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Spontaneous Generation and Disease Causation: Anton de Bary's Experiments with *Phytophthora infestans* and Late Blight of Potato

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Abstract. Anton de Bary is best known for his elucidation of the life cycle of *Phytopthora infestans*, the causal organism of late blight of potato and the crop losses that caused famine in nineteenth-century Europe. But while practitioner histories often claim this accomplishment as a founding moment of modern plant pathology, closer examination of de Bary's experiments and his published work suggest that his primary motiviation for pursing this research was based in developmental biology, not agriculture. De Bary shied away from making any recommendations for agricultural practice, and instead focused nearly exclusively on spontaneous generation and fungal development – both concepts promoted through prize questions posted by the Académie des Sciences in the 1850s and 1860s. De Bary's submission to the Académie's 1859 *Alhumbert* prize question illustrates his own contributions to debates about spontaneous generation and demonstrates the practical applications of seemingly philosophical questions – such as the origin of life.

Keywords: Late blight, Potato, Anton de Bary, *Phytopthora (Peronospora) infestans*, Plant pathology, Plant physiology, Botany, Spontaneous generation, Mycology

On January 28, 1856, the Paris Académie des Sciences offered the *Grand Prix des Sciences Physiques* to the best response to the following challenge:

> Study the means of formation and the structure of spores and other organs that contribute to the reproduction of fungi (*Champignons*), their physiological role, the germination of the spores, and

> particularly for parasitic fungi, their means of penetration and development in other living things (*corps organisé vivants*).[1]

The sponsoring commission, which included Isidore Geoffroy St. Hilaire, Henri Milne-Edwards, Marie Jean Pierre Flourens, André-Marie-Constant Duméril, and Adolphe Théodore Brongniart, noted that different fungi may be similar in their means of growth, anatomy, and physiology, but their reproductive organs varied widely in form and function.[2] Precise observations of fungal reproduction had been made only for a handful of species, and the Académie hoped to solicit similar observations for a wider range of species. Using the microscope and chemical methods, the commission suggested, would lead to a new understanding of fungal reproduction.

The Académie expected the winning entry to address three particular points:

1. Formation, development, and comparative structure of the spores and the stylospores (*spermaties*) in the different groups of fungi;
2. Nature of the stylospores and the physiological role of these bodies in the reproduction of the fungi, determined by positive experiments;
3. Germination of the spores and propagation of parasitic fungi, either on the inside or on the outside of living plants (*végétaux*) and animals.[3]

The Académie did not award the Grand Prix as planned, however; only one entry had been received by the deadline of December 31, 1857. The Académie extended the deadline to April 1860, but cancelled the prize question outright when no further entries were forthcoming.[4]

At the Académie's meeting on September 29, 1862, acting Principal Secretary Jean-Baptiste Dumas read aloud an unsigned letter from a correspondent who had been pursuing studies relevant to the 1857 competition, but was unable to complete his research before its cancellation. The correspondent wrote that he planned to submit his research to a different question instead – the Académie's now-famous question on spontaneous generation. Proposed in 1859 by a slightly different committee, this challenge offered the *Prix Alhumbert pour les sciences naturelles* to whoever was able, "through well-conducted experiments, to cast new light on the question of so-called spontaneous generation (*génération dites spontanées*)." The challenge continued,

[1] Académie des Sciences, 1856, p. 161.

[2] *ibid.*

[3] *ibid.*, p. 163. A stylospore is a spore that forms directly on the mycelium – or vegetative organ of a fungus – rather than on a specialized branch.

[4] *ibid.*, 1861, p. 608.

"The Commission requires experiments that are precise, rigorous, and conducted equally under all circumstances, and from which, in a word, can be deduced results clear of all confusion born from similar experiments."[5] Especially of interest were experiments that could adequately account for the effects of external factors such as temperature on the development of the spores (*germes*) of lower plants and animals.

