Every ecologist knows the basic history of evolutionary theory—Lamarck’s inability to sway many naturalists with his flawed arguments, and Darwin’s only gradual success with his masterful presentation. Much less well known is the struggle to establish the germ theory of disease, with many defenders and skeptics for well over a century before it gained acceptance. Although the germ theory of disease is narrower in scope than evolutionary theory, its practical importance is at least as great, and therefore its history merits greater familiarity. We saw in part 29 (Egerton 2008) that both mycology and phytopathology made notable advances during the 1700s—with small steps toward a germ theory—though these advances were not widely known or appreciated. Researchers during the 1800s built upon the work of their predecessors. The science of phytopathology began with investigations of fungi, the earliest known culprits, and fungi were already being accused during the 1700s, though there was a hung jury rather than a clear verdict of guilt (Egerton 2008). If guilt was to be established, it was important for botanists to have a standard system to classify what they knew and make clear what was a new discovery when one occurred. Mycology and phytopathology had to advance together. Although John Needham (1713–1781), whom we met in part 24 (Egerton 2007:147), discovered a “worm” (a nematode now named *Anguina tritici*) in wheat galls (Needham 1743:640–641), D. J. Raski (1959:386) dates the beginning of nematology to the *Histoire naturelle des helminthes ou vers intestinaux* (1845) by Felix Dujardin (1801–1860). In 1854–1856, Dujardin’s fellow countryman, Casimir-Joseph Davaine (1812–1882) studied the nematode that causes seed-gall disease in wheat, and in 1868 Davaine also discovered a bacterium that caused a plant disease. Not until the late 1800s were there evidences of a virus as a cause of plant disease. Nature is one, but scientists partition it into sciences they can master. Although phytopathology and animal parasitology developed as separate sciences, their concerns overlap in cases of fungal diseases of animals, including humans, and nematode diseases of plants; and both phytopathology and parasitology overlap with bacteriology and virology concerning bacterial and viral diseases. The history of nematode parasitism of plants was outside the scope of both Large’s (1940) and Ainsworth’s (1981) histories of phytopathology, but nematodes are discussed in textbooks on phytopathology (Heald 1926:831–851, Walker 1969:533–548, Agrios 2005:826–874). With many simultaneous developments occurring, it seems best to discuss studies of fungi first, nematodes and bacteria second and third, and viruses last, in Europe, then in North America.

Europe

The botanist who began to put the fungal house in order was Christiaan Hendrik Persoon (ca.1761–1836), of Dutch heritage, from Cape Town, South Africa (Donk 1974, Ainsworth 1976:255–258, Magnin-Gonze 2004:170–171). In 1775 he went to Germany, where he received his botanical education, and
in 1802 he moved to Paris, where he lived alone, in poverty. He corresponded with many botanists and exchanged specimens with them, exemplified by his correspondence with James E. Smith (Ramsbottom 1934). In 1828 he gave his botanical collections to the Dutch government in exchange for a pension, and when he died he left his remaining collections and his library to the Dutch government.

Persoon published his first article on fungi in 1793 (Schmid 1933), introduced his classification system in 1794, and produced his Synopsis methodica fungorum in 1801, which became the foundation of modern mycological systematics. Geoffrey Ainsworth (1976:258) found that “almost all the hundred genera and subgenera he recognized are universally accepted genera of today.” Persoon brought together the rusts and smuts in the Dermatocarpi, and he named Fontana’s small parasitic plants, brown and black rust on wheat, Uredo linearis and Puccinia graminis (Fig. 2). However, Persoon accepted the prevailing belief that these species could arise from abnormal sap or tissue in the host plant, and his description of P. graminis was vague (Ainsworth 1981:34, 43).

John Ramsbottom (1913:81–84) evaluated Persoon’s classification of the Uredinales, which classification is probably a fair sample of Persoon’s Synopsis. Persoon began a revision of his Synopsis entitled Mycologia Europea (three volumes, 1822–1826), but left it incomplete, no doubt because a
younger rival, Elias Magnus Fries (1794–1878) had begun his authoritative *Systema Mycologicum* (three volumes, 1821–1829 + 2 supplements, 1830–1832).

Fries quite literally followed in the footsteps of Linnaeus: his father was a church vicar from the southern Swedish province of Småland, he studied at the Växjö secondary school, then at the University of Lund, and eventually became Professor of Botany at Uppsala University (Eriksson 1952, 1972, Fries 1952, Ainsworth 1976:259–263). At Lund he studied fungi, but before going there he had independently learned between 300 and 400 species (compared to Linnaeus’ listing of 92 species). He found Persoon’s classification unsatisfactory, and Fries’s *Systema Mycologicum* “did for mycology what Linné did for phanerogamic taxonomy,” with his descriptions being “models of accuracy and conciseness” (Fries 1952:180). His *Systema* is the starting point for fungal names, just as Linnaeus’s names are for higher plants; however, Fries worked without a microscope. He did not confine his studies to fungi, but his other studies are beyond the scope of this discussion. Elias Fries was the first of four generations of Fries botanists.

We met Sir Joseph Banks (1743–1820) in part 43 (Egerton 2012:200) as the botanist with whom Thomas Andrew Knight (1759–1838) corresponded, and who published Knight’s letters in the *Philosophical Transactions of the Royal Society of London*. Banks was president of the Royal Society for 42 years, so he was a well-respected member of the scientific community (Foote 1970, Carter 1987, O’Brien 1997, Knight 2004). In 1805, in response to an extensive outbreak of black rust (*Puccinia graminis*) in 1804 (Ordish 1976:115), Banks published *A Short Account of the Cause of the Disease in Corn, called by Farmers the Blight, the Mildew, and the Rust*, in which he asked, “Is it not more than possible that the parasitic fungus of the barberry and that of wheat are one and the same species, and that the seed transferred from the barberry to the corn [wheat], is one cause of the disease?” (quoted from Ramsbottom 1913:85). He had read Felice Fontana’s *Observazioni sopora la ruggine del grano* (1767), which was little known in
Britain. One would think that Banks also would have read *A Botanical Arrangement of All the Vegetables Naturally Growing in Great Britain* (edition 1, 1776) by William Withering (1841–1899), that claimed the *Berberis* shrub “should never be permitted to grow in corn [grain] lands, for the ears of wheat that grow near it never fill, and its influence in this respect has been known to extend as far as 300 or 400 yards across a field” (quoted from Ramsbottom 1913:81). However, Banks’ claim that “It has long been admitted by farmers, though scarcely credited by botanists, that wheat in the neighbourhood of a barberry bush seldom escapes the Blight” (quoted from Ramsbottom 1913:85) seems to indicate he had not read Withering’s work. Banks’ pamphlet had two interesting enlarged color illustrations by Francis Bauer (1758–1840) of the rust imbedded in the wheat tissue (one reproduced in Ordish 1976: plate 3; see also Ainsworth 1969:15).

Knight had scooped Banks; in 1804 he had already sown wheat around a barberry bush and other wheat from the same source a considerable distance away (Parris 1968:21–22). The wheat near the barberry bush became diseased, as did the bush (quoted from Ramsbottom 1913:86).

*Examining the barberry bush attentively, I found upon its fruit a species of fungus similar in colour to that on the straws of the wheat, but its seed vessels were larger, and more spherical. I was, however, much disposed to believe the parasitical plants of the same species, and that the difference in the form and size of the seed vessels arose only from the difference of the nutriment they derived from the wheat and from the acrid juice of the barberry.*
The distant (control) wheat was healthy until Knight took a diseased barberry branch to that wheat, where he sprinkled the branch with water and brushed it over the healthy wheat, which 10 days later became diseased. Simultaneously, he took some diseased wheat to healthy wheat, moistened the diseased wheat and brushed it over the healthy wheat, but the healthy wheat remained healthy. Ramsbottom (1913:86) suggested that Knight was “the first to try inoculation experiments on heteroecism.” Knight did not write a letter to Banks about this experiment until after he had read Bank’s pamphlet, on 20 March 1806. In 1806, Banks published a second edition of his pamphlet and appended Knight’s letter (Banks 1806:26–36, Ramsbottom 1913:85, Dawson 1958:501).

