



Emerging Pathogens and Diseases: Where do they come from?

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INTRODUCTION

The plant disease paradigm is a fundamental principle in plant pathology [1]. For a plant disease to manifest first requires the interaction of a susceptible host, a virulent pathogen, and a favourable environment. Both plants and pathogens evolve in response to each other and this co-evolutionary arms race leads to pathogen emergence and invasion via the colonization of new hosts in native and/or agricultural communities [2].

The extent to how a changing environment, combinations of microbial species and interactions between different plant host species influence the emergence of a “new” disease is poorly understood. Plants are frequently colonised and/or attacked and challenged by an array of beneficial and pathogenic microorganisms [3]. A recent study of the apple flower identified 1600 bacterial species; 26% were unidentified species [4]. Horizontal Gene Transfer (HGT) [5] between bacterial species within a community is also a constant evolutionary driver that defines the individual species within a community. The impact of introducing a new bacterial species into a microbial community, such as the phyllosphere of a plant, or the pathogenicity potential of existing individual species within that microbial community is not well defined.

Following are some brief case studies on the emergence of plant diseases (fire blight, Annual Rye Grass Toxicity (ARGT), Ratoon Stunting Disease (RSD)) caused by phytopathogenic bacteria. Each case study highlights a potential change in the pathogen or plant host and/or environment that has favoured the onset of disease.

Fire blight disease of apple and pears caused by *Erwinia amylovora*

Fire blight is a very serious and most perplexing disease of pear and apple and several ornamental plants in the rose family (*Rosaceae*). During serious blight outbreaks between 1901 and 1904 in the San Joaquin Valley in southern California, 95% of 150,000 pear trees were killed or removed.

Fire blight is a native American disease, as first descriptions were reported from the upper Hudson River valley in New York in the late 1700's. The bacterium was probably present on native rosaceous plants (crab apple, blackberry) and first symptoms were described on early pear and apple orchards planted by the colonists from Western Europe. Spread of the fire blight disease most likely occurred with the planting of apple and pear orchards by the early settlers as they moved westward across North America [6]. During the latter part of the 19th century, the disease reached the west coast of the USA and caused enormous losses wherever apples and pears were planted [7].

Since the early 1900's, fire blight has spread on contaminated sources (budwood, trees, or fruit crates) to four distant island nations: Japan (1903), New Zealand (1919), Bermuda (1938) and England (1957), in addition to Egypt (1964) on the northeast corner of Africa. From the latter two locations, the disease has spread in 40 years to nearly all countries of Europe and the eastern Mediterranean region as far east as Armenia and Iran. To date, fire blight has been officially recorded in 43 countries [8].

Annual Rye Grass Toxicity (ARGT) on grazing animals caused by *Rathayibacter toxicus*

ARGT was first described in South Australia in the 1960's as a disease in sheep and cattle grazing annual ryegrass that was characterized by incoordination, convulsions and death. The disease was a result of a causative nematode-bacterium complex parasitizing annual ryegrass (*Lolium rigidum*) that was grazed by sheep and cattle [9]. Price [10] identified that a nematode (*Anguina funesta*) acted as a 'mechanical vector' for corynebacterium species (*Rathayibacter toxicus*). It was not until 1983 however, that *R. toxicus*, which is widely distributed in Australia, was shown to be the source of the toxin [11]. Most outbreaks occur during summer grazing in the wheat-sheep zones where annual ryegrass is a component of grass pastures [12]. Sporadic reports of ARGT have also been documented in South Africa, with the grass infection possibly introduced from Australia in contaminated ryegrass seed [9].

Annual ryegrass originated in the temperate regions of Europe and Asia and was deliberately introduced into Australia as a pasture species around 1880 [13] and is now widespread and a serious weed of pastures across Australia. The occurrence of ARGT in Australia appears to be largely determined by the geographic distribution of the three organisms: *R. toxicus*, *A. funesta* and *L. rigidum* [14] and the grazing of livestock in gall-infested *L. rigidum* plants. It is likely therefore that this disease of sheep and cattle has arisen by the introduction of both annual rye grass by the early white settlers, presumably to improve pastures, and the livestock animals.

Ratoon Stunting Disease (RSD) of sugarcane caused by *Leifsonia xyli subsp. xyli*

RSD caused by the bacterium, *Leifsonia xyli subsp. xyli*. Evtushenko (Lxx), is highly contagious on cutting implements and is carried in sugarcane (*Saccharum officinarum*) planting material. The bacteria lives in the xylem cells and interferes with water mobility in the plant resulting in a dramatic effect on water use efficiency and nutrient balance and can cause losses from 5-60% [15]. The disease was first recognized in Australia, in the summer of 1944/5 affecting the newly released hybrid variety Q28 [16]. RSD was detected in imported canes in Australian quarantine in the early 1950's [17] and was quickly followed by reports of RSD in a number of sugarcane countries from around the world [18]. Lxx does colonize a number of plant species in the *Saccharum* complex including *S. spontaneum* [19] and it has been hypothesized that *S. spontaneum* is the likely natural host for this bacterium [18].