When the Alhumbert submissions were opened, the correspondent was revealed to be Anton de Bary, then *ordentliche* professor of botany at Freiburg. De Bary's expertise in fungal development made him particularly well-suited to addressing the question's first two criteria – the formation, structure, and reproductive structure of spores. In his submission, "*Recherches sur le développement des quelques champignons parasites*," de Bary responded to the Académie's points by elaborating upon his early research on various parasitic fungi, including species of *Cystopus* (now known as *Albugo*), *Peronospora*, and *Ustilago*.[6] But it was the third criterion, which required a demonstration of the germination and growth of parasitic fungi, that prompted him to draw his examples from these specific genera of fungi rather than from any of the many other organisms in his repertoire. All three were associated with and, de Bary insisted, causes of, plant disease.

In his recent study of late blight and potato breeding, R. Steven Turner has described de Bary as the "champion" of the "fungal hypothesis" – the claim that late blight of potato (among other plant diseases) was caused by a parasitic fungus whose life cycle produced consistent symptoms, including the characteristic white fringe and blackened tissue.[7] Turner identifies de Bary's work as having laid the foundation for over a century of research on reproduction in *P. infestans* and disease resistance in potatoes, yet the experiments de Bary conducted warrant further examination within the context of nineteenth-century botany. Though his contributions to scientists' understanding of plant disease cannot be denied – the practitioner legend that all plant pathologists can trace their heritage back to de Bary may well hold true – de Bary was surprisingly reticent on matters of preventing or controlling late blight. Instead, he chose to leave such matters to the discretion of agriculturalists. His reasons for this are unclear in his published work, but were not likely a result of his academic position. While de Bary never held an appointment at one of Germany's

[5] *ibid.*, 1859, p. 536.

[6] de Bary, 1863.

[7] Turner, 1980.

emerging agricultural or technical institutes, his early research focused predominantly on fungi associated with plant diseases. Likewise, his university-based contemporaries also dabbled in what we might now consider applied research; in 1866, for example, Ferdinand Cohn, secured funding to establish an institute of plant physiology at Breslau by agreeing to teach courses in agriculture and to conduct research for the Ministry of Agriculture as needed without additional compensation.[8] If university botanists of de Bary's generation were comfortable shifting from the lab to the field, his own reluctance to venture into practice was not likely a limitation imposed by his academic appointment.

Even so, his deference to the agriculturalists stands out because of its contrast to his historical stature, which has traditionally rested upon his research with *Peronospora infestans* (now *Phytophthora infestans*) – the causal organism of late blight of potato, the disease that ravaged potato fields throughout Europe during the 1840s. This hagiography, which owes more to the infamy of the Irish Potato Famine than to any detailed historical consideration of his experimental process, assumes that de Bary was driven by a profound interest in plant disease. Yet his reluctance to speak on matters of disease prevention or the agricultural application of his conclusions suggests that epidemics and famine were not his main inspiration, even as he drew upon (and published in) botanical and agricultural journals alike. Instead, the experiments he conducted and the language with which he described them suggest that de Bary's main concerns were elucidating the development and physiology of *P. infestans* and determining the origin of cryptogams.

This emphasis on development provided powerful evidence against spontaneous generation; the same experiments that allowed him to show the correlation between fungal life cycles and disease symptoms on potato plants also allowed him to demonstrate that *P. infestans* grew only from its own spores and could not appear *de novo*. Yet despite these meticulous observations, historians of spontaneous generation have consistently overlooked de Bary's research on plant pathogenic fungi in favor of the more colorful debate between Louis Pasteur and Félix-Archimède Pouchet. When placed within debates about spontaneous generation, however, de Bary's research on *P. infestans* illustrates the extent to which spontaneous generation was entwined with disease causation and the role of scientific institutions in shaping research questions. Ultimately, de Bary's efforts to meet the conditions set by the Académie des Sciences in the *Alhumbert* competition set an experi-

[8] Rosen, 1901; Klemm, 2002.

mental standard for cryptogamic botany, and his conclusions serve to illustrate the complex relationship between philosophical questions on the nature of life and practical concerns about plant disease and agriculture.