In Philadelphia, a retired physician and immigrant from Bath, England, Anthony Fothergill (1732?–1813), read a paper on 11 November 1806 to the Philadelphia Society for Promoting Agriculture, arguing that grain rust was caused by “clusters of a fungus or parasitical plant…[that] insinuate themselves into the pores of the absorbent vessels of the stem, and deprive the grain of the sap destined for its nourishment” (1808, quoted from Campbell et al.1999:33). In 1818, a Danish schoolteacher, Schoeler, carried out the Knight experiment with the same results (Parris 1968:24). Knight recommended in 1818 the use of flowers of sulfur against pear scab (*Lycoperdon cancellatum*), and in 1834 he recommended the sprinkling in early spring of peaches with sulfur and lime to control leaf curl. John Roberts recommended in 1821 using soap to cause wetting of sulfur in water to control peach mildew, and in 1824 he reported his successful experimental transmission of peach mildew (Knight 1818, 1842, Roberts 1824, Parris 1968:25–26, Ainsworth 1976:160, 1981:32–33, 110).

Phytopathologist George L. McNew divided the history of his profession into five periods, with periods three and four occurring during the 1800s (McNew 1963:166–172). Period three he labeled “The Predisposition Period,” during which the prevailing view was that fungi originated in diseased plant tissue. Three authors published treatises on plant pathology and remedies in 1807. Freiherr von Werneck and Filippo Ré represented the consensus in not accepting fungi as the cause of the diseases (Whetzel 1918:32, Parris 1968:23, Walker 1969:20–22). Isaac-Bénédict Prévost (1755–1819) did think fungi caused diseases (Keitt 1939a, Large 1940:76–79, Robinson 1975, Ainsworth 1981:30–32). He was the son of a teacher-pastor in Geneva, but at age 22 he became a tutor in Montauban, France, where he remained for the rest of his life. His interests included mathematics, physics, chemistry, biology, and philosophy. In 1797 a member of the Society of Montauban read a memoir on the carie or charbon (bunt or smut) disease of wheat, and the society asked other members to study the problem. Prévost read previous studies on the problem, including those by Duhamel du Monceau (1728) and Mathieu du Tillet (1755), discussed in part 29 (Egerton 2008), and especially Henri Alexandre Tessier’s *Traité des maladies des grains* (Paris, 1783), which attributed rust to stoppage of transpiration, caused by mists. Tessier repeated some of Tillet’s experiments and agreed that bunt was contagious (Parris 1968:19). Tessier also stressed the importance of soaking wheat seeds in lime water before planting, which greatly diminished the loss of wheat from bunt in France (Large 1940:76). The owner of a nearby estate made available to Prévost land for large-scale experiments.

George Keitt (1939b) provided an excellent guide to and evaluation of Prévost’s achievement along with his English translation of the *Mémoire*. Prévost could not discover everything he wished, but he was remarkably thorough and clear in the presentation of his findings. Tillet had demonstrated in 1755...
that bunt (carie, charbon) of wheat was spread by an infectious agent (Egerton 2008:237), but had not demonstrated that the agent was a fungus. Prévost was able to induce the bunt dust to germinate in water and illustrated its growth, thus demonstrating the dust was fungal spores. He illustrated its progressive development on the first of his three plates.

To those believing a fungus arose within the wheat plant, he replied that he only observed branches from the spore growing from the surface into the seedling (1939:34–35, paragraphs 44–46). Fruiting bodies grew within ears of wheat and spores were disseminated by the wind. Prévost scattered spores on soil where wheat seed was planted and concluded that it was only at germination, or soon after, that infection occurred (Prévost 1939:38, paragraph 57). He discovered that copper dust suspended in water,
or liquid copper compounds, inhibited spores from germinating (Prévost 1939:72–82, paragraphs 138–177, Ainsworth 1976:160–161, 1981:124–126), and arsenic was reported in England to have the same effect (Prévost 1939:83, paragraph 178). The Society of Montauban sent a published copy of Prévost’s Mémoire to the Institute [de France?] in Paris for evaluation. Tessier reported for a review committee favorably, but no actions were taken.

Mendel was by no means the only author of a clear publication that failed to gain the recognition deserved upon publication. Lavoisier, Lyell, and Darwin achieved their scientific revolutions in part because of prominence in their scientific communities. Prévost and Mendel were amateurs on the periphery of their scientific communities, who were neglected, rather than being either challenged or widely recognized. Nevertheless, Prévost gave the first experimental proof of the pathogenicity of a microorganism, and he first described motile spores (zoospores) in fungi (Ainsworth 1976:62, 147).

In Italy, Agostino Maria Bassi (1773–1856), like Prévost, was on the periphery of science, but his important parasitical work did attract the attention it deserved, because he promoted it (Belloni 1961:20–26, Robinson 1970, Ainsworth 1976:163–168). He grew up near Pavia and attended its university. He studied law there to please his parents, but his interests were in science, and he studied under Antonio Scarpa, anatomist, Alessandro Volta, physicist, and Giovanni Rasori (1766–1836), pathologist. Rasori (portrait in Belloni 1961:22) wrote a book defending the contagium vivum theory that appeared posthumously in 1837. Both Rasori and Bassi were influenced by the writings of Lazzaro Spallanzani (1729–1799), who discredited the theory of spontaneous generation and accepted the conclusions of Fontana and Targioni-Tozzetti that small parasitic plants can cause diseases in vascular plants (Egerton 2008:235, 239).

The silk industry could flourish in northern Italy and in France, if it could eliminate an epidemic disease, muscardine (calcinaccio). In 1807 Bassi decided to investigate; he grew silkworms, assuming the disease (Bassi 1958:4)

\[ \text{...arose spontaneously in the silk worm and was due to some difference in the atmosphere, the food, or the method of breeding, or rather to the various fumes emanating from the fermenting litter...} \]

He varied environmental conditions for different groups but failed to induce the disease in his caterpillars. When he obtained diseased caterpillars, he found the disease spread at temperatures of 7°–30°R, but not above 38°R (Bassi 1958:8). The disease could be transmitted to caterpillars of other species. It was contagious, because it always began with one or a few caterpillars, whereas an environmental or physiological disease could affect all individuals about the same time. Bodies of dead caterpillars became covered with “a patina or efflorescence like flakes of pure snow” (Bassi 1958:7).

In 1835, Bassi announced that muscardine was caused by a fungus, and he added a footnote (Bassi 1958:10, note on 15)

\[ \text{The eminent and meritorious compilers of the celebrated Giornale Fisico-Chimico, Professors Configliachi and Brugnatelli, were the first to put forward the hypothesis that the mark disease is produced in the silk worm by the development of a species of fungus, basing it on the fungus-} \]
like smell that comes from worms which have died of that disease; and although, in that journal, they appealed to breeders to undertake experiments to test this supposition, these last, perhaps regarding it as the figment of a heated imagination, neglected it, whereas, had they taken the trouble to examine it, they might easily have found, by experiment, that these able men were not mistaken and had really hit on the truth.