Modern day sugarcane is the result of genetic back crosses of *S. officinarum* which is derived from *S. robustum* and a number of other *Saccharum* species including *S. spontaneum* [20]. In the early stages of the 20th century a combination of several pests and diseases threatened to collapse the global sugarcane industry [18]. A backcrossing strategy termed "nobilization" that was pioneered by Jeswiet [21], whereby wild *S. spontaneum* clones growing in Java where crossed with *S. officinarum* clones resulted in the POJ breeding lines that were resistant to major diseases, including Sereh disease. Clone POJ2878, also known as the Javan Wondercane, was so successful that it quickly traversed the world, and was adopted wherever cane was commercially grown. It is likely that the global distribution of RSD in the first half of the 20th century is linked with the rapid distribution of the POJ breeding lines and that transmission of Lxx into this progeny from *S. spontaneum* was during the mechanical processes associated with the in-field interspecific hybridization activities [18].

CONCLUSION

The catastrophic outbreaks of the exotic pathogens in an ecosystem are driven by the increase in human population, human interference, the increase in global trade frequency and co-evolution of both host and pathogens. The three examples presented here have resulted from human interference in native ecosystems. The first disease, fire blight, likely emerged due to the introduction of a susceptible host plant (apple and pears) into a new environment (North America) where a bacterial species (*E. amylovora*) was colonising native host plants. The second disease is caused by a native bacterium, *Rathayibacter toxicus* that emerged after the introduction of annual rye grass (*L. rigidum*) and grazing animals. And the third example is the emergence of RSD of sugarcane caused by Lxx which is likely the result of a breeding program between two species of *Saccharum* in the early stages of the 20th century.

The challenge for plant pathologists is to get in front of the plant disease cycle and predict the emergence of new diseases. We understand the push-pull nature of plant disease emergence and prevention. Science does predict change in climates and resultant changes in agricultural production areas. To some extent it is also possible to identify changes in agricultural practices. However, our understanding of the interactions between microbial species and agricultural production systems is poor. It is difficult to measure change without first defining "what is normal". Using metagenomics approaches it is now possible to baseline profile the microflora that colonise plant communities and could be used in prediction models to measure "change". It may be possible to identify high risk scenarios conducive to the emergence of plant pathogens or identify genetic markers that are associated with disease

emergence, and collectively use this information to derive strategies to predict new and emerging bacterial plant diseases.

REFERENCES

- [1] Agrios, G.N., *Academic Press*, **2004**.
- [2] Britton, K. and Liebhold, A.M., *New Phytologist*, **2013**. 197(1): p. 9-10.
- [3] Imam, J., Singh, P.K. and Shukla, P., *Front Microbiol*, **2016**. 7: p. 1488.
- [4] Shade, A., McManus, P.S. and Hendelsman, J., *M Bio*, **2013**. 4(2).
- [5] Koonin, E.V. and Wolf, Y.I., *Nucleic Acids Res*, **2008**. 36(21): p. 6688-6719.
- [6] Bonn, W.G. and van der Zwet, T., *CAB International*, **2000**. 50(3): p. 418-418.
- [7] Burrill, T.J., *Amer Assoc Adv Sci Proc*, **1880**. 29: p. 583-597.
- [8] van der Zwet, T. and Beer, S.V., *U.S. Dept Agric*, **1999**. 631.
- [9] Riley, I.T., et al., *Vet Hum Toxicol*, **2003**. 45: p. 160-162.
- [10] Price, P.C., *Adelaide*, **1973**.
- [11] Payne, A.L., et al., *Toxicon*, **1983**. 3: p. 345-348.
- [12] Jago, M.V. and Culvenor, C.C.J., *Aust Vet J*, **1987**. 64: p. 232-235.
- [13] Kloot, P.M., *Aust J Bot*, **1983**. 31: p. 421-435.
- [14] McKay, A.C. and Ophel, K., *Annu Rev Phytopathol*, **1993**. 31: p.151-167.
- [15] Teakle, D.S., Appleton, J.M. and Steindl, D.R.L., *Physiological Plant Pathology*, **1978**. 12: p. 83-91.
- [16] McDougall, W.A., Steindl, D.R.L. and Elliot, J.T., *Cane Growers' Quarterly Bulletin*, **1948**. 12: p. 31-34.
- [17] King, N.J., *Cane Growers' Quarterly Bulletin*, **1953**. 17: p. 10-13.
- [18] Young, A.J., *Plant Pathology*, **2016**.
- [19] Roach, B., *Sugar Cane*, **1992**. 3: p. 1-11.
- [20] Panje, R.R. and Babu, C.N., *Cytologia*, **1960**. 25: 152-172.
- [21] Jeswiet, J., *Proceedings of the International Society of Sugar Cane Technologists*, **1927**. 2: p. 115-122.