Spontaneous Generation and Models of Disease Causation

The Academie's 1856 Grand Prix question reflected an interest in "lower plants" that had emerged from Matthias Schleiden's focus on cryptogams as experimental models and spoke directly to a question that had intrigued many cryptogamists: how do algae and fungi reproduce?[9] To de Bary, identifying the reproductive means of cryptogams was a crucial component of understanding how late blight developed in potato fields. Because the sexual organs of fungi, algae, and other cryptogams were often well-hidden (hence the very term "cryptogam"), many mid-century botanists, Ernst Hallier and Carl von Nägeli among them, proposed that they generated spontaneously.[10] How botanists explained the whitish fringe they invariably observed on blighted plants was, in turn, directly relevant to whether they considered the disease itself to be contagious or the result of localized environmental conditions that weakened individual plants.

As a result, discussions of spontaneous generation mirrored discussions of disease causation. The jumble of explanations growers and naturalists offered for the appearance of late blight included nearly every possible cause of disease available in mid-century medical discourse. Letters published in *The Gardeners' Chronicle* in 1845 and 1846, the first 2 years of widespread crop loss in Ireland, for example, forwarded everything from too much rain to poor air quality in the affected fields to weak plant stock. These explanations reflected, respectively, models for disease that cited external stressors, misamatic theories, and even classic humoral pathology. Other authors attributed the disease to zymosis: an approach that combined the salient features of miasmatic theories – that stagnant pools of toxic air caused disease – with those of contagionist theories – that specific particles transmitted disease between individuals. Under this explanation, late blight was the consequence of planting a crop in fields that had not been properly cleared of rotting plant matter and were contaminated by organic particles that could spread from plant to plant.

[9] Farley, 1982; Mylott, 2002.

[10] Farley, 1977.

Yet despite the range of arguments available, strictly environmental causes were by far the most popular – especially those that cited excessive rainfall, temperatures that were unseasonably hot or cold, and winds of uncommon velocity or direction. Those who favored environmental explanations relied heavily upon spontaneous generation to explain the disease's appearance, whereas contagionists – including de Bary – insisted that the fungus seen on blighted plants was the result of infection by a single organism that had grown from seeds of their own kind.

The nature of both endophytic and epiphytic fungi – that is, those found inside and on the surface of plant tissue, respectively – was a popular topic for debate among botanists in the early nineteenth century.[11] In 1833 the Viennese botanist Franz Unger claimed that these fungi were morbid excretions of their hosts and argued that plant diseases were the result of poor growing conditions combined with a plant's inherent weakness.[12] He claimed that most plant diseases involved the plant's vital fluids (*Säften*), and stated,

> The faulty formation (*Ausbildung*) and scores of abnormalities in the chemical actions of the sap, as with similar defects of greater life fluids (*Lebenssaftes*), are the origins of countless diseases that manifest through the defective formation of plant material, through the accumulation of excretions, through breaking-up of parenchyma, through altered consistency of secretions, etc..."[13]

He called these growths "*Exantheme,*" a term that had been primarily used to describe animal rashes, and regarded them as symptoms of an imbalance or abnormality in the plant's vital fluids – a claim reminiscent of classical humoral pathology. Such assumptions had been common for centuries, as de Bary later noted, and only recently had botanists identified endophytic fungi as unique organisms that were different from their host. He wrote, "Ancient botanists and agriculturalists knew of endophytic fungi, yet took them for pathological products of the tissues where they were found. Several endophytes were seen as proper organisms by botanists of the last century."[14]

Although later authors on plant disease dropped the direct parallel to animal pathology, many still explained fungal growths as the spon-

[11] Braun, 1965.

[12] Unger, 1833.

[13] Unger, 1833, quoted in Braun, 1965, p. 47; Gliboff, 1998.

[14] de Bary, 1863, p. 7.

taneous result of plant weakness. As such, the existing plant tissue gave rise to the fungal growths – a process commonly referred to as "heterogenesis," or spontaneous generation from living or dead organic tissue – as a response to an external stressor. In Britain, the scientific commission appointed by Prime Minister Robert Peel insisted that the blackened, withered leaves and strange filamentous growths that characterized late blight were the result of the potato plants' weakening by climatic conditions or inadequate agricultural practice. Yet even the strictest of environmental theorists acknowledged that the filaments were not part of the plant itself, but were a completely different organism. They even referred to the organism by its specific Latin designation: first *Botrytis infestans* and, after 1845, *Peronospora infestans*.[15] They were equally insistent that the growths were the product of heterogenesis resulting from external stress – or, that the fungi were distinct organisms, but had been produced from the plant's own tissue. Although such arguments did not identify the precise mechanisms that produced these growths, they were epistemologically similar to past claims that maggots spontaneously generated from meat, in which one organic form was derived from another.