Although Bassi blamed the silkworm growers for their indifference to the professors’ suggestion, one may wonder how many growers ever read this physico-chemical journal or even knew how to undertake an experiment. Neither Bassi nor his English editors gave a more precise reference to the professor’s comments than is in this footnote. Nor does Bassi indicate whether his investigation was undertaken in response to their comments, but if he had made his discovery without having read their suggestion, he perhaps would have said so. In another footnote, he stated (Bassi 1958:17)

*If this parasitic plant is observed with the great microscope of the illustrious De Amici, which magnifies the object more than thirty million times (sic.), it will be possible to see all its minutest ramifications in it, and perhaps its reproductory organs as well.*

He explained that the “disease-bearing dust” was spread by air, water, and dogs, cats, rats, mice, flies, and contaminated food for silkworms (Bassi 1958:23). The English translation of Bassi’s treatise is limited to part 1, on theory (1835). In 1836 Bassi published part 2, on ways to combat the disease. These included disinfecting or burning containers and materials used in raising them (Ainsworth and Yarrow 1958:x–xi). Bassi realized that his discoveries would be controversial, and he requested permission to perform his experiments for faculty members at the University of Pavia. They agreed, and afterwards provided a testimonial signed by nine professors, including Pietro Configliachi and Brugnatelli, mentioned in the footnote quoted above. Bassi then published their testimonial in his preface to part 1 (Bassi 1958:1–2). Giuseppe Balsamo-Crivelli named the fungus Bassi discovered *Botrytis Bassiana* (now *Beauveria bassiana*), and an abridged French translation of Bassi’s part 1 was published in Paris in 1836. Both botanist Jean-Francois-Camille Montagne (1784–1866) and entomologist Jean-Victor Audouin (1797–1841) investigated Bassi’s claims for the Académie des Sciences in Paris and published confirmations, with a plate of illustrations of the fungus (Audouin 1836, Montagne 1836, Belloni 1961:25–28; Audouin’s illustration is reproduced by Ainsworth 1976:167). This was the first known fungal parasite of an animal; Prévost had already demonstrated a fungal parasite of plants.

In the early 1840s, two Jewish physicians published evidence of fungal disease in humans. David Gruby (1810–1898) was a Hungarian who obtained his doctorate in Vienna in 1840 and immigrated to Paris, where he practiced and published (Kisch 1954:193–226, Ainsworth 1976:169–171). Although the claim that he was one of the most brilliant biologists of the 1800s (Zakon and Benedek 1944:155) seems an exaggeration, his discoveries were important for both medicine and phytopathology. His six important scientific papers (1841–1844), all rather brief, are translated from French into English (Zakon and Benedek 1944:157–168). In 1841 he published his microscopic observations showing that favus was caused by a fungus; in 1842 he indicated that ringworm of the beard was caused by an ectothrix trichophytosis. He named neither of these fungi, but in 1843 he identified the cause of human microsporosis and named the fungus *Microsporum audouinii* to honor Victor Audouin. Even though his discoveries were easily repeated by any competent microscopist, the Paris medical community remained
skeptical of his conclusions (Zakon and Benedek 1944:156). He also discovered several microscopic invertebrate parasites during the early 1840s (Foster 1965:115–116), before devoting the rest of his life to a very successful medical practice. Robert Remak (1815–1865) was Polish and immigrated to Berlin at age 18. In 1837 he discovered the fungal nature of favic crusts but did not publish this until 1845. He was then a physician in a clinic run by Professor J. L. Schönlein, and Remak named the fungus *Achorion schoenleinii*, honoring Schönlein (Kisch 1954:227–296, Ainsworth 1976:168–169).

Following in Gilbert White’s footsteps as an English clergyman–naturalist, Miles Joseph Berkeley (1803–1889) had a childhood interest in nature that led eventually to his becoming the foremost British mycologist of the 1800s (Massee 1913, Whetzel 1918:55–57, Ramsbottom 1948, Ainsworth 1969:14, Taylor 1970, Staffeu and Cowan 1976–1988, 1:192–195, Desmond 1977:60, Buczacki 1991, Elliott 2004a). He attended Cambridge University, 1821–1825, but left two years before John Stevens Henslow became its professor of botany. As an undergraduate, he collected algae, mosses, and mollusks. Berkeley’s earliest published papers were on mollusks, and he only began to specialize on fungi in 1832, when William Dalton Hooker invited him to write the fungal volume of *The English Flora* (Berkeley...
Contribution

1836). Berkeley’s introduction to plant pathology occurred in 1845, when the potato murrain, that had been discovered in Liège, Belgium in 1842, and occurred in the eastern United States in 1843 (Stevens 1933, Campbell et al. 1999:38–39) and in Western Europe in 1844, then spread to Britain and Ireland (Berkeley 1948:14–17, Bourke 1964, 1969). When its seriousness in Ireland became evident, British Prime Minister Sir Robert Peel formed a scientific commission to study the matter (Large 1940:26–27, Woodham-Smith 1962:44–47), under London Botany Professor John Lindley (1799–1865; Stearn 1999) and London Chemistry Professor Lyon Playfair (1818–1898), who had received his Ph.D. under Liebig (Large 1940:26–27, Woodham-Smith 1962:44–47). The commission went to Ireland but did not find a clear cause of the disease.

Montagne (mentioned above) found a fungus associated with blighted potatoes, which he named *Botrytis infestans* (now *Phytophthora infestans*) on 30 August 1845 (portrait in Virville 1954:198; Ainsworth 1976:154–155, 1981:54–55). He was uncertain about whether the fungus caused the murrain, but sent his comments and drawings to his regular correspondent, Berkeley (Lamy 1989), who, at first (1845) shared Montagne’s uncertainty (Walker 1969:23). However, Dr. C. F. A. Morren, head of a school of agriculture in Liège, believed the fungus caused the murrain (1845), and he conducted crude inoculation experiments to support the claim (Walker 1969:23). Berkeley agreed with Morren before publishing his own study on the disease (1846).
Berkeley also published Montagne’s drawings (one of which is in Fig. 7). Berkeley presented the case for the disease having an environmental cause and cited the authorities who supported this theory (1948:23–28), and then he argued the case for “The decay is the consequence of the presence of the mould, and not the mould of the decay” (Berkeley 1948:28). He ended his article (Berkeley 1948:35–37) with a Latin and English description of Botrytis infestans Mont. and related species (many of which, like infestans, are no longer placed in that genus). Large (1940:32) comments that Berkeley’s article swept away many but not all objections. Berkeley did not observe spore germination, nor did he document the actual infection of the potato plants.

Berkeley became both mycologist and plant pathologist, and he entered the fray in 1847 over the cause of a new grapevine disease, discovered by a gardener, Tucker, in 1845 near Margate, England. Tucker reported that he controlled this powdery mildew with a mixture of sulfur and lime in cold water. Berkeley studied the mildew, named it Oidium Tuckeri, and illustrated it with hyphae growing within the leaves, as he had drawn B. infestans. The French physician–mycologist Joseph-Henri Lévillé (1796–1870, portrait in Virille 1954:218) did not accept the claim that O. Tuckeri caused the downy mildew, and he found no hyphae within the leaf tissue, only on the leaf surface (Large 1940:44–45). Berkeley translated Lévillé’s discussion and added his own response (Lévillé and Berkeley 1851). Lévillé was right about O. Tuckeri not having any detectable hypha within the leaf tissue, but wrong in thinking that morbid tissue gave rise to the fungus. In 1851, Dr. Zanardine, in Venice, argued that the surface fungus, O. Tuckeri, was indeed parasitic, because it had little suckers he called “fulcra” (now “haustoria”) that obtained sustenance from leaves. Skeptics could still argue that the fungus may not have arisen if the leaf was not already moribund, but the consensus was shifting toward Berkeley.
Contributions

(Large 1940:44–49). Berkeley published 173 articles on “Vegetable Pathology” in the Gardner’s Chronicle, 1854–1857, some of which are reprinted (Berkeley 1948:41–108). He also published a steady stream of articles on fungi: 35 articles on 2050 British fungi, 1837–1885; 7 of these articles are only his and 28 were coauthored by C. E. Broome; all are now reprinted (Berkeley and Broome 1967). In addition, he was one of three European mycologists who described fungi brought back from elsewhere in the world, the other two being Montagne in France and Elias Magnus Fries (1794–1878) at Uppsala University, Sweden, whose Systema mycologicum (three volumes, 1821–1832) is recognized as one of the two foundations of modern fungal nomenclature, along with Persoon’s Synopsis (Fries 1950:61–63 + portrait on frontispiece, Eriksson 1972, Ainsworth 1976:273). Berkeley published his descriptions of foreign fungi in batches of 10 descriptions per article (or at least 10 in the first article, and various numbers after that, but all called “decades” nevertheless), for 27 articles, 1844–1856, three of which were coauthored, and all of which are now reprinted (Berkeley 1969). Berkeley was a careful observer but was not an experimentalist.

