant or recessive, and there is a gene-for-gene interaction between them and an elicitor gene in the pathogen. HR is sometimes temperature sensitive (e.g. [137, 138]). There are single gene resistances to some important insect vectors, e.g. to *Aphis gossypii* (cotton aphid), *Aphis glycines* (soyabean aphid) and *Amphorophora idaei* (large raspberry aphid) [139–141]. There is also polygenic resistance to pathogens, which is effective to varying degrees and expressed in different ways, such as resistance-to-vector transmission or seed transmission, or resistance-to-systemic movement of the pathogen in the plant (e.g. [142–144]). Mature plants can express 'mature plant

resistance' which inhibits systemic movement following late infections (e.g. [145]). Systemic acquired resistance (SAR) eliciting production of pathogenesis related proteins occurs following infection with a viral or bacterial pathogen. A chemical signal transported through the plant induces SAR to develop in new growth not yet exposed to infection, providing partial resistance to infection [115].

Plants also defend themselves against infection through RNAi-based post-transcriptional gene silencing, which results in the mRNA of a particular host gene being destroyed or blocked. Induced gene silencing can spread systemically throughout the plant, and be transferred from stock to scion plants via grafting. It is an important component of the plants resistance response that allows it to react after an initial localized pathogen encounter [146, 147]. These effects may be part of a generalized response to pathogens that down-regulates any metabolic processes in the host that aid the infection process. However, plants such as *Arabidopsis thaliana* express multiple dicer homologues that are specialized so as to react differently when the plant is exposed to different types of viruses. Also, some plant genomes express endogenous siRNAs in response to infection by specific types of bacteria [148–150]. Plants also have a variety of passive and biochemical defences against bacterial pathogens and insect vectors, such as a waxy cuticle, bark, bud scales, modified stomatal shape, dense layers of hairs, suberized cells, tylose formation, lignification of woody tissues, thickening of vascular bundle walls and production of secondary metabolites (e.g. caffeine, nicotine and alkaloids). Some, e.g. waxy cuticle, bark formation, bud scales and dense layers of hairs, also help defend plants against infection by contact transmitted viruses. Some of these defences can also be actively induced by bacterial infection or insect vector feeding, e.g. thickening or lignification of cell walls, and production of gums and resins, cork and abscission layers, papillae of callose and tyloses. Stress factors, such as increased temperature, can limit a plants' ability defend itself by responding in such ways. Plants can also be 'genetically engineered' for virus resistance [114, 115].

General features of environmental influences on pathosystems

Climate change can influence the environment on micro to macro scales that range from microclimate to local, regional, subcontinental, continental and global. Its effects on disease epidemics also need to be considered at all these levels [3]. The classic plant disease epidemic triangle includes interactions between plant hosts, pathogens and environment (e.g. [114, 115]). Each interacts with the other, macro- and micro-environments influencing both hosts and pathogens. At microclimate and, sometimes, local scales, the environment is influenced by hosts and by pathogens through their effects on growth of their hosts (Figure 2). However, with vector-borne pathogens, a

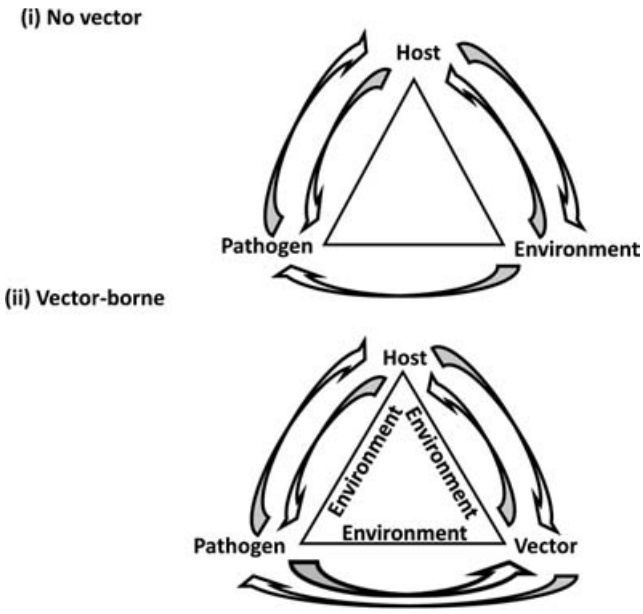

Figure 2 Disease triangles for non-vector and vector-borne pathosystem scenarios. Arrows represent interactions occurring between the different triangle components.

vector must also be included making a disease triangle with the pathogen, the host and the vector in each of its corners, each interacting separately with the environment [83, 151, 152]. This complicates things further because of additional interactions between environment and vectors, and between vectors and both pathogens and hosts. Thus, vector feeding can alter plant growth, thereby altering microclimate. Also, infection with a pathogen can modify host physiology and appearance making it more attractive to a vector, or a virus or mollicute may multiply within a vector resulting in altered vector behaviour (e.g. [4, 43, 95, 112, 153]).

When considering possible effects of alterations in climate on viral and bacterial epidemics, detailed epidemiological knowledge is required for each pathosystem including about the nature of the primary infection source, how the pathogen spreads into and within a plant stand, how it spreads over distance to invade new sites and how it survives outside the main growing period. Then a clear picture needs to be constructed of the factors driving epidemics of the pathogen concerned, i.e. which are the key ones favouring spread and which delay its epidemics. For an insect or mite vector, knowledge of its life cycle is needed, including whether the vector is exposed or concealed, the type of feeding of young and adults, time spent feeding, dispersing and sheltering, periods of activity and quiescence and mode of persistence. Knowledge is also needed of its host range, behaviour and the influence of environmental factors upon population density. For a nematode vector, the knowledge required includes its periods of activity and quiescence, type of feeding, time spent feeding, method of surviving in the absence of the cultivated plants, host range, the effect of soil

structure and other environmental factors on populations and mobility in soil and means of dispersal. Fungal vectors produce zoospores that swim from root to root in soil moisture films. For them, the information needed includes knowledge of the impacts of soil moisture and temperature on zoospore activity, periods of zoospore activity, capacity of resting spores to survive in the absence of host plants, the host range, and how it disperses between locations. Information required for a cultivated plant involved includes type, time of sowing, planting time, spacing between plants, rate of growth, critical growth stages, time to maturity, tolerance of herbicides, fertilizer requirements and the impacts of wind, rainfall and temperature upon growth (e.g. [4, 76, 77, 95, 112, 154]).

Modelling and forecasting together provide a very important avenue for highlighting the roles of climate change factors on pathogen epidemics (e.g. [22]). The critical environmental parameters (temperature, rainfall, atmospheric influences, etc.) driving pathogen and vector behaviour and disease epidemic development can be evaluated independently, leading to weather-based predictive models and decision support systems for viral and bacterial epidemics [15, 154]. A literature search found 21 published papers on modelling virus epidemics that used weather data in their predictions [154]. The vectors involved were aphids, whiteflies, thrips, leaf hoppers, plant hoppers, mealybugs and a protist. Several models assessed the likelihood that virus epidemics might occur in previously unaffected regions on three continents if viruses or vectors became introduced or climate change caused their distributions to alter [155–160]. However, more such modelling studies involving many other pathosystems are needed to establish the likely effects of climate change on epidemics caused by viruses or bacteria.

Acting simultaneously or sequentially, plants encounter multiple stresses from combined abiotic (e.g. drought, flooding, hail, extreme heat or cold, and high winds) or biotic factors (e.g. infection with viral or bacterial pathogens or fungal vectors, and feeding by insect, mite or nematode vectors) that result in decreased growth and internal reallocation of resources. The plant stress hypothesis predicts that environmental stresses on plants decrease plant resistance to pests or diseases by altering biochemical source–sink relationships and foliar chemistry. These changes in the nutritional landscape for insects can facilitate insect vector population outbreaks during periods of moderate stress on host plants (e.g. [161, 162]). Such increases in insect vector numbers would, in turn, increase spread of viruses or bacteria that they transmit. Many pathogen epidemics represent scenarios where multiple stresses occur including epidemics caused by more than one pathogen, but most climate change experiments involve single parameters, such as temperature, CO_2 and single pathogens. Therefore, such experiments do not address climate change scenarios where several parameters change simultaneously and continuously so limiting their usefulness in making predictions.

Review Framework

Plant hosts, vectors and pathogens are influenced by (i) the direct consequences of climate change, especially altered rainfall patterns, increased temperature and greenhouse gasses, drought and greater wind speeds; and (ii) indirectly by things like regional alterations in areas cropped, ranges of crops grown, cultivation systems, and distribution and abundance of vectors, and reservoir and weed alternative hosts. In turn, these factors influence pathogen geographic ranges and relative abundance, their rates of spread, the effectiveness of host resistances, the physiology of host–virus interactions, the rate of virus evolution and host adaptation and the effectiveness of control measures [4]. Therefore, the inherent challenge with this review is that the scope is very wide, involving all possible climate change effects, and all the diversity in behaviour and life cycles of viral and bacterial pathogens and their vectors. We developed two frameworks to enable this to be done (Table 1). The first framework defined what is meant by the direct and indirect effects of climate change. We excluded ozone (O_3) from this because its role in plants is more important as a pollutant than a greenhouse gas [38]. The second framework defined the important biological processes for host, vector and pathogen regarding sensitivity to climate. We then put these two frameworks together to create a logical structure for the material in the review. This ensured that all aspects of climate change were cross-checked against all relevant aspects of viral and bacterial pathogen, host and vector biology across the breadth of the topic. The first framework was then used to structure the following section.