(Heinrich) Anton de Bary (1831–1888) was the leading physiological mycologist and a leading phytopathologist of the 1800s and was an outstanding experimentalist (Whetzel 1918:45–47, Jost 1930, Robinson 1971, Sparrow 1978). He was also one of the most respected and revered professors in that century. He was the son of a physician in Frankfurt am Main who raised fruit trees and flowers, and as a teenager Anton joined a local group of amateur naturalists on field trips into the nearby countryside. A physician–botanist at the Senckenberg Medical Institute, Professor Georg Fresenius, introduced him to the study of algae and fungi. After graduating from the Frankfurt Gymnasium (high school) in 1848, de Bary studied medicine at Heidelberg, Marburg, and Berlin, receiving his M.D. degree in 1853. He had published his first scientific paper in 1852, on the water mould Achlya prolifera, from a peat bog near Berlin (illustration in Sparrow 1978:234). In 1853 he published his M.D. dissertation on the sexuality of plants and a book on fungal rusts and smuts. Berkeley had published strong evidence that the potato murrain was caused by a fungus, but without convincing all skeptics. De Bary’s Untersuchungen über die Brandpilze und die durch sie verursachten Krankheiten der Pflanzen (1853) undermined the remaining doubts that fungi do cause diseases. McNew’s fourth period of phytopathology is “The Etiological Period,” which began with de Bary’s Untersuchungen (McNew 1963:167–168).

De Bary’s “phytopathological classic” is now translated into English (De Bary 1969), and one can see why it ended doubts on disease causation. It is divided into three parts: I. Specific observations concerning the form and development of the brand fungi; II. Systematic conclusion; III. Concerning the relationship of the brand fungi to the brand and rust diseases of plants. By describing various life cycles (I), he provided the basis for a new classification (II), which enabled botanists to discuss specific fungi in relation to specific vascular plant hosts. Paradoxically, botanists who believed the fungi were products of disease rather than the cause—he provided extensive citations in his notes (De Bary 1969:64–67)—still thought of the fungi as distinct species. In contrast, he listed only 10 botanists who were convinced that fungi were the cause of disease (De Bary 1969:68). De Bary pointed out that one can observe the germination of spores and show their capacity to infect, the hereditary nature of the brand fungi, and their relation to stomata of plants they infect. He also provided eight plates showing fungi in relation to their hosts. Any remaining skeptics would have to show how the leading authority on these fungi was wrong. None tried it.
He returned to Frankfurt to practice medicine and realized he was interested in diagnosing illnesses, but not in curing patients, and he lost interest in being a physician. In 1854 he discovered that a myxomycete spore gave rise to a naked flagellated myxamoeba, not a hypha, and later he postulated that myxamoebas fuse to form a plasmodium (Sparrow 1978:226).

De Bary attended a botany meeting at the University of Tübingen, where Professor Hugo von Mohl helped him obtain a faculty position at the University of Freiburg im Breisgau, where he taught, 1855–1866, and established the first teaching botanical laboratory. In 1866 he moved to the University of Halle, where he expected to develop a botanical institute, which only materialized in his last year there, 1871–1872. After the German victory in the Franco-Prussian War, he moved in 1872 to the University of Strasbourg and was elected the rector of the university. In October 1872, the first American who arrived there to study with him found that university enrollment had shrunk from a prewar 1500 to 250, and there were only three other botany students (Harris 1945:12–13). However, de Bary soon became head of a fine botanical institute, and the building he had built for it was so spectacular that a later American student wrote an article on it with two drawings and two floor plans and a drawing of its greenhouse (Dudley 1888; photograph of institute in Sparrow 1978:248). De Bary excelled at leading field trips and guiding laboratory research and the writing of dissertations (Ayres 2005:42–49). He advised 94 doctoral students, with dissertations mainly in mycology, but some on other cryptogamic species (students listed

Fig. 8. Fertilization and overwintering stage of *Cystopus candidus* Lev. fungus causing white rust of cabbage and mustard. De Bary 1863, from Large 1940:102.
in Sparrow 1978:245). He had his students study a species’ life cycle. He ended the debates about the cause of late blight in potatoes (1861) and the heteroecious life cycle of wheat–barberry rust (1863). He published the first textbook on the morphology and physiology of fungi, lichens, and myxomycetes (1866, revised and enlarged edition, 1884). His achievements in mycology and phytopathology are too numerous to list them all here, but Ayres devotes an excellent chapter to de Bary, which includes a succinct outline of his achievements (Ayres 2005:44). De Bary also played an important role in lichenology (Mitchell 2012), to be discussed in part 52 of this history, on symbiosis.

The other founder of modern plant pathology was Julius Kühn (or Kühn, 1825–1910), who wrote the first modern textbook on it (Whetzel 1918:47–53, Large 1941:93–94, Wilhelm and Tietz 1978, Ainsworth 1981:36–38). He was a remarkable scientist because he was largely self-taught. He was from Pulsnitz, Germany, the son of a landowner. He began his career, 1848–1855, as manager of a 750-ha estate in Silesia. In 1850 he wrote to a prominent botanist, Matthias Jakob Schleiden (1804–1881), who is remembered as a founder of the cell theory. Kühn was concerned about a beet disease, which Schleiden thought was caused by unbalanced nutrition (Wilhelm and Tietz 1978:351). In 1851 he had
more luck when he wrote to G. Ludwig Rabenhorst (1806–1881), who later became the founding author of *Kryptogamen-Flora von Deutschland, Oesterreich und der Schweiz* (11 volumes, 1884–1960). Kühn sent him an alga that plugged his field drains, which turned out to be a new species Rabenhorst named *Leptothrix kuehneana* (which did not, of course, unclog the drains). That began a valued correspondence that lasted for many years. Kühn published three of his early scientific papers in Rabenhorst’s journal, *Hedwigia* (1855–1858).
In 1856 Kuehn published two articles on the life cycle of *Claviceps purpurea*, a parasite that causes ergot in rye grains and causes ergotism in animals and humans that is often fatal. His biographers (Wilhelm and Tietz 1978:353) think that the article in the Leipzig weekly, *Agronomische Zeitung*, was a prototype for his textbook, *Die Krankheiten der Kulturgewaechse* (1858). The article refuted Schleiden’s theory that cultivated plants have a disposition to disease, using Kuehn’s inoculation of wild grasses with spores as evidence. In 1855–1856, he attended the Agricultural Academy at Bonn-Poppelsdorf, and in 1857 the University of Leipzig awarded him a Ph.D. on the basis of his published papers. He then managed another Silesian estate for five years, and then his textbook enabled him to obtain a new agricultural chair at the University of Halle in 1862, where he remained for the rest of his career. He taught some 5000 students at his Agricultural Institute, though only one of them became a phytopathologist, Reinhold Wolff, in 1873.

The Tulasne brothers, Louis-René (1815–1885) and Charles (1816–1884), collaborated on the study of fungi, after an inheritance in 1839 freed the former from the study of law and the latter from the study of medicine (Ainsworth 1976:26–28, Viennot-Bourgin 1976,). Louis attended botanical lectures in Paris and then became an assistant naturalist at the Muséum d’Histoire Naturelle, and Charles became an outstanding botanical illustrator. Berkeley had demonstrated that there are two fruiting forms in four genera of Gasteromycetes, which Louis confirmed and expanded in a series of 10 memoirs; the last of these memoirs, *Fungi hypogaei* (1851), “remains one of the foundations of the modern study of this group” (Viennot-Bourgin 1976). In his second memoir on Uredinales (1854), Louis showed that *Uredo* and *Phragmidium* represent different stages of the same species (Ramsbottom 1913:88–89). The brothers’ major contribution to mycology was “the establishment of pleomorphism among fungi by a wealth of accurate and detailed observation, first in a series of papers dealing with pyrenomycetes, discomycetes, and basidiomycetes published between 1851 and 1860” (Ainsworth 1976:28). The climax of their work was a magnificent *Selecta fungorum carpologia* (three volumes, 1861–1865, English version 1931).

Another English amateur botanist who rose to professional prominence was Mordecai Cubitt Cooke (1825–1914), but unlike Berkeley, he lacked a university education, though he did take botany courses at the Science and Art Department at South Kensington (Ramsbottom 1915:172). He held a variety of jobs, including teaching and museum work. In 1862 he helped found The Society of Amateur Botanists and became its only president. In 1865 he became editor of the new *Hardwicke’s Science Gossip*, and a reader suggested establishing an amateur microscopical group, since the Royal Microscopical Society did not meet amateur needs. This led to the formation of the Quekett Microscopical Club, and Cooke was also asked to become its president, but declined. He successfully urged the club to adopt the French metric system of measurements. In 1861 he published an introductory botany textbook, the first of some 32 books he would publish, all on botanical topics, excepting one on reptiles and amphibians (Ramsbottom 1915:175–176, Freeman 1980:91–93). Cooke’s *Rust, Smut, Mildew, and Mould: An Introduction to the Study of Microscopic Fungi* (1865) was possibly the first book on plant diseases written for a popular audience, with 12 color and 4 black and white plates, containing 269 figures drawn by John Edward Sowerby (Fig. 2). As mentioned above, Berkeley described 2050 species of British fungi; Cooke, in his *Handbook of British Fungi* (1871), described 2810 species. He gave up editing *Hardwicke’s Science Gossip* in 1872 to found *Grevillea*, devoted to cryptogamic botany, which he edited until 1892. In 1887 the Royal Botanic Garden, Kew bought his herbarium of 46,000 specimens and 22,000 drawings. “Cooke had a greater influence on the study of fungi than any other Englishman with the possible exception of Berkeley” (Ramsbottom 1915:184).
Harry Marshall Ward (1854–1906) was one of de Bray’s students. As a teenager, he joined the Nottingham Naturalists’ Society. In 1872 he attended the Science School in London, founded in that year and later renamed Royal College of Science (Thiselton-Dyer 1913, Desmond 1977:639, Junnker 2004, Reisz 2004, Ayres 2005:27–29). He attended Cambridge University, 1876–1879, then went to Würzburg to study plant physiology under Julius Sachs, where he met Francis Darwin, who went with him to visit de Bary’s laboratory in Strasbourg (Ayres 2005:41). Rather brief training at Strasbourg qualified him to be a British government cryptogamist, sent in early 1880 to Ceylon (Sri Lanka) to study coffee leaf disease. The disease, which caused the loss of leaves, had been discovered on a single estate in 1869, but had since spread rather widely. George Thwaites, Director of the Royal Botanic Garden, Peradeniya, had sent infected leaves to Berkeley, who described from them a new genus and species, Hemileia vastatrix (Ayres 2005:1–5). Ward used both de Bary’s methods and the technique of Dr. Charles H. Blackley to study hay fever (1873), of hanging sticky microscope slides among coffee leaves to collect spores.
Ward succeeded in two years in describing the life cycle of *H. vastatrix*, but he was unable to find an effective means to end the epidemic, and coffee declined as a crop in Ceylon (McCook 2006:178–183, 2011:96–100). After leaving Ceylon, Ward returned to Strasbourg for two months of further study under de Bray. After returning to England, he taught at Owen College, Manchester and became active in the British Association for the Advancement of Science. In 1885 he became professor of botany at the Royal Indian Engineering College.

Ward’s major contribution was establishing physiological plant pathology (Ayres 2005:39), though he built upon de Bary’s work. “A Lily Disease” (Ward 1888), his most important paper, focused on controlling disease by studying the interactions between plants, parasites, and environment. The fungus secreted something from the tips of hyphae that broke down host cell walls. Kuehn had coined the word “enzyme” in 1867, yet in 1888 there was no agreement on its meaning (Ayres 2005:80–81). Ward saw two patterns of fungal attack: (1) biotrophic pathogens, such as rusts, that caused minimal disruption to host, because enzymes released by hyphae enabled tips to grow into the cell wall, with host cells living longer, and (2) necrotrophic pathogens, such as *Botrytis*, that released pectic enzymes disrupting cells, allowing hyphae to grow rapidly, but killed host cells. *Botrytis* had been regarded as saprophytic, but Ward showed it could also become parasitic. Ward spoke of ferments or enzymes (1888:365). Despite the article’s importance, a modern scientific journal editor would likely ask the author of a 60-page article of this sort for a much more concise presentation of evidence.

Francis Darwin had been lecturing on botany at Cambridge University since 1884, but when the chair became vacant in 1895, he supported Ward for it. Ward received the position and remained active as teacher, administrator, and researcher.

Casimir-Joseph Davaine (1812–1882), according to a Canadian phytopathologist (Estey 1975:549), was “one of the most remarkable biologists of the nineteenth century,” with important contributions to phytopathology, parasitology, and microbiology. He was the son of a distiller in St.-Amand-les-Eaux and was educated in northwestern France before going to Paris to study medicine. He never held an academic position, but did research while practicing medicine in Paris, beginning in 1838 (Théodoridès 1968, 1971). His research, 1854–1856, on the nematode *Anguina tritici*, which caused seed-gall disease in wheat, was his “most comprehensive achievement in plant pathology” (Estey 1975:551) and the first documented nematode disease in plants. In 1856, the Académie des Sciences awarded him 1500 francs for his nematode studies. He was also first to document a plant disease caused by bacteria, which he transmitted from one plant to another by inoculation (Davaine 1868a, translated in Estey 1975:549).

Among the succulent plants, or the vegetables with very tender and moist parenchyma, many times I have seen a change that first appeared at the root and shortly invaded the rest of the plant completely destroying it in a few days. This change, which reduced the tissues to a kind of rot, is caused by the development of bacteria that differ from those of anthrax in as much as they have movement. We can easily transmit this disease from one plant to another by inoculation: around the inoculation point an oil-like spot appears that grows and takes over the whole plant if the diseased part is not cut off.

Davaine knew that Pasteur heated wine to eliminate contaminating microorganisms, and therefore he tried, successfully, to kill bacteria without killing the plant, at a temperature slightly above 52°C.
(Davaine 1868b). On another occasion, Pasteur acknowledged following Davaine’s lead.