Direct climate effects

Mean temperature

In general, heating plants at somewhat higher temperatures than normal increases their susceptibility to virus inoculation with infective sap [95, 163]. Increased susceptibility arising from elevated mean temperature may therefore increase the incidence of contact-transmitted viruses, such as *White clover mosaic virus* in white clover pasture (e.g. [127, 128]). Over the range of temperatures at which different kinds of plants are normally grown, increasing the temperature of already infected plants usually increases the rate of virus multiplication and systemic movement within them. However, increase above a certain level has the opposite effect [95], e.g. *Tobacco rattle virus* multiplied most rapidly in tobacco at 18–22°C, but at 26°C there was a marked decline in virus concentration after a peak was reached [164]. Faster rates of virus multiplication and systemic movement within plants usually decrease the delay before first appearance of systemic disease symptoms. Also, there is often a correlation between severity of disease symptoms and

Table 1 Frameworks used to analyse possible influences of climate change parameters at microclimate to regional climate levels on biological parameters for epidemics of viral and bacterial plant pathogens

Climate change parameters	Biological parameters for vector and host	Biological parameters for pathogen
(a) *Direct*	Changes in vector distribution	Ability to survive extremes weather events within plant hosts
Mean temperature	Changes in vector abundance	Ability to survive desiccation and ultraviolet light outside hosts
Maximum mean temperature (including heatwaves)	Changes in vector activity	Ability to multiply as epiphytes or saprophytes
Minimum mean temperature (including freezing)	Methods of vector survival between growing periods	Ability to persist and spread outside host or vector cells in soil or water
Mean rainfall and altered rainfall patterns	Vector ability to survive extremes of temperature	Entry via wounds and spread by rain splash
Extreme rainfall-related events (including monsoonal rain, hail, flooding and drought)	Vector ability to survive extreme rainfall-related events	Air-borne vector transmission
Relative humidity (including leaf microclimates)	Vector ability to survive extreme high winds	Soil-borne vector transmission
Wind speed and direction	Influence of increased greenhouse gasses on vectors	Contact transmission
Greenhouse gas concentration	Vector infestation of alternative cultivated or weed reservoir hosts	Transmission by seed, pollen or vegetative propagation
General climate instability	Alterations to host physiology influencing attractiveness to vectors	Importance of alternative cultivated plant or weed reservoir hosts
(b) *Indirect*	Alterations to host physiology affecting efficiency of vector transmission	Ability to persist and multiply inside vectors
Altered ranges of cultivated plants grown	Alterations to plant morphology influencing attractiveness to vectors	Ability to multiply and spread within plant hosts
Alterations in regional areas cultivated	Alterations to plant morphology influencing direct pathogen infection	Changes in rates of systemic movement within plant hosts
Alterations in alternative cultivated or weed reservoir hosts	Alterations to host or vector phenology	Ability to evolve rapidly and invade new hosts
Changes in cultivation systems	Alterations in vector activity due to presence of another vector	Generalist or specialist pathogen
	Alterations to temperature sensitivity of host resistance to vector or pathogen	Alterations in multiplication and symptom expression in single or mixed host infections
	Alterations in effectiveness of chemical control measures against vectors	Alterations in effectiveness of chemical control measures against bacteria
	Alterations in effectiveness of cultural control measures against vectors	Alterations in effectiveness of cultural control measures
	Alterations in effectiveness of biological control measures against vectors	Alterations in effectiveness of biological control measures

virus concentration reached in plants, as with TMV and PVX at the range 16–28°C in tobacco [165, 166]. However, this does not always occur, e.g. with TSWV in tomato in which most severe symptoms developed at 36°C, despite maximum virus concentration developing at lower temperatures [167, 168]. Thus, somewhat increased mean temperatures, causing faster rates of virus multiplication and systemic movement, would be expected to increase rate of symptom appearance and severity of virus disease with many, but not all, virus–plant pathosystems.

Increased temperature can influence the behaviour of viruses present in mixed infection. For example, when barley plants were inoculated with a mixture of *Brome mosaic virus* and TMV and held at 20°, 25° or 31°, systemic infection with TMV only occurred at 31°C [169]. In mixed infection of tobacco plants with PVX and PVY, the increase in PVX accumulation caused by PVY presence was greatest at 30°C [170]. When PVX was present in mixed infection with several other viruses, including TMV and CMV, it was able to move systemically at 31°C but not at lower temperatures [171].

As the mean temperature increases, generalist viruses with high-temperature optima for multiplication within their host plants that are adapted to warmer regions and infect many different host species are likely to expand their geographical ranges from the areas with tropical or subtropical climates they currently occupy. This expansion would be to areas of higher latitude that were previously to cool for them and to formerly cooler higher elevations in mountainous regions within the tropics or subtropics [4]. For vector-borne viruses such expansion would be limited if the ranges of their vectors were to remain unchanged, but the opposite scenario is predicted for key tropical vectors of such viruses such as whiteflies and thrips (discussed below). Conversely, the geographical distributions of viruses with lower-temperature optima for multiplication within their host plants are likely to contract to areas of higher latitude or higher elevations in mountainous regions within the tropics and

subtropics. Again, for vector-borne viruses such expansion would tend to be limited if the ranges of their vectors were to remain unchanged, but the opposite scenario is predicted for key temperate region vectors such as aphids (discussed below). Of course, such predictions exclude protected cropping situations where plants are grown in temperature-controlled environments that include heating in cold climates and cooling in warm climates.

Changes in mean temperatures can alter the scale of virus epidemics by modifying selection pressures and virus evolution rates. Warmer temperatures can increase virus evolution rates, leading to more virulent strains with broader natural host ranges, higher virus multiplication rates in reservoir hosts and increased vector transmission efficiencies (e.g. [172]). For example, when tobacco plants infected with CMV were held at 25°C or 33°C, the higher temperature enhanced spontaneous mutations in the coat protein gene of the virus and this change altered virulence in regard to systemic movement (e.g. [173]). Using a fungal example, it is worth emphasizing here that being subject to a combination of increased temperature and another environmental stress can even alter the virulence of otherwise harmless pathogens that colonize plants without causing symptoms, converting them into damaging pathogens. For example, a combination of increased temperature and light exposure led to melanin production by the fungal endophyte *Diplodia mutila*, converting a harmless endophyte into a devastating pathogen [174].

Different types of bacterial plant pathogens are likely to be affected in different ways by climate change-induced shifts in their geographical distributions, and those of their vectors and hosts. Increased mean temperatures are projected to increase the global distributions of heat-loving bacteria, such as *R. solanacearum*, *A. avenae* subsp. *avenae* and *B. glumae* [44, 117]. Similarly, predictions are for an increased distribution of more themophylic plant-pathogenic xanthomonads but a decrease in the occurrence of cold-loving plant-pathogenic pseudomonads [44, 135]. Evidence supporting such predictions comes from studies in which elevated temperature increased the incidence of bacterial wilt and spot diseases in pepper caused by fastidious xylem-invading bacteria *R. solanacearum* and *X. campestris* pv. *vesicatoria*, respectively [175]. In warm climates, bacterial plant pathogens causing soft rots in plant tissues of a wide range of plant species in the field and after harvest during transit or storage, e.g. *Bacillus* spp. and *E. chrysanthemi*, are projected to increase their distributions. Conversely, the distributions of bacterial pathogens, which cause soft rots in temperate climates, e.g. *E. carotovora* subsp. *atroseptica* and *Clostridium* [134], are projected to contract [44]. There is a need for many more in-depth studies on the effects of elevated temperatures on bacterial infections in plants, resembling those mentioned above on bacterial wilt and spot diseases in pepper [176].

Alterations in mean temperature influence virus seed transmission rates, for example, when the nematode-transmitted, soil-borne viruses *Arabis mosaic virus* (ArMV) and *Strawberry latent ringspot virus* (SLRV) infect the key alternative weed host *Stellaria media* (chickweed). ArMV was more frequently seed-transmitted if infected chickweed mother plants were grown at 14°C (34%) than at 22°C (11%), but the reverse was true for SLRV, 14°C (0%) and 22°C (29%) [177]. Thus, increased mean temperatures might be expected to increase the SLRV reservoir for spread from *S. media* to susceptible crops with SLRV but decrease it with ArMV.

Increased mean temperature decreases the effectiveness of single dominant gene resistances that have temperature dependency and become ineffective when temperatures exceed a threshold [138, 178, 179]. Thus, when formerly virus-resistant cultivars are grown under warmer conditions, currently effective temperature-sensitive resistance genes (e.g. [4, 137, 138]) may become ineffective, resulting in epidemics in these cultivars. There are many examples of temperature-sensitive virus resistance genes, but the classic example is the N gene in some tobacco species (*Nicotiana* spp.). Samuel [180] observed that *N. glutinosa* plants inoculated with TMV developed small necrotic local lesions in inoculated leaves at 21°C, formed larger and more quickly spreading necrotic lesions at 28°C, but spread systemically causing mosaic without necrosis at 35°C. Thus, the localized hypersensitive response was overcome at high temperatures allowing the virus to spread throughout the plant. Extensive necrosis of tissues invaded by the virus at high temperatures results when the plants are transferred back from 32 to 22°C [163]. Some bacterial resistance genes may be rendered more effective by higher temperatures while others are not, e.g. in rice, resistance genes such as *Xa7* are more effective against some races of *X. oryzae* pv. *oryzae* at high than low temperatures, whereas other resistance genes are not [181]. Breeding of new rice cultivars likely to perform better in the tropics should therefore focus on incorporating resistance genes such as *Xa7* and avoiding others that are likely to be compromised under increasing temperature scenarios. Diminishing mean temperature decreases the effectiveness of plant antiviral resistance mechanisms based on gene silencing [182, 183]. Such a decrease would be likely to favour spread of virus epidemics in plants under lower temperature scenarios.

Increased mean temperature alters plant physiology by influencing secondary metabolite pathways, thereby altering the nutritious value of leaves to insect vectors. It also alters the patterns of gene expression of defence signalling routes against some insect vectors [178, 184, 185]. In addition, increased temperature increases stomatal conductance, which influences efficiency of photosynthesis [186, 187]. This would alter virus multiplication within cells thereby influencing systemic movement and virus acquisition by insect vectors.