Mikhail Stepanovich Voronin (Michael Stephanovitch Woronin, 1838–1903) was the founder of Russian phytopathology (Chupp 1934, Senchenkova 1976). He was born into a wealthy family and studied geology and botany at St. Petersburg University. He graduated in 1858 and went to Germany for two years and studied under Holle in Heidelberg and de Bary in Freiburg, studying both algae and fungi. Being independently wealthy, he returned to St. Petersburg and established his own laboratory and only taught courses on mycology at the university in 1869–1870 and 1873–1875. He gave money to the St. Petersburg University to build its Botanical Institute and subsidized other botanical projects. Novorossysk University in Odessa awarded him an honorary doctorate in 1874, and in 1898 he was elected a member of the Russian Academy of Sciences and headed its cryptogamous plants section.

Of Voronin’s 106 research publications, the most important for phytopathology were “Research on...
Contributions

the Development of the Rust Fungus *Puccinia helianthi*, which Causes the Sunflower Disease” (1871), “Organism Causing the Disease of Cabbage Known as Kila” (1877), and “On “Drunken Bread” in the Southern Ussuri Region” (1890). A collected edition of his most important writings was published in 1961. In his study on ergotism from contaminated rye bread, he identified 15 fungi, 4 of which caused the disease. He identified the cause of cabbage clubroot as a slime mold he named *Plasmodiophora brassicae*, and his study on it (1877) is a classic, translated into English (1934).

Since fully-grown cabbage plants could get clubroot, all stages of growth were susceptible. The growths were variable, as seen in two illustrations of whole roots (Fig. 12). He also provided four microscopic drawings showing fungus and cabbage tissue.

The number of spores produced per fungus was unusually large, and their size unusually small. They were released into the soil as the infected cabbage roots disintegrated. The spores burst and myxamoeba
oozed out and with whip-like cilia moved through soil water in quest of root hairs or root epidermal cells. It had no cell wall, and Voronin did not even find a cell membrane. Knowledge of its life history enabled Voronin to suggest a remedy: after cabbage was harvested, roots had been left in the ground; they needed to be dug up and burned. Cabbage seeds were grown in beds and then seedlings transplanted into fields. The seedlings should be carefully inspected to eliminate any sickly ones being transplanted. Finally, cabbage should be grown on a rotational pattern—never the same field in consecutive years (Voronin 1934:23–28).

Simultaneous with awareness of bacterial plant disease was awareness of viral plant disease (Johnson 1942, Waterson and Wilkinson 1978:23–31). By the mid-1800s, tobacco was an important crop in The Netherlands, and in 1857 a college student working on a farm discovered an unknown disease that eventually was made known to the German director of the agricultural experiment station at Wageningen, Adolf Mayer (b. 1843; Fig. 14a). Mayer studied it for several years, named it tobacco mosaic disease, illustrated affected leaves in color, ruled out a nutritional disease, but was unable to find either fungi or nematodes in diseased plants. However, he found that substance from diseased plants ground up and mixed with water would infect healthy plants, yet using Robert Koch’s methods, he could not isolate bacteria. He filtered the infective fluid through filter paper, and it was still infective, but when filtered through double filter paper it was not. Heating the infective fluid at 80°C for several hours killed its infectiveness. He therefore concluded that it was likely a bacterial disease (Mayer 1886, 1942).

Dmitri Iosifovich Ivanovsky (D. Ivanovski 1864–1921) (Ivanovski 1942) was an undergraduate at St. Petersburg University in 1887, when he and another student were sent to Ukraine and Bessarabia to study wildfire disease at tobacco plantations (Gutina 1973). He concluded it was not contagious, and received his degree in 1888. In 1890 another disease appeared in tobacco plantations of the Crimea, and the Department of Agriculture sent him to study it. He thought that what Mayer interpreted as two stages of tobacco mosaic disease were actually two different diseases. Mayer seems to have been correct on this point, but what impressed other scientists was that Ivanovsky...
found that the sap of infected plants was still potent after it had been filtered through Chamberland filter-candles (Ivanovsky 1942). Like Mayer, he thought it likely was a bacterial disease.

Martinus Willem Beijerinck (1851–1931) was the son of a tobacco dealer who went bankrupt (Hughes 1978, Bos and Theunissen 1995). At the Delft Polytechnic School, his roommate was Jacobus van’t Hoff, who won the first Nobel Prize for Chemistry in 1901. Beijerinck received a diploma in chemical engineering in 1872, then switched in graduate school at the University of Leiden to botany and in 1877 received his Ph.D. with a dissertation on plant galls. He held several positions before returning to Delft Polytechnic as a professor in 1895, where he became a popular teacher and active researcher (Williams 1960:73–87). His investigations in microbiology were quite diverse.

Beijerinck began teaching at the Agricultural School in Wageningen in 1876 and was a colleague of Adolf Mayer, who showed him his experiments on tobacco mosaic disease in 1885. When Mayer was unable to find a bacterium causing the disease, he went on to other things. Beijerinck (1898, 1899) reasoned that the cause might be a liquid poison from a bacterium like tetanus, where the bacteria might not be located at the site of the symptoms, and so not be discovered there (Beijerinck 1942:33–34). However, diligent search did not discover them elsewhere in the plant, and he concluded that the disease was not caused by microbes, but by a “contagium vivum fluidum.” However, that term was too long and he soon called it “virus,” which was the first use of this term for this substance (Johnson 1942:6). It remained infectious when filtered through porcelain that could remove bacteria. A very small amount of the filtered virus could cause the infection of numerous tobacco leaves, which would not have occurred with a small amount of toxin such as from tetanus bacteria. He was able to grow this virus on agar plates and then infect plants with virus grown beyond the point of inoculation (Beijerinck 1942:35–36). His curiosity exceeded Mayer’s, for he found that the virus can be dried without loss of strength and that it could survive the winter in the soil. The virus was destroyed by boiling and even at 90°C (Beijerinck 1942:42–44). Since Erwin Smith (see below) had not found a bacterium that caused peach yellows in 1894, Beijerinck suspected that it also was caused by a virus, which Walker (1969:610) thought was correct, but Campbell 1983:23) says it is caused by a mycoplasma.

In 1894, a privately funded Willie Commelin Scholten Phytopathology Laboratory was organized in Amsterdam, and in 1895 Jan Ritzema Bos (b. 1850) became director for a decade (Faasse 2008). Bos had been a professor at the Agricultural School in Wageningen, where he had developed a strong relationship with farmers concerning plant diseases. Also in 1895 the Laboratory began publishing Tijdschrift over Plantenzieken.

North America

Original contributions to phytopathology from North America were sparse and minor before the U.S. Civil War (Stevenson 1959). In New York State, pomologist and agricultural editor John J. Thomas published an article on “The Diseases and Insects Injurious to the Wheat Crop” (1844) in which he drew upon the European literature to argue that fungi cause smuts and rusts, and he used a microscope to see “a small plant of as regular and uniform a growth as the wheat itself” (quoted from Campbell et al. 1999:34). America’s potato crop suffered from a blight in 1843, and in 1844 James Teschemacher (1790–1853), an English immigrant to Boston, who published papers on several different sciences, wrote
in a local agricultural journal that the blight was caused by a fungus, and he had used a microscope to see its spores. He placed a diseased potato beside a healthy one under a bell jar and also buried a diseased potato beside a healthy one and found the healthy ones still healthy after five days, but diseased after two weeks (Peterson et al. 1992, Campbell et al. 1999:42–44). In 1847, Samuel Gookins reported to an American horticultural and farming journal that Ruben Ragan, at his Indiana farm, had shown Gookins where he had inoculated a healthy pear tree with sap from a pear tree that had fire blight, and some days later the healthy tree showed signs of infection where it had been inoculated (Baker 1971:611–612, Campbell et al. 1999:56). Although Burrill would argue in 1879 that fire blight is caused by bacteria (see below), before that time it was often assumed that all contagious plant diseases were caused by fungi. By the 1850’s, America was gaining converts to the fungal theory of plant diseases (Campbell et al. 1999:57–59), and an influential Englishman (Berkley 1948:15) cited Teschemacher’s observations.

Although the Civil War in the United States was a setback to higher education, since young men went off to war, the secession of the South enabled Congress to make significant progress, because sectional strife within Congress ended. In 1862 Congress passed and President Lincoln signed bills creating both the U.S. Department of Agriculture (USDA) and land grant colleges in every state, which would provide agricultural education that included phytopathology (Elder 1962, Stefferud 1962a, b). Although a federal bureaucracy was formed and a USDA building completed in 1868 (True 1937:41–48), there was no progress in phytopathology during the 1860s either in Washington or in the states, but by the 1870s both the USDA and state colleges became important for phytopathology in the United States and eventually in the world (Baker et al. 1963, Campbell et al. 1999:62–65). In 1871, the USDA hired Thomas Taylor (b. 1820), who had designed and tested ordnance for the War Department during the Civil War, to man the Department’s one microscope to study plant diseases. He quickly accepted the fungal theory of disease for grape, pear, peach, and lilac diseases and published annual reports on these and other diseases, 1872–1877, but by the late 1870s, he was in competition with professionally trained mycologists and he moved into the study of food adulteration (Stevenson 1959:19, Campbell et al. 1999:129–133, 407). In 1885, a botany committee from the American Association for the Advancement of Science petitioned the new Commissioner of Agriculture, Norman J. Coleman, urging him to expand the research of USDA to include the study of plant diseases. He supported the idea, and in turn urged academic botanists to lobby Congress to appropriate funds for a Section of Mycology in USDA’s Division of Botany. Botanists flooded Congress with letters, funds were appropriated, and Frank Lamson-Scribner became head of mycology (Stevenson 1959:20, Campbell et al. 1999:136–139). Lamson-Scribner then wrote to Bessey for guidance in establishing priorities for his research (Griffith et al. 1994).

Yale graduate Samuel William Johnson (1830–1909) became Liebig’s outstanding American student, 1854–1855; he returned to teach agricultural chemistry at Yale (Rossiter 1975:127–148). Johnson embarked on a crusade to convince states to establish agricultural experiment stations (Rossiter 1975:149–171), and Connecticut did establish the first such station in 1876, and by 1880 California, New Jersey, and North Carolina had followed suit. However, it was a struggle to keep them funded, and in 1887 Congress passed the Hatch Act to provide federal funds for state experiment stations (Byrd 1962, Knoblauch and others 1962, Marcus 1985). In the 1890s, these stations began to make very substantial research on plant diseases (True 1937, Campbell et al. 1999:181–203).

In 1872, William Gilson Farlow (1844–1919), a Harvard M.D. and Professor Asa Gray’s assistant,
was the first American to go to Strasbourg to study mycology under de Bary (Setchell 1927, Harris 1945). Farlow’s father was a successful Boston businessman and state legislator, and he could afford to send home a trunk full of books, including the Tulasnes’ *Carpologia Selecta Fungorum*, which he commented to Gray was expensive (Harris 1945:18). When Farlow returned home in 1874, he became assistant professor of botany at Harvard’s Bussey Institution, a school of agriculture and horticulture founded in 1870 by the will of philanthropist Benjamin Bussey. While there, Farlow conducted research on economically important plant diseases (Setchell 1927:5–6, Campbell et al. 1999:93–98 et passim, Peterson 1999), though when he became a professor of botany at the Harvard campus in 1879, he switched to mycological research. Nevertheless, he did train phytopathologists. His student and former assistant, Roland Thaxter (1858–1932), served three years as a phytopathologist at the Connecticut Agricultural Experiment Station, 1888–1891, before returning to Harvard to teach cryptogamic botany (Weston 1933, Clinton 1936, Horsfall 1963, Lamb 1976).

Charles Edwin Bessey (1845–1915), son of an Ohio teacher, received his B.S. degree from Michigan Agricultural College in 1869 and in 1870 became professor of natural history at Iowa State College of Agriculture at Ames, to teach botany and horticulture (Pool 1915, Ewan 1970, Overfield 1975, 1992,
Campbell et al. 1999:98–105, Shor 1999). He spent two winter vacations of three months each studying botany under Asa Gray at Harvard and received an M.S. degree from his alma mater in 1872. Iowa State College granted him a Ph.D. degree in 1879 based upon his botanical publications. In 1884 he became professor of botany at the University of Nebraska in Lincoln, where he remained for the rest of his career. Bessey developed strong ties with both the botanical profession and investigators of plant diseases. He followed the teachings of de Bray and began publishing on parasitic fungi in 1877, and continued doing so through 1903 (Pool 1915:513–516). However, he took all botany as his domain, and he dominated botany in America by editing journals, publishing scientific papers and botany textbooks for high schools and colleges, and by training other botanists. One of his three sons, Ernst Athearn Bessey, became an internationally known mycologist (Bessey 1955).

Davaine’s reports of a bacterial plant disease were clear and succinct, but went unnoticed in Germany and the United States. Thomas Jonathan Burrill (1839–1916), a farmer’s son, who was born in Massachusetts and grew up in Michigan, graduated from the State University of Illinois in 1865,
In 1867 he was botanist for John Wesley Powell’s first expedition to the Colorado Rocky Mountains (Ewan and Ewan 1981:33), and in 1868 he began teaching at Illinois Industrial University (in 1886 renamed University of Illinois) and soon became professor of botany and horticulture. By 1874, he introduced plant pathology into his courses and his publications. Fire blight, as noted above, was known to be contagious by the 1840s but assumed to be caused by a fungus. That was still being assumed by Burrill in his first two papers on fire blight in 1877 and 1878, but in 1879 he cautiously suggested that the bacteria he found in infected plant fluid caused the disease (Clark 1961:249–250, Baker 1971:614–615, Ainsworth 1981:64–65, Campbell et al. 1999:107–116). In 1882, he named the bacterium *Micrococcus amylovorus* (now *Erwinia amylovora*). Others confirmed his results (Matta 2007:198–199). For all this and much more, including 17 articles listed in their bibliography, Campbell et al. (1999:109, 376–377) call Burrill “father of American plant pathology.”

Bacteriology was pursued for its relevance to both plant and animal diseases, and both pathologies benefited from that. For example, Dr. George M. Sternberg (1838–1915), Surgeon-General of the U.S. Army, published his own translation of a French textbook of bacteriology by Antoine Magnin (1880), the first such textbook published in America (Clark 1961:51–52). He published an enlarged edition in 1884, and in 1892 published his own *Manual of Bacteriology*.