Temperature is the predominant influence on insect herbivores modifying their development, survival, distribution and abundance [94, 153, 188, 189]. Moreover, considerable shifts in the distribution and abundance of arthropod vectors of plant viruses can result from small alterations in average temperatures [17, 94]. Aphid vectors of viruses react strongly to small changes in mean temperatures owing to their low developmental threshold temperatures, short generation times and great capacity for reproduction [153]. An additional five generations of aphids/year is predicted in temperate zones from a warming of 2°C [190]. The risk of serious epidemics of aphid-transmitted viruses therefore increases as their populations and activities increase. Whitefly vectors also react strongly to climatic changes because of their short generation times and great capacity for reproduction. With the important whitefly virus vector species *Bemisia tabaci* (sweet potato whitefly), 25–28°C is optimal for development [191], and much shorter adult-to-adult generation times occur at high (31–33°C) than low (17°C) temperatures [192]. Thus, rising mean temperatures increase the risk of damaging epidemics of viruses transmitted by *B. tabaci* in formerly cooler regions. Increased mean temperatures can also increase thrips vector populations by hastening their development rates, leading to more generations per year. However, as different thrips species have different thermal thresholds, a change in species composition is projected from global warming. Thus, the range of the important tropical vector species *Thrips palmi* (melon thrips) is expected to expand into areas formerly too cold for it, displacing vector thrips species adapted to cooler temperatures [90]. This in turn is projected to cause expansion of damaging topsoviruses transmitted by *T. palmi* (e.g. *Capsicum chlorosis virus* and *Groundnut bud necrosis virus*) into new regions, and contraction to cooler regions of topsoviruses transmitted solely by displaced species, e.g. *Iris yellow spot virus* transmitted solely by its cooler adapted *Thrips tabaci* (onion thrips) vector. Increased temperatures would stimulate the mealybug vectors (*Dysmicoccus brevipes* and *D. neobrvipes*) of *Pinneapple wilt-associated virus* and the mite vector *Aceria tosichella* (the wheat curl mite) of *Wheat streak mosaic virus* (WSMV), favouring increased epidemics of these viruses in pineapple and wheat crops, respectively [4, 193–195]. When Reynaud *et al.* [196] recorded the incidence of three tropical maize viruses and their leafhopper or planthopper vectors over 3 years in field experiments in Reunion Island, vector numbers and virus incidence were closely associated with temperature fluctuations, both increasing rapidly above 24°C. Thus, rising temperatures are likely to favour occurrence of severe epidemics of these hopper-transmitted viruses in areas previously unaffected by them.

Mean temperature can influence the efficiency of pathogen transmission from infected to healthy plants by insect vectors. For example, the incidence of *Pea enation mosaic virus* in pea crops is very dependent of its efficiency of transmission by its *Acyrthosiphon pisum* (pea aphid) vector. This transmission was considerably greater at 20 and 30°C than at 10°C [197]. With this kind of relationship, enhanced mean temperature might enable such viral pathogens to expand their range to areas formerly too cold for them to be transmitted efficiently. However, distributions of some other viruses might contract from regions with increased temperatures because of diminished virus transmission efficiencies at higher temperatures, as occurs with *T. tabaci* and TSWV [198]. Mean soil temperature influences the efficiency of transmission of soil-borne viruses. For example, the efficiency of transmission of *Raspberry ringspot virus* (RRV) to cucumber seedlings by its nematode vector *Longidorus macrosoma* was 80, 20 and 0% at 20, 25 and 30°C, respectively. The nematodes survived well at all three temperatures, but root feeding seemed inhibited at the higher temperatures [199]. Over time, increased temperatures might cause the geographical ranges of such temperate crop viruses to contract from the currently warmest parts of their ranges.

Survival and reproduction of vector nematodes in soil depends on mean soil temperature, soil moisture, soil type and presence of suitable hosts. Maps of the geographical distributions of longidorid and trichodorid virus-vector nematode species in the UK were modified to take account of climate change predictions [158]. Temperature was considered the critical factor, but soil moisture was deemed unimportant since dry conditions rarely occur. Because different temperature optima apply for feeding, hatching, reproduction and survival of different vector nematodes species, such data were used to predict changes in their activity and distribution. Based on a 160–200 km increase in nematode range for each temperature increase of 1°C, *Xiphenema diversicaudatum*, *L. macrosoma* and *L. attenuatus* were predicted to extend northwards causing epidemics of ArMV, RRV, SLRV and *Tomato black ring virus* in raspberry and strawberry crops. Introduction of susceptible crops was predicted to increase outbreaks of nematode-transmitted viruses that currently have restricted distributions, e.g. *Pea early browning virus* transmitted by *Trichodorus virulentis*, to areas previously too cold for them [158]. Jones [4] extrapolated from this research to temperate regions generally, predicting increased importance and distribution of nematode-transmitted viruses in Canada, Northern Europe, Russia, Northeast Asia and Southern South America.

Maximum or minimum mean temperature

In temperate regions, milder shorter winters resulting from climate change would favour survival of host-borne, plant debris-borne or vector-borne primary bacterial inoculum. This would be likely to increase the incidence of epidemics of plant-pathogenic bacteria. However,

such an increase in epidemics might not always be reflected in greater losses. For example, ice nucleation active pseudomonads (*Pseudomonas syringae* pvs *syringae* and *morsprunorum*) serve as nuclei for ice formation and therefore cause frost injury to plants when temperatures fall below freezing [136]. As the frost line moves northward in the northern hemisphere, reduced risk of the bacterial canker and dieback they cause in stone fruit trees is anticipated [44]. In the southern hemisphere, such risks should reduce as the frost line moves southward.

In temperate regions, survival of insect vectors is expected to increase with milder mean winter temperatures, and higher mean summer temperatures are likely to increase their development and reproductive rates. For example, with aphids, fewer days with frost and shorter cold spells increase their ability to over-winter in such regions, permitting them to expand their geographic ranges and increase the period in which they are active each year [48]. Increased winter temperatures induce earlier starts to aphid annual life cycles [200, 201], increase the proportion of winged aphids [202], which are responsible for most virus transmission, and stimulate their flight activity [203]. In northwest Europe, a 1°C increase in average winter (January–February) temperature advances the initiation of spring aphid migration by 2 weeks, especially with the important aphid vector species *Myzus persicae* (green peach aphid) [204]. Although the actual rate of advance varies with aphid species and region, over the next 50 years in Europe the overall date when aphid species are first caught is anticipated to advance by an average of 8 days [153]. Examples of aphid-borne virus diseases likely to become more widespread in temperate regions under increasing mean winter and summer temperature scenarios include 'leaf roll' in potatoes, 'yellow dwarf' in cereals and 'virus yellows' in sugar beet [4, 5, 205, 206].

In North America, increased winter temperatures are anticipated to increase the geographic range and severity of epidemics caused by xylem-limited plant pathogenic bacteria that overwinter in their insect vectors, e.g. *X. fastidiosa* subsp. *fastidiosa*, which causes Pierce's disease of grapevine and infects several other crops, and *X. fastidiosa* subsp. *multiplex* that causes phony peach disease and infects several other tree species [207]. *X. fastidiosa* is vectored by xylem-sucking insects such as sharpshooter leafhoppers (e.g. *Homalodisca coagulata*) and the spittlebug (*Philaneous spumarius*) [208]. Diseases caused by *Xylella* spp. occur in warmer regions currently and are rare or absent in regions with colder climates, but this is expected to change as the planet warms allowing their vectors to move northwards. Because of increased survival of its corn leaf beetle (*Chaetocnema pulicaria*) vector resulting from milder winters, Stewart's disease of maize, caused by *E. stewartii*, is also expected to increase in regions with colder climates [20]. With phytoplasma, milder winters are expected to result in increased survival not only of their leafhopper vectors but also of their infected perennial weedy hosts. Therefore, with climate change, epidemics of phytoplasmal diseases, such as aster yellows in a range of solanaceous crops, are expected to become more important at higher latitudes, e.g. in Canada [20].

For the important whitefly vector *B. tabaci* to flourish, an average monthly temperature of at least 21°C in the hottest month of the year, and a dry season with a period of 4 months of monthly rainfall of less than 80 mm, are needed [157]. As climate change progresses, in middle-latitude regions these conditions are likely to occur over increasingly wide areas, increasing the incidence of damaging begomovirus epidemics. *B. tabaci* and *Trialeurodes vaporariorum* (glasshouse whitefly) are both important vectors of different whitefly-transmitted virus diseases in China and the Andean region of South America. Both flourish in warm conditions but *B. tabaci* is less cold-tolerant than *T. vaporariorum*. In both regions *B. tabaci* is tending to displace *T. vaporariorum* because of increasing mean winter temperatures in places formerly too cold for it in winter while *T. vaporariorum* is increasing its distribution in formerly cooler regions. In the Andean mountains in South America, this is resulting in displacement of *T. vaporariorum* by *B. tabaci* at lower altitudes while *T. vaporariorum* is expanding its range at higher altitudes. In turn, this shift in vector distribution is influencing whitefly transmitted virus distributions, e.g. of *Potato yellow vein virus* which is transmitted by *T. vaporariorum* [209–212].

Unusually high air temperatures can reduce insect vector populations [95] so prolonged heatwaves may diminish epidemics of the bacteria or viruses they transmit. Moreover, plant pathogenic bacteria are often sensitive to increased temperature. Some are eliminated by hot air treatment of whole infected plants, seedlings or vegetatively propagated plant parts (scions, cuttings, sprouts, bulbs, tubers and corms) [115]. Many bacterial pathogens in or on seeds survive until germination, and sowing infected seed lots provides initial seedling infection sources scattered at random through crops from which spread to healthy plants occurs. Also, transport of bacterial pathogens in seed is an important means of survival and transfer in time and space [213]. Hot air treatment successfully removed bacterial diseases caused by *Xanthomonas* and *Pseudomonas* species from seeds of several crops including tomato, tobacco, rice, barley, cucumber and pumpkin [214]. Prolonged heatwaves associated with climate change, therefore, have the potential to diminish bacterial infections in plants and the losses they cause. Further research simulating heatwave conditions to treat infected seed or vegetative propagules would help to clarify the extent to which this is likely.