Burrill’s contributions to phytopathology were soon eclipsed by his younger contemporary, Erwin Frink Smith (1854–1927). Smith grew up in Gilberts Mills, New York, near Syracuse, son of a tanner and shoemaker. He had an early interest in fishing and in ants and was an avid reader (Rodgers 1952:1–5, Clark 1961:228–232, Aycock 1975, Ainsworth 1981:66–70, Wolf 1999, Matta 2007:202–205). In 1870 his father bought a farm near Hubbardston, Michigan. In that town he met the druggist and postmaster Charles F. Wheeler, who was interested in botany. Smith did not graduate from high school until 1881, and in the same year he was junior coauthor with Wheeler of *The Flora of Michigan*, which described 1634 species (True 1927, Rodgers 1952:18, Campbell 1983:23). He graduated from the University of Michigan in 1886, and in 1889 received his Ph.D., with a dissertation on the peach yellow disease. He began work in the USDA Section of Mycology in 1886 and remained there for the rest of his career. He became the world’s leading authority on bacterial plant diseases, though most of his renown came during the 1900s, beyond the limits of this survey. In 1897–1901 he engaged in a well-known debate with German botanist Alfred Fischer (1858–1913) about whether there were any bacterial diseases in plants. Fischer had studied under Sachs and de Bary and was more physiologist than pathologist (Matta 2007:207–212). That debate is now reprinted in English (Campbell 1981). Fischer might have suspected he was skating on thin ice when he acknowledged in 1897 that nonpathological bacteria occur in the roots of Leguminosae (Campbell 1981:1). Voronin had published his discovery of this in 1866 (Baker 1971:613, 633). Despite that, Fischer claimed that pathological bacteria could not penetrate plant epidermal cell walls. Dismissively, he noted: “new descriptions of plant diseases caused by bacteria keep springing up and, truly, what worthless descriptions and what non-critical trials.” Smith, in the *Centralblatt für Bakteriologie*, replied in kind: “It is seldom in a genuinely scientific book that one finds so many unwarranted assumptions and serious misstatements in the space of a single page” (Campbell 1981:4) as in Fischer’s textbook on bacteria. Smith reported that eight plant diseases were attributed to bacteria, and that six of them were established beyond a reasonable doubt; he furnished bibliographic citations to the literature on those six (Campbell 1981:7–8). Fischer responded in the same journal to Smith’s six-page account with a seven-page retort, in which he explained why he could not accept the
conclusions in the literature cited by Smith (Campbell 1981:9–16). Fischer no doubt hoped that he had ended the debate, but Smith would have the last words, in a 34-page + illustrations response that the *Centralblatt für Bakteriologie* published in 4 parts (1899–1901, cited from Campbell 1981:17–51 + 11 plates).

The Society of American Bacteriologists, covering the United States and Canada, barely got organized during the 1800s—its organizational meeting was held on 27–29 December 1899 (McClung 1978).

**Other countries**

The study of phytopathology beyond Europe and North America was also important during the 1800s. What I have found about it may not be exhaustive.

Australia was in the 1800s a British colony, and some of its immigrants were educated in Britain or Europe. Wheat rust was a disease that struck wheat crops 10 times, 1799–1889 (Fish 1970:13). In 1864, the Victoria Board of Agriculture appointed a committee, chaired by Government Botanist Ferdinand von Mueller, to find the cause and prevention of rust disease. The committee recommended “early sowing careful selection of wheat varieties as a means of control” (Fish 1970:13). In 1898, William J. Farrer, a Cambridge University graduate who had immigrated to Australia in 1869, was botanist in the New South Wales Department of Agriculture. He researched production of rust-resistant wheat varieties, being advised by A. E. Blount, Colorado Agricultural Experiment Station. “The influence of Farrer’s varieties was so great that the area sown to them rapidly increased as the overall production of wheat in Australia expanded” (Fish 1970:14, quoting I. A. Watson, 1958). Scotsman Daniel McAlpine had studied under Thomas H. Huxley and William T. Thistleton-Dyer at the Royal College of Mines, London. He immigrated in 1884 and became lecturer in biology at the University of Melbourne (Fish 1970:14). In 1889 a rust epidemic led to McAlpine being appointed in 1890 as Consulting Vegetable Pathologist to the Department of Agriculture, Victoria. He wrote a treatise on Australian fungi in 1895 and a monograph on Australian rusts in 1906. He became known as “father of Plant Pathology in Australia” (Fish 1970:15). An American, Nathan Augustus Cobb, had obtained a Ph.D. in Germany in helminthology and went to Australia in 1889 and became Consulting Pathologist for New South Wales. While in Australia he wrote about a dozen scientific papers on nematodes, and his 1895 demonstration that gumming disease of sugarcane was caused by a bacterium impressed Erwin F. Smith, who had the USDA employ him in 1905. Cobb became “father of American plant nematology” (Buhrer 1969).

In Brazil, phytopathology began with the French naturalist–collector Augustin (Auguste de) Saint-Hilaire (1779–1853), who traveled there in 1816–1822 (Guerra 1975). He described wheat rust in several of his writings, which Anna Jenkins quoted in French (1945). Jenkins, from the USDA, was a long-time collaborator with A. A. Bitancourt at the Biological Institute of Saõ Paulo in phytopathological studies, beginning in 1934 (Bitancourt 1978:12–13).

In Japan, descriptions of diseased plants can be traced back to 713 and later, but there was no understanding of cause or remedy (Akai 1974:13–14). In 1649, the government prohibited foreigners from entering Japan or Japanese from leaving, except that Dutch ships were admitted to one port once a year. That situation ended in 1866, when a United States warship demanded its end. Afterwards, the
government was anxious for Japanese scholars to be brought up to date in western science. Japanese scholars studied in American and some European universities, and western professors taught for one or more years apiece in Japanese universities. The earliest teacher on phytopathology was a German professor from Brandenburg, Friedrich M. Hilgendorf, in 1873–1876 (Akai 1974:14). Shinnosuke Matsubara, a professor at the Tokyo Imperial University, published the first textbook on botany based upon western science in 1882, and it had a chapter on plant pathology. For the rest of the 1800s, Japanese and foreign botanists described plant diseases in Japan and attempted to discover the causal fungi (Akai 1974:15).

Whatever is published about history of New Zealand phytopathology is unknown to me. E. H. C. McKenzie was located at a government Plant Diseases Division when he wrote “Mycological History and Exploration in New Zealand” (1983). Perhaps he also published on history of phytopathology.

Conclusions

During the 1800s, phytopathology made enormous progress. It benefited from progress made in plant physiology—it helps in understanding diseased plants to first understand normal plant functioning. Progress in mycology, bacteriology, virology, and nematology were also essential contributions from related sciences. Many parasitic fungi turned out to be rather simple multicellular organisms that have complex life histories. As knowledge of plant diseases increased, governments responded by supporting agricultural research institutions and university instruction in plant diseases. In 1862, the United States created both land grant colleges that taught agricultural sciences, and a Department of Agriculture. Both creations grew to become centers for world leaders in phytopathology. Perhaps western European countries still remained in the forefront of research by 1900, but if so, they would suffer a setback during World War I that the United States did not experience, and by then the United States was leading the world in phytopathology because of better research funding and more facilities.

In retrospect, one sees many steps toward a germ theory of disease in this story, which, as in the 1700s, were little appreciated at the time.

Literature guide

There are ample historical sources on the phytopathological literature of the 1800s. Excellent places to begin are Geoffrey Ainsworth’s fine Introduction to the History of Mycology (1976) and Introduction to the History of Plant Pathology (1981). However, earlier works provide additional details. Herbert Whetzel’s Outlines of the History of Phytopathology (1918:32–106, 1977) has a good overview and extensive references; Ernest Large’s classic Advance of the Fungi (1940, 1962) is well written, illustrated, and documented. Narrower in scope and briefer is G. McNew’s “The Ever Expanding Concepts behind 75 Years of Plant Pathology” (1963); Gert Orlob’s “Concepts of etiology in the history of plant pathology” (1964:220–268); Garnet Carefoot and Edgar Sprott’s Famine on the Wind: Plant Diseases and Human History (1967) is briefer than Large’s history and less well illustrated, but more recent, with a good bibliography; George Ordish’s The Constant Pest: A Short History of Pests and Their Control (1976) covers both fungi and insects, since the Neolithic era; and James Horsfall and Ellis Cowling’s “The Sociology of Plant Pathology” (1977) is a history of phytopathologists in America, with a three-page list of “Hall of Fame” pathologists’ achievements. Chronologies of phytopathology

Phytopathology textbooks often have historical introductions (including Heald 1926:7–44, Walker 1969:14–46, Agrios 2005:8–28). Large (1940) and Ainsworth (1981) tell the interesting story of phytopathology’s impact on agriculture. Raymond Doetsch’s *Microbiology: historical contributions from 1776 to 1908* (1960) is a sourcebook of fifteen authors.

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Contributions October 2012 335


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