Although brief heat- or cold-shock treatments can induce symptom development, prolonged heatwaves are likely to cause remission of virus symptoms in infected plants by reducing their virus contents (e.g. [121, 122, 215]). Occasionally, prolonged heatwaves might eliminate

virus infections where systemic invasion is already incomplete or the virus involved has unstable particles. Heat treatment studies to eliminate viruses from virus-infected growing plants or dormant plant parts, such as tubers, budwood or sugar cane sets, usually used temperatures of 30–40°C for 6–10 weeks [95, 115]. Although heatwaves would rarely last this long, heat treatment for as little as 10 days at 37° days in a humid atmosphere freed potato tubers from infection with *Potato leaf roll virus*, which is phloem-limited [216]. In contrast, treatment of potato tubers at 37°C for 65 days was unsuccessful at eliminating PMTV, which is not restricted to vascular tissues [217].

Many viruses are seed-borne, surviving either as surface contaminants of seed coats or through infection of embryo cells. Where embryo infection occurs they usually survive as long as the seed does, but seed coat contaminants, which are mostly contact-transmitted viruses with stable particles, are potentially susceptible to inactivation. However, heat increases arising from heatwaves seem unlikely to be sufficient. For example, treatment of dry tomato seeds required 2–4 days at 70°C to eliminate seed coat contamination with TMV without impairing seed germination [218, 219]. More research is required to determine whether the heat arising from protracted heatwaves might sometimes be sufficient to inactivate seed transmission of seed coat contaminants with viruses with unstable particles.

Mean rainfall and altered rainfall patterns

Depending whether they increase or diminish, alterations in amounts and patterns of rainfall arising from climate change are likely to increase or decrease epidemics of non-fastidious plant-pathogenic bacteria in different parts of the world. Most are spread to their hosts by rain splash. Also, many have flagellae that propel them short distances through moisture films, enabling them to locate openings through which to enter their hosts (e.g. [115]). Moist conditions and rainfall are therefore critical elements driving their epidemics. However, dry conditions commonly favour their survival in plant residues. Most are soon killed or inactivated by desiccation when not protected by a host plant or protective extracellular polysaccharide exudate, ooze or slime. When they are moist, there is only a brief opportunity for infection to occur before they die off. For example, with protective exudate *E. amylovora* survived for more than 300 days at different temperatures and relative humidities, but only 13 days after removal of this protection. Also, *Corynebacterium flaccumfaciens* that causes bacterial wilt of common bean, survived for 5 years in dried bacterial ooze [213]. A change to shorter periods of diminished rainfall which provide sufficient moisture to remove protective exudates but insufficient to prevent drying before infection occurs would diminish non-vector-borne bacterial infections and epidemics, e.g. in mid-latitude regions, where arid and semi-arid environments already tend not to be conducive to many of them because of their high moisture requirement for infection.

Epidemics of pathogens transmitted by insects are likely to change if climate change alters rainfall patterns. For example, in mid-latitude regions of Asia, Australia, Latin America and Africa, the probability that damaging epidemics of viruses transmitted by *B. tabaci*, such as many begomoviruses, would occur increases considerably if they develop the longer dry seasons between cropping periods that favour *B. tabaci* population increase [157]. In contrast, in mid-latitude regions with Mediterranean-type climates and rain-fed, winter cropping, hotter, dryer summer conditions may decrease epidemics of aphid-borne viruses by diminishing their abilities and those of their aphid vectors to over-summer, e.g. with aphid-borne BYDV and *Beet western yellows virus* in parts of southwest Australia [220–222].

Epidemics of viruses transmitted by fungi or nematodes in the soil are projected to alter in response to altered rainfall patterns. Epidemics of fungus-transmitted viruses are likely to become prevalent over increasingly wide areas in temperate regions [4]. This is because of increased activity and movement of vector zoospores resulting from increased soil moisture and temperature, which would increase epidemics of furoviruses and bymoviruses of field crops such as cereals, potatoes and sugar beet. Regions likely to be affected include Canada, Northern USA, northern Europe, Northeast Asia and Southern South America [4, 101, 223–226]. Important examples of such viruses include *Soil-borne wheat mosaic virus*, *Wheat spindle streak virus*, *Beet necrotic yellow vein virus* and PMTV (e.g. [101, 223, 224, 227]). Conversely, as mean rainfall declines in mid-latitude and some sub tropical regions, epidemics of viruses with fungal vectors would be expected to become less widespread. This is because decreased soil moisture in rain-fed fields would diminish both activity and movement of fungal vector zoospores of the pecluviruses found in India and Africa, such as *Peanut clump virus* and *Indian peanut clump virus* [228, 229]. Similarly, declining soil moisture is projected to diminish the incidence of nematode-transmitted viruses in rain-fed crops in mid-latitudes [4].

An example where the process of seasonal shifts in rainfall patterns as a consequence of climate change has already caused shifts in pathogen distribution is provided by a fungal pathogen in south west Australia. This region has a typical Mediterranean climate with hot dry summers and wet winters. Substantial winter/spring rainfall is critical for epidemics of northern anthracnose (*Kabatiella caulivora*) in annually self-regenerating subterranean clover pastures [230]. Over the last three decades, south westerly cold fronts bringing winter/spring rainfall from the Indian Ocean have declined with distance from the coast (e.g. [4]). This drying process has caused the boundary of northern anthracnose epidemics in pastures

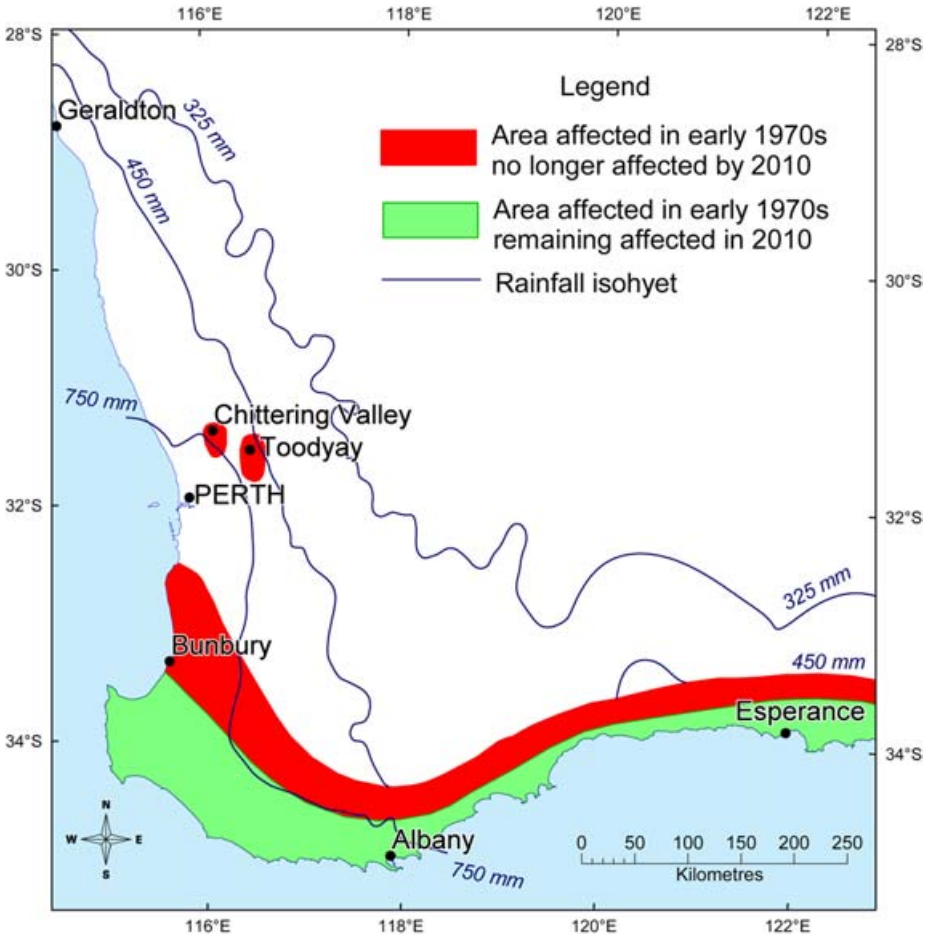

Figure 3 Contraction between 1970 and 2010 in the areas of southwest Australia containing subterranean clover pastures affected by northern anthracnose disease caused by *Kabatiella caulivora*. This contraction over the last 30 years was caused by a gradual decline in the numbers and intensities of southwesterly cold fronts bringing winter/spring rainfall from the Indian ocean. This drying process caused the boundary of northern anthracnose epidemics to contract nearly 200 km towards the western and southern coastlines.

to contract by nearly 200 km towards the western and southern coastlines (M.J. Barbetti, unpublished data) (Figure 3).

Extreme rainfall-related events

The increased frequency of extreme drought or flood events projected to arise from climate change would be likely to influence plant viral and bacterial disease epidemics in several ways. Some of these would enhance and others diminish their epidemics. The combination of disease and drought can be additive causing greater damage to crops than caused by either stress parameter alone, as occurs with the combination of *Beet yellows virus* or *Maize dwarf mosaic virus* infection and drought [231, 232]. Such additive effects would increase the incidence of economic losses caused [4]. However, in pot experiments, virus infection delayed the onset of drought symptoms in plants under dry conditions, thus prolonging their life [233]. More information is therefore needed on the effects of drought on virus symptoms in infected plants in the field. Prolonged drought stress on crop plants can limit insect vector population size, thereby decreasing losses arising from epidemics [95]. Drought-induced lack of soil moisture in fields inhibits both activity and movement of fungal vector zoospores, thereby preventing spread of the viruses they transmit, but flooding is likely to have the opposite effect unless conditions become too anaerobic for their survival. Although in the short term neither drought or flood conditions would inactivate the dormant fungal vector resting spores that allow such viruses to persist in the soil between growing seasons, they might tend to diminish gradually over time. Drought-induced lack of soil moisture and prolonged flooding might also be expected to inhibit activity of vector nematodes thus preventing spread of the viruses they transmit.

Both drought and flood can influence survival of unprotected viral or bacterial pathogens in contaminated soil and their abilities to act as sources of inoculum for spread to susceptible crops. For example, the dry,

compacted or waterlogged soils typical of drought or flooding, respectively, favour survival of the stable, contact-transmitted virus TMV in plant debris in soil but well-aerated soils favour its inactivation [234]. Thus, drought and flooding might both favour TMV spread to crops. Plant residues in or near a soil surface are generally more favourable to bacterial survival than those incorporated in the soil because they are protected from antagonistic soil microflora [213]. In the arid climate of Sudan, *Xanthomonas malvaceorum* survives between cotton crops on soil debris on the soil surface but in wetter Tanzania it can barely survive in this way [213]. Thus, unless plant pathogenic bacteria are saprophytes, their survival in soil would be favoured by drought but limited by flooding or waterlogging.

An increasing frequency of heavy rainfall events is predicted to result from climate change. These are expected to influence epidemics in different ways depending on how pathogens spread. They would wash insect vectors such as aphids, thrips and whiteflies off foliage, thereby slowing virus epidemics by diminishing vector populations, as occurs with monsoonal rains and *T. palmi* in Northern Australia [235]. In contrast, foliar infections with plant pathogenic bacteria often increase after rains wash bacteria into natural openings or wounds, so their epidemics are likely to increase. With epidemics of contact-transmitted viruses and non-fastidious plant pathogenic bacteria, greater frequency and intensity of summer storms associated with heavy rainfall or hail that increase rain splash and wounding of plants, and also provide moisture, would be expected to accelerate their spread, favouring more severe epidemics [20]. With water-borne viruses, an increase in flooding from summer storms would accelerate their dispersion *via* irrigation and drainage channels, streams and rivers (e.g. [124–126]). However, waterlogging kills pupal life stages of thrips vectors in the soil [236] and oviposition of *B. tabaci* is impaired by heavy rain [88, 237], thereby slowing down epidemics of viruses they transmit by diminishing vector populations.

Relative humidity

Relative humidity determines things such as duration of leaf wetness and is closely tied to temperature, and so is more predictable than many other climate parameters. Plant pathogenic bacteria that are thickly coated with protective extracellular polysaccharide slime survive better at the plant surface than others because the slime attaches them and protects them from desiccation. Those that survive as epiphytes on plant surfaces, such as leaves, flowers and fruits, do so by absorbing nutrients that leak from plant cells [115]. Even with a local microclimate typified by moist surfaces and high relative humidity, other plant pathogenic bacteria are unlikely to survive long unless they get washed into natural openings or wounds. Although they have flagellae, the self-mobility within water films on plant surfaces that enables them to find natural openings is restricted to short distances, and such films are unlikely to be present under conditions of low relative humidity. Low-humidity climate change scenarios that cause a dry microclimate at the leaf surface are therefore non-conducive to epidemics of most non-fastidious bacterial plant pathogens.

Spread of contact-transmitted viruses in crops and pastures is favoured by the lush plant growth and soft tender leaves that develop under conditions of high relative humidity. Such plants are much more susceptible to infection than the hard-leafed plants typical of low-humidity conditions. This is because the wounds through which such viruses have to penetrate a plants' protective cuticle before they can invade damaged cells develop much more readily when growth is soft (e.g. [77, 238]). Thus, high relative humidity climate change scenarios would be expected to favour their epidemics but low relative humidity scenarios would have the opposite effect.

Relative humidity can have marked effects on the rate of multiplication and movement of airborne virus vectors [95]. For example, when flight performance of *A. glycines*, the vector of *Soybean mosaic virus* in soyabean, was studied, at optimum flight temperature (16–28°C) the optimum relative humidity for flight was 75% [239]. Also, the observation is often made that very high humidity favours development of entomopathogenic fungi that kill aphid vectors in field crops (e.g. [222, 240, 241]). The influence of relative humidity on virulence of *Verticillium lecani* toward *Rhopalosiphum padi* (oat aphid), an important vector of BYDV in cereals, was tested at a temperature regime (27°C) at which 100% mortality occurred within 3 days [242]. When the effects of 100, 75, 32 and 12% relative humidity values were compared, aphid fecundicity was lower and mortality higher at all of them. However, sporulation of the fungus on dead aphids occurred sooner at 75 and 100%, and a dense mat of mycelium that covered their bodies never formed at 32 or 12%. More research is needed to establish the effects of increased or decreased relative humidity scenarios on the behaviour and survival of different kinds of arthropod vectors.

Wind speed and direction

Vector-borne pathogens can be distributed over long distances by strong winds. For example, viruliferous aphids are sometimes transported over very long distances initiating new virus infection foci far away from the initial virus inoculum source, e.g. with wind movement of cereal aphids vectors of BYDV from the south to north in the USA [152, 243]. Therefore, projected periodic increases in wind velocity associated with climate change are likely to increase dispersal of insect and mite vectors via wind currents, especially those vector species that already disperse readily [244], leading to virus epidemics

over wider areas. Conversely, when wind speed is very high for long periods it can inhibit spread of vector-borne pathogens, e.g. winged aphids tend not to fly when wind speed is too great [95], which would inhibit spread of aphid-borne viruses when such winds continue uninterrupted over prolonged periods.

Wind direction is very important in determining the predominant direction of spread of vector-borne pathogens. The importance of prevailing wind in establishing large differences in virus incidence upwind and downwind of a virus infection source has been demonstrated for aphid-borne (e.g. [245]), thrips-borne [246] and mite-borne [193] viruses. The same applies with leafhopper vectored viruses, e.g. the *Beet curly top virus* vector *Circulifer tenellus* (beet leafhopper) cannot make progress against a headwind stronger than 2 miles/h [95]. Prevailing wind is particularly important for dispersion of mite-borne viruses, such as *Blackcurrant reversion virus* vectored by *Cecidophyopsis ribis* (gall mite) and WSMV vectored by *A. tosichella*, because they are tiny and wingless and depend on wind for dispersion away from an infection source [193, 247]. Thus, projected alterations in prevailing wind direction associated with climate change are likely to alter the predominant directions in which insect and, especially, mite vectors of viral and bacterial plant pathogens are dispersed.

Some plant-pathogenic bacteria that lack vectors, e.g. *E. amylovora*, develop aerial strands made of bacterial ooze and remain viable inside them for long periods. These strands are readily dispersed by winds [213]. Projected alterations in prevailing wind direction associated with climate change are likely to alter the directions in which such stands are dispersed, potentially giving rise to outbreaks in regions formerly free from infection.

Increasing incidences of intense storms with very high wind speeds and altered traditional trajectories are predicted in future climate change scenarios. Such storms are likely to be major influences in distributing vector-borne viruses and bacteria more widely around the globe. They are also likely to promote occurrence of damaging epidemics in regions where vector-borne pathogens already occur. With epidemics of contact-transmitted viruses and non-fastidious plant pathogenic bacteria, greater frequency and intensity of summer storms with high winds that increase wounding of plants would be likely to accelerate their spread favouring more severe epidemics [20].

Tropical summer cyclones associated with exceptionally strong winds and torrential rainfall develop every year in the Indian Ocean to the north of Western Australia (Figure 4). Increasing numbers and intensities of such storms are anticipated during the next 20 years, which would increase damaging WSMV epidemics in wheat crops in the southwest Australian grainbelt. Wheat crops are grown in winter/spring, when, as mentioned above, southwesterly cold, rain-bearing fronts pass from the coast inland, rainfall declining with distance from the coast. This winter/spring rainfall pattern gives rise to the grainbelt's high, medium and low rainfall zones. In summer, tropical cyclones from the Indian Ocean to the north that make landfall along the central coastline turn to become north westerly winds that spread thunderstorms into the interior of the continent. Most miss the grainbelt as they move southwards, but occasionally this region receives thunderstorms with torrential rainfall. Current climate change predictions are that these cyclones would increase and become more intense, leading to a greater frequency of summer thunderstorms, especially in the eastern grainbelt (low-rainfall zone) (e.g. [248]). The combination of high temperatures and cyclonic rainfall stimulates growth of a substantial 'green ramp' of grasses and volunteer cereals well before wheat is sown in late autumn. This warmth and abundant growth provide ideal conditions for *A. tosichella* to multiply before the growing season and spread WSMV to newly emerged wheat crops causing damaging epidemics [4, 193]. In contrast, an example where rainfall from these tropical cyclones is expected to diminish pathogen epidemics in the same region is provided by a fungal disease. Epidemics of blackspot disease of field pea caused by *Mycosphaerella pinodes*, *Phoma medicaginis* var. *pinodella*, *Ascochyta pisi* and *Phoma koolunga* are initiated by airborne ascospores released from infested crop stubbles [249–251]. Timing of pseudothecia maturity and subsequent ascospore release after harvest is triggered by rainfall, heavy dew and high humidity [251]. The increasing incidence of summer thunderstorms predicted would favour earlier maturity of pseudothecia and subsequent ascospore release before pea crops are sown. This would cause the major ascospore showers to occur and dissipate before pea sowing time leading to significant reductions in pea blackspot disease epidemics [252–254].

Greenhouse gas concentration

There is limited information on the responses of plants to virus infection when elevated greenhouse gas concentrations are present in the atmosphere, but elevated CO_2 increased resistance to PVY infection in tobacco plants [255]. Elevated CO_2 also has the potential to suppress pathogen-induced virus resistance [3]. Thus, although increasing temperatures alter the rate of development, duration and frequency of virus epidemics by changing rates of virus multiplication, modifying host resistance and changing the physiology of host–virus interactions, elevated CO_2 may diminish the effect of temperature on epidemics by increasing virus resistance. However, elevated CO_2 can also have the opposite effect if it increases the biomass of virus-infected plants, thereby increasing the virus reservoir for spread to healthy plants by insect vectors, e.g. as occurs in oat crops in which the increased biomass of BYDV-infected oat plants increases the virus

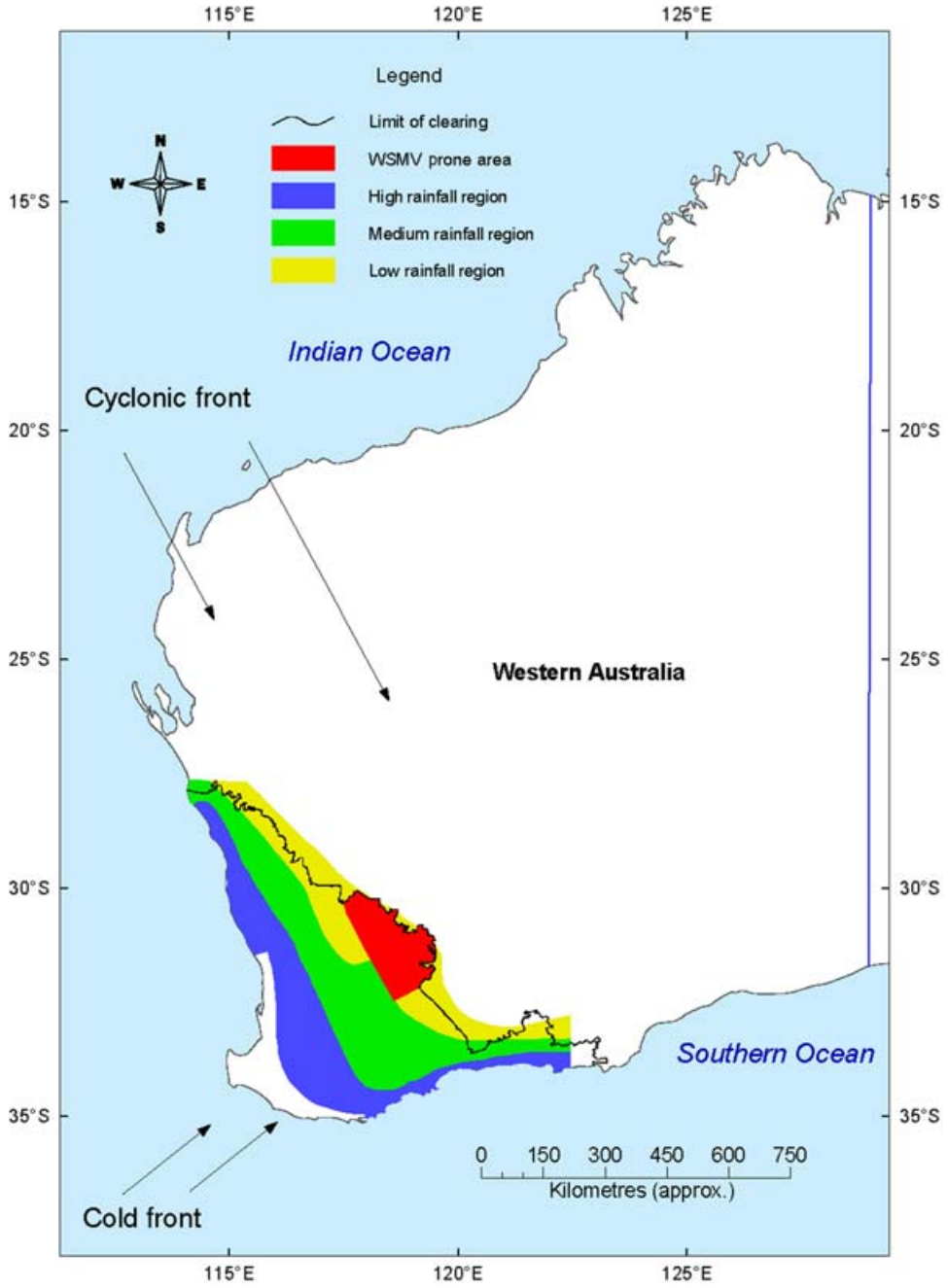

Figure 4 Projected influence of increasing numbers and intensities of tropical summer cyclones on the occurrence of *wheat streak mosaic virus* epidemics in wheat crops in the grainbelt of south west Australia. The cyclones bring thunderstorms with torrential rainfall, which stimulates growth of volunteer cereals and grass weeds. The combination of abundant growth and high summer temperatures provides ideal conditions for the vector mite (*Aceria tosichella*) to multiply before the growing season starts and then spread the virus to emerging wheat crops, especially in the eastern central grainbelt (modified from Jones [4]).

reservoir for spread to healthy plants by its aphid vectors [256].

With bacterial cell growth in the liquid culture, Wells [257] found inhibitory effects at >3 and >10% CO_2 on growth of *Erwinia* spp. and *Pseudomonas fluorescens*, respectively, but none at 0.03–3% CO_2. Inhibitory effects on soft rot development in tomato fruits infected by *Pseudomonas marginalis* were caused by 2–10% CO_2 [258]. In contrast, doubling ambient CO_2 concentration marginally increased the incidence of bacterial wilt and spot diseases in pepper caused by *Ralstonia solanacearum* and *X. campestris* pv. *vesicatoria*, respectively [176]. These limited findings indicate that, depending on the pathosystem involved, elevated CO_2 may either diminish or accelerate disease epidemics caused by plant-pathogenic bacteria.

Elevated CO_2 concentrations influence different insect vector species in different ways. Elevated CO_2 concentrations have the potential to influence aphid numbers but experiments with elevated ambient CO_2 had mixed effects on aphids, the results being highly species-specific [175, 259–265]. When such factors were included, conflicting results were obtained with model predictions over the effect of climate change on abundance and distribution of cereal aphid vector species in Canada and the UK, populations increasing in summer in some regions but diminishing in others [159, 266]. Elevated ambient CO_2 concentrations at 700 ppm had no effect on *B. tabaci* but affected *T. vaporiarum* negatively at 1000 ppm [267, 268]. Thus, elevated CO_2 seems more likely to influence epidemics of viruses transmitted by the latter species. Elevated CO_2 had little effect on populations of the important thrips tospovirus vector species *Frankliniella occidentalis* [269, 270], but may increase the population sizes of some other thrips vector species [263]. Elevated CO_2 may compromise host resistance to insect vectors. This occurred with red raspberry resistance to the aphid vector *A. idaei*, which transmits several raspberry viruses [271].

As with increased temperature (see above), elevated atmospheric CO_2 alters plant physiology. The efficiency of photosynthesis depends on intercellular CO_2, which is related to the concentration of atmospheric CO_2 [186, 187]. Increased atmospheric CO_2 also influences secondary metabolite pathways, altering the nutritious value of leaves to arthropod vectors, and the patterns of gene expression of defence signalling routes against some of them [178, 184, 185]. Such effects on photosynthesis and secondary metabolites would be likely to influence the spatial and temporal dynamics of virus epidemics indirectly. Alterations in plant growth or morphology arising from increased atmospheric CO_2 are likely to influence vector behaviour also. For example, faster growth resulting in rapid canopy development in a crop often decreases aphid vector landing rates thereby decreasing spread of aphid-borne viruses in the crop, for example with CMV and BYMV in lupin (e.g. [76, 77]). Increased atmospheric CO_2 might be expected to decrease virus epidemics in such cases.

From the above, there is obviously insufficient information on the direct effects of elevated CO_2 on multiplication or spread of viral and bacterial infections within plants, its effects on their vectors or the ways it influences their epidemics. Indeed, there is apparently no information whatsoever on such effects involving spiroplasma or phytoplsama. There is also no information on the influences of the greenhouse gasses nitrous oxide or methane on viral and bacterial infections within plants or their vectors. Of particular interest here is examining the effects of these parameters on disease epidemics in field situations, including in free-air CO_2 enrichment (FACE) facilities and in crops grown in regions with higher temperatures or where drought conditions occur [65, 272, 273].

General climate instability

General climate instability impacts on plants, vectors and pathogens alike and alters pathosystem dynamics, which, in turn, drives an accelerated rate of pathogen evolution in response to changing circumstances. Furthermore, the whole process is being exacerbated now by climate change and, in many regions, rapid human population growth, at a time when world trade in plants and plant products increasingly moves plants away from their centres of domestication and disperses previously localized pathogens and their vectors widely. The relaxing of national plant quarantine regulations to meet revised World Trade Organisation regulations also contributes to this trend [4].

Increasingly intense and frequent extreme weather events and a generally less predictable climate are expected to increase uncertainty over the effectiveness of control measures against epidemics caused by viruses and bacteria. Such uncertainty would in turn compromise decision-making over when control measures are needed and, if so, which to deploy to ensure a successful outcome [4]. Also, social factors, such as diminished availability of agricultural labour, would change options available for managing virus epidemics [3]. Crops may become physiologically less able to withstand infection when they are grown in regions where the temperature has become too high for their optimal growth. Also, when cultivars with temperature-sensitive resistance genes are planted, these may become ineffective. Timing of spays with oils, repellents or pesticides to coincide with peak arthropod vector flight times is likely to become more difficult to predict. Cultural control measures likely to be less reliable in suppressing epidemics include planting upwind of infection sources when prevailing wind patterns vary, planting early maturing cultivars or harvesting early to avoid exposure of crops at peak insect vector flight times, and manipulation of sowing date to avoid co-incidence of peak times for insect vector flights with vulnerable early crop growth [4]. Non-selective control methods would be especially important when attempting to limit spread of unknown or little understood viruses or bacteria occurring as a consequence of climate change. In such cases, tactics that employ generic information on measures used effectively with related cropping situations and pathosystems would be important. Generic information refers here to information about the effectiveness of non-selective control measures against several different pathogens that spread in similar ways, in contrast to selective control measures that are specific, only targeting single pathogen species or, single variants or strains within pathogen species [77, 78].

As climate instability increases, predictive models would be needed increasingly to establish when and where control measures are necessary [15, 154, 222, 240, 274, 275], which combinations of control measures are likely to be effective (e.g. [156]) and the risks

that new pathogen emergence scenarios may arise (e.g. [157]).

In undisturbed communities of wild native plants, the natural control measures that help suppress virus epidemics include: (i) spatial separation (isolation) between similar native plant communities diminishes inoculum sources needed to initiate epidemics; (ii) abundance of vectors is diminished by naturally occurring predators and parasites; and (iii) admixture with other wild plant species that are non-hosts decreases virus spread to susceptible hosts. Also, co-evolution of host plants with viruses over millennia results in selection of natural host resistances and tolerances to viruses and vectors [4, 78, 83, 84, 91, 113]. Fragmentation and disturbance of natural vegetation can disrupt these natural control measures so it is likely to exacerbate epidemics of indigenous or introduced viruses in native plant communities. Direct stresses on native plants caused by climate change, such as general climate instability, extreme weather events, enhanced temperature and diminished rainfall, are likely to exacerbate these epidemics in similar ways. Little is known about how best to control them, but knowledge of the natural control measures that operate in undisturbed native plant communities provides clues, e.g. release of predators or parasites that target introduced vector species might help diminish virus spread without disturbing or negatively impacting on natural vegetation or its native animal population [4].

From the above, it is clear that research is urgently needed that addresses how best to manage viral and bacterial disease epidemics stimulated or aggravated by climate change in cultivated plants and native vegetation.

Indirect climate effects

Alterations in ranges of cultivated plants grown and regional areas cultivated

In the future, as conditions in formerly cooler higher latitude regions become warmer under climate change predictions, the regional areas growing cultivated plants and their annual growing periods are projected to expand. Warmer conditions would be generally more conducive to development of infections with many plant-pathogenic vector-borne viruses and bacteria, and their vectors, as well as to development of infections of those without vectors, as described above. Moreover, the increased areas of cultivation and annual growing periods would be likely to increase overall pathogen inoculum levels and diminish isolation between infected and healthy plantings. This would increase the spread of many plant-pathogenic viruses and bacteria, and the development of serious disease epidemics caused by them [4]. In contrast, in middle-latitude, arid or semi-arid regions, the reduced rainfall and increase in periods of drought projected to arise from climate change are likely to decrease the diversity and areas of crops and pastures being grown. Such a scenario of less conducive climatic conditions for many viral and bacterial pathogens and vectors, decreased field sizes and annual growing periods and greater isolation between plantings would be likely to decrease the distributions, incidences and epidemic rates of most that remain, and the diversity, distribution and activity of their vectors. However, this might not always apply, e.g. to begomoviruses in some moderately semi-arid regions, because, as mentioned above, dry seasons with a period of at least 4 months of monthly rainfall of less than 80 mm are needed for *B. tabaci* to flourish [157].

As the ranges of cultivated plants currently restricted from being grown in cooler regions expand due to increases in temperature or rainfall, the distributions of most of the plant pathogenic viruses and bacteria that infect these plants, and their vectors, are also projected to expand with them (e.g. [4, 60]). Dispersion of viral and bacterial pathogens to such regions is likely to occur through introduction of infected seed, infected vegetative propagules, contaminated soil or dust, or infectious arthropod vectors borne on wind currents. Such dispersion is also likely to arise through international and regional trade in fruits, vegetables or grains. Indeed, such dispersion is already happening owing to the continually expanding volume and rapidity of trade in plants and plant products, and movement of plants away from their centres of domestication, and so climate change would merely be accelerating a process already underway. The consequence is exposure of native plants to new encounters with newly introduced pathogens and vectors that spread to them from introduced cultivated plants. The same process exposes introduced cultivated plants to new encounters with indigenous pathogens spreading from native plants or already established in other domesticated plants. The process of emergence is already well documented for viruses, especially in the tropics and subtropics [4, 17, 83, 88, 106, 131]. Such new encounters provide new opportunities for rapid, adaptive virus evolution and host species jumps, thereby increasing the rate of invasion of introduced plants by indigenous viruses emerging from native plants, and of indigenous flora by introduced viruses [4, 106, 107, 131, 276]. Begomoviruses in particular evolve rapidly, making host jumps to new species they have not met previously [89, 107, 131, 276]. As such viruses did not co-evolve with the plants they encounter for the first time, damaging epidemics are likely to arise [4, 17, 78, 91, 106]. Generalist viruses adapt to new hosts better than specialist viruses as the geographic ranges of their plant hosts and vectors change and new encounters occur with host plants that they have not met previously [4]; so climate change is likely to favour their epidemics over those of specialists. Also, when viruses and vectors that native vegetation has not been exposed to before are introduced to it through new encounters, the natural control measures that operate to limit virus epidemics in native plant communities may be rendered

less effective by stress caused by climate change, as mentioned above. This could aggravate epidemics of introduced viruses in native vegetation.

By way of contrast, in drying middle latitude, arid and semi-arid regions, climate change is likely to decrease new encounters between indigenous viruses and introduced hosts, and introduced viruses and native plants. This is because diminishing rainfall and more frequent droughts would decrease the amount of land used for cropping and so lessen fragmentation of remnant native vegetation [4, 277]. New epidemics in introduced plants caused by viruses emerging from the local flora, or in native vegetation caused by newly introduced viruses, would then diminish.

Alterations in alternative weed or cultivated reservoir hosts

Seed transmission of viruses and bacteria in weeds is well documented, as is the role of weeds themselves as important alternative host infection reservoirs for spread of pathogens to nearby crops by vectors (e.g. [4, 77, 83, 84, 89, 98, 99, 113, 115, 177, 213, 220, 278]). Much of the discussion in the previous session therefore also applies to weeds. Thus, when seeds of weed species not previously found in a region are introduced inadvertently as contaminants during trade in plants and plant products, seed-borne inoculum of viruses and bacteria can be introduced at the same time (e.g. [4]). Weed species formerly unable to establish because they possess higher temperature or rainfall requirements may subsequently be able to establish owing to altered environmental conditions resulting from climate change. Contamination of their seeds could introduce plant pathogenic bacteria and viruses that then invade native vegetation that never encountered these pathogens before. Alternatively, the introduced weeds could become infected by pathogens emerging from indigenous vegetation or prove exceptionally attractive hosts to new insect vectors. They might then provide pathogen infection or vector infestation reservoirs that accelerate spread of emerging viruses or bacteria to newly introduced crops (e.g. [89]). In addition, the introduced weeds might readily tolerate infection with an already occurring viral or bacterial pathogen or infestation with a local vector, but the pathogen concerned might be very damaging to an important crop grown locally. If weed control is inadequate, their presence would aggravate damaging epidemics of already-occurring viruses or bacteria in the vulnerable local crop.

The importance of other species of cultivated plants as alternative reservoir hosts for viral or bacterial pathogens or their arthropod vectors from which these pathogens can spread to nearby crops is also well documented (e.g. [75, 76, 77, 78, 84, 86, 95, 278]). Following alterations in climate, cultivated plants introduced to regions formerly unsuited to them may be tolerant of viral or bacterial pathogens that are very damaging to vulnerable cultivated plant species growing nearby. Their presence may therefore aggravate damaging viral or bacterial epidemics in the vulnerable crop, which might be one grown traditionally in that region before it became suitable for plantings of the newly introduced crop. An example is given in more detail in the next section of a scenario where a traditional crop suffered damaging begomovirus epidemics after a vector-tolerant crop became grown much more widely in the same region. In brief, an increase in soyabean production greatly increased the population of *B. tabaci*, which in turn greatly accentuated damaging epidemics of *Bean golden mosaic virus* (BGMV) in common bean [89].

Changes in cultivation systems

The current rapid expansion in human activity and need to feed the burgeoning human population includes adopting ever more sophisticated agricultural practices. It also promotes more widespread cultivation in monocultures. Changes in cultivation systems likely to arise in areas rendered more suitable for large-scale plant cultivation as a consequence of climate change include: agricultural intensification, extensification and diversification; greater use of chemical control measures; use of irrigation in dry regions or to provide all-year-round cropping; and increased use of protected cropping [4, 106]. Such changes in cultivation systems often favour frequent and damaging virus and bacterial epidemics. For example, with RYMV in rice in Africa, continuous cropping using irrigation, extensive growth of grass weeds and volunteer rice, and decreased inter-field distances facilitated build-up of RYMV and its beetle vectors. Infection soon became prevalent in many areas, thereby changing the disease RYMV caused from a localized minor one, to one causing severe recurrent epidemics. Intensification and extensification of rice cultivation across Africa then drove wide-scale propagation of RYMV resulting in frequent epidemics over a much wider area of the continent [106]. Also, in Brazil *B. tabaci* and indigenous begomoviruses co-existed initially without seriously affecting cultivated plants. This changed in the 1970s with intensification and extensification of soyabean production, the area planted increasing from 1 million ha in the early 1970s to 13 million ha by 1983. Soyabean is a favoured host of *B. tabaci*; so the increase in production area caused a marked increase in its population. This substantial vector population increase resulted in devastating epidemics of BGMV in nearby common bean crops, despite this being a relatively poor host for *B. tabaci*. The severe yield losses suffered resulted in a major switch away from common bean to other crops such that, although formerly the main producer of common bean in the world, Brazil now has to import to meet internal demand [130].

In arid and semi-arid mid-latitude regions, the option of growing rainfed crops without adoption of stringent

water-saving measures is likely to become increasingly limited as the climate dries and becomes hotter. Dryland cropping is already difficult to sustain in many, mid-latitude regions, such as southern Australian grainbelts, where land degradation is also a serious concern. Cultivation practices that improve rainfall-use efficiency involve minimizing moisture losses from soil and weeds and maximizing the proportion of rainfall available to the crop [279]. Such practices are likely to become even more necessary in the future if cropping is to survive. However, some of them, such as wide row spacing, low sowing rate and delayed sowing, increase crop attractiveness for aphid vector landings, which accelerates virus spread by delaying canopy closure resulting in more damaging virus epidemics (e.g. [76, 77]).

Protected cropping environments are often highly conducive to epidemics caused by viruses and bacteria because the artificially maintained irrigation regimes, high relative humidities and warm temperatures used, and cultural practices employed, favour them or their vectors, including whiteflies, thrips, aphids and fungal root-infecting vectors. Fortunately, the high value of the crops grown often permits deployment of comprehensive, but expensive, integrated disease management approaches (e.g. [77]). Therefore, at least in developed countries, under protected cropping any impacts of climate change in aggravating epidemics caused by viruses and bacteria are likely to be contained, apart possibly from the influence of increased greenhouse gas concentrations.

Conclusions

Our review addresses the many multifaceted and geographically distinct ramifications of climate change likely to influence infections and epidemics of vector-borne and non-vector-borne viral and bacterial plant pathogens worldwide. This constitutes a challenging undertaking because the information currently available about probable alterations in most of the diverse biological parameters involved is limited, and in some cases completely lacking. Moreover, additional complications arise from presence of vectors in most of the pathosystems concerned making generalizations across diverse types of host plants and pathogens more difficult than in situations where vectors are absent. Within this context, the review seeks to give as comprehensive a coverage as possible of available research data on the probable influences of climate change on diseases caused by viruses and bacteria in plants in different regions of the world.

Depending on the combinations of pathosystem, cultivation practices or ecologies concerned, each climate change parameter can influence plant hosts, pathogens and vectors directly. The important climate parameters here are alterations in temperature, rainfall, relative humidity, wind speed and direction and greenhouse gasses. In addition, complex climate-change-induced temporal and spatial shifts in crop, reservoir host and weed distributions, and alterations in cultivation systems can influence hosts, pathogens and vectors indirectly. To identify the changes likely to arise and any information gaps, we developed frameworks for (i) each important direct and indirect climate change parameter and (ii) each significant biological (host, vector and pathogen) pathosystem parameter. These frameworks were then cross-checked one against the other. This analysis revealed that alterations in climate are likely to modify diverse components of viral and bacterial disease epidemics in many different ways, including altering host morphology, physiology and resistance to vectors or pathogens, and vector and pathogen life cycles, abundance, diversity, reservoirs and inoculum. Depending on the type of pathosystem and circumstances, in many instances climate change seems likely to enhance viral and bacterial disease epidemics in higher and lower latitude regions. In other instances, it is likely to have the opposite effect, especially in drying mid-latitude regions. Moreover, as temporal and spatial shifts in their distributions cause newly introduced crops and weeds to meet indigenous vegetation for the first time, new encounter scenarios between cultivated and wild plants would inevitably increase. This would be expected to accelerate the appearance of epidemics caused by (i) new or little understood viral pathogens that emerge from indigenous vegetation to threaten newly introduced cultivated plants; and (ii) newly introduced pathogens and vectors that arrive with newly introduced cultivated plants and invade native plant communities. Furthermore, climate change is likely to diminish the effectiveness of some control measures, and viral and bacterial epidemics are projected to become less predictable, causing increasing difficulties in suppressing them successfully using current management technologies. In many cases, losses in cultivated plants and damage to natural vegetation resulting from viral or bacterial diseases are likely to increase considerably with potentially serious consequence for world food security and plant biodiversity. Successful adaptation of the global food system to future climates requires a research effort that targets the specific challenges climate variability imposes on production, such as those arising from epidemics of viral or bacterial diseases.

Many plant pathogen epidemics represent scenarios where multiple stresses occur, but most experiments do not address climate change scenarios where several parameters change simultaneously and continuously, so limiting their usefulness in making predictions. We found published research which examined the influences of a single environmental variable, such as elevated temperature, on mixed infections with plant viruses, but no such experiments with mixed plant bacterial pathogen or multiple vector species scenarios. Moreover, very few experiments examined the influences of multiple environmental variables on single viral or bacterial pathogen infections or single vector species. The challenge in the

future would be to manipulate multiple environmental parameters across multiple pathogens and vectors together on a single host species to define future expansions or contractions in individual and combined disease epidemics. Such studies are urgently needed to provide a clearer picture of the damage that epidemics of viral and bacterial pathogens are likely to cause as climate change progresses.

Cultivated plants grown in regions where environmental conditions are unsuitable for optimal growth are likely to become physiologically less able to withstand infection with viral and bacterial pathogens. Also, host resistance measures against both types of pathogen, and several cultural control measures against viruses, are projected to become less effective. Moreover, climate instability is likely to compromise decision-making over when control measures, such as pesticide applications against vectors, are needed. Mitigating losses caused by viral and bacterial disease epidemics stimulated or aggravated by climate change would therefore require a supple, adaptable, intelligent approach that emphasizes shrewd and locally appropriate use of disease management tactics. Future research would need to focus on developing integrated disease management approaches that are well validated by field experimentation and effective against plant viral or bacterial epidemics under diverse climate change scenarios. Such approaches need to include control measures that are non-specific, and so likely to remain effective under widely diverse climate change scenarios (e.g. [4, 77, 78]). They would also need to emphasize rapid application of technological advances that improve the capacity for shrewd and locally appropriate decision-making. Use of predictive models identifying whether damaging epidemics are expected to occur in a locality would become increasingly important, as would their use to help establish which control measures are likely to remain effective, or become less effective or ineffective, and when the critical time to deploy such measures would be for different climate change scenarios and pathosystems (e.g. [15, 154]).

Additional recommendations for future research that arise from this review are:-

1. There is a need for more sophisticated, 'big picture', modelling studies that establish where climate change is likely to result in damaging viral or bacterial epidemics in regions where the pathogens concerned were previously considered of little importance. Similarly, more climate change scenario modelling is needed to establish when significant viral or bacterial pathogens or vectors are likely to invade regions where conditions were formerly unsuitable for them.
2. It will become increasingly important to identify locations where climate change induced temporal and spatial shifts in crop, reservoir and weed host distributions are likely to foster new encounter scenarios that result in damaging epidemics caused by emerging viral pathogens. To achieve this, research is needed to (i) understand which climate change parameters accelerate evolution of generalist virus pathogens, including their ability to make host species jumps, become more virulent and overcome host resistances; and (ii) devise models that use this information to help identify where new encounters that result in virus emergence are likely to occur in a given locality, and the probability that damaging virus epidemics of emerging viruses would occur as a consequence of climate change.
3. Research in native plant communities is needed to better understand the likely influences of different climate change scenarios on epidemics arising from new encounters with introduced viruses or vectors. This is currently a much neglected topic, but the information provided would assist greatly in development of measures that mitigate the potential effects of these epidemics on natural ecosystem health and biodiversity. Quarantine measures to minimize the chances that pathogens become introduced with cultivated plants when these are moved to locations previously unsuitable for them would become increasingly valuable in this regard.
4. Major shifts in species balance can occur when plants debilitated by virus infection are unable to compete well with healthy plants of other species in mixed-species populations (e.g. [81, 127]). Such shifts are likely to be accentuated by climate change, so research is needed on its influences, particularly that of enhanced temperature, where viral or bacterial epidemics are occurring within only one individual component species within mixed-species native plant communities or pasture swards.
5. Although the influence of elevated temperature on plant viral and bacterial diseases has received greater attention than the influence of other climate parameters, important issues that need to be addressed were neglected in such studies. Examples include the influence of temperature on multiplication and movement of mollicutes within plants, multiplication of viruses and mollicutes within vectors, and the transmission efficiencies of viruses or bacteria by some types of vectors (e.g. beetles, planthoppers, spittlebugs and fungi). Moreover, more research is needed on the possible effects of prolonged heatwaves in mitigating or enhancing damaging epidemics. Examples here include determining their possible influences on spread of contact-transmitted viruses within pastures, or providing more information over the possibility that they might eliminate some viruses or bacteria from contaminated seed lots or vegetative propagules.
6. We found no information concerning the possible effects of elevated concentrations of the greenhouse gasses nitrous oxide or methane on any of the biological parameters associated with viral or bacterial diseases of plants. Also, research on the influences of

CO_2 on viral or bacterial infections of plants was minimal, and there was no research on its effects on mollicutes. Moreover, although there was limited information on its effects on some insect vectors, there was none for mite, nematode or fungal vectors. Addressing these deficiencies concerning the influences of greenhouse gasses should be an important focus for future research.

7. We found no information regarding the likely effects of climate change on several biological parameters, e.g. pollen transmission, ability to multiply within vectors, biological control and cultural control measures against bacterial pathogens. We also found only minimal information about their influences on several others, e.g. pathogen spread outside hosts or vectors in water or soil; ability of bacteria to multiply as epiphytes or saprophytes; transmission by seed; alternative cultivated or weed reservoir host infection with viruses, bacteria or fungal vectors, or their infestation by arthropod or nematode vectors; and alterations in plant morphology relevant to viral or bacterial infections, or build-up of arthropod vectors. Again, addressing these deficiencies should be an important focus for future research.
8. Very little research has been done on the influence of alterations in relative humidity on viral or bacterial disease. Further research is needed on this, particularly over the influence of altered relative humidity on the activity and survival of arthropod vectors and non-fastidious plant-pathogenic bacteria within the critical leaf surface microclimate.
9. More information is required on the effects of drought and flood on viral and bacterial diseases. For example, studies with different virus pathosystems gave conflicting results regarding the influence of drought, and this situation needs to be resolved. Also, data are needed on the influences of drought and flood on the dissemination of water-borne viruses, and the probability that their epidemics would be mitigated or enhanced.
10. Experiments investigating the effects of diverse climate change parameters on viral and bacterial diseases under controlled environment conditions provide important initial information concerning the likely consequences of climate change scenarios. However, it is important to follow the most significant of these up by examining their effects on epidemics of such diseases in field situations, including in FACE facilities and in crops grown in regions with high temperatures or where flood or drought conditions occur.

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