In contrast, the contagionists – represented most strongly in discussions of late blight by de Bary in Germany and the Rev. Miles J. Berkeley in England – agreed that the fungal fringes were an organism distinct from the potato plant, but argued that the fungus was the direct cause, not a result, of late blight. De Bary in particular argued vehemently against both environmental causes and spontaneous generation, and offered extensive observational and experimental evidence that supported contagionist explanations. Ongoing debates about the cause of late blight (and plant disease in general) illustrate how competing theories of generation, by virtue of their implications for disease causality, turned an agricultural problem into a highly intellectualized point of contention.

Late Blight and the "Fungal Theory"

De Bary was a latecomer to investigations of late blight. When he began his research in the 1850s, he drew upon an extensive body of literature

[15] Waterhouse, 1970. I use *"P. infestans"* to refer both to the prior classification of the organism as a member of the genus *Peronospora* and its later designation as *Phytophthora*.

on the disease – his experimental notes listed nearly 150 published papers on late blight of potato published between 1782 and 1859, accompanied by various levels of commentary and critical annotations.[16] Reports of the disease began appearing in print consistently after 1819 and multiplied rapidly following the 1842 European outbreak. When British Prime Minister Robert Peel finally named Dr. Lyon Playfair, John Lindley, and Sir Robert Kane to his now-famous scientific commission on the Irish potato epidemic in late autumn 1845, continental botanists' efforts to identify the cause of the disease had been underway for nearly half a century.[17]

Botanists disagreed on what to call the fungus – it bore three taxonomic designations in 1845, and two more by 1876 – and how it reproduced, but the morphologies published papers described were nearly identical.[18] Almost all accounts described loose, delicate filaments on the leaves and stems of blighted plants. These filaments, or conidiophores, were highly branched with knobbed joints, and each branch bore multiple ovoid conidia, or asexual spores. Microscopic investigations of blighted plants showed the presence of the fungal mycelium – the vegetative body of cylindrical tubules that progressed through the intracellular space in the plant tissue and left behind brown or black spots on the surface of the plant. These same symptoms developed in infected tubers as well; the disease began as small brownish-black lesions that quickly spread into large, spongy, sunken areas. The conidiophores appeared on the surface of these areas, covering the tuber with the same whitish fringe seen on blighted leaves.

Berkeley observed these structures on potato plants in the mid-1840s and proposed that the disease was caused by an external parasite – the very same organism that was found on blighted leaves.[19] Berkeley, an accomplished and respected naturalist, described this phenomenon to Dr. Charles Montagne, a French physician and naturalist with whom he had previously corresponded on matters botanical. Montagne had noticed the same growths on blighted fields in France, and, after discussing their respective specimens, the two concluded that the fungus on the English and French plants was one and the same. In late August 1845, Montagne

[16] de Bary, Nachlass, Mpe 15 Bll. 15,2-15,19.

[17] Large, 1940; Salaman, 1985; Bergman, 1967.

[18] Waterhouse, 1970. *Phytophthora infestans* is now considered an oomycete (a group of organisms that differ from true fungi in their physiology and manner of reproduction and are phylogenetically distinct), but I have retained the term "fungus" to reflect nineteenth-century understanding of the organism's biology and phylogeny.

[19] Woodham-Smith, 1962.

presented the fungus as a newly discovered species to the Société Philomatique in Paris as *Botrytis infestans*.

In January 1846, Berkeley published "Observations, Botanical and Physiological, on the Potato Murrain" in the *Journal of the Horticultural Society of London*.[20] He argued that *B. infestans* was the cause of the blighted symptoms seen in potato fields across the United Kingdom and continental Europe, and declared,

> ...after an attentive consideration of the progress of the disease and of almost everything of value that has been written on the subject, and after duly weighing the peculiar difficulties with which it is attended, I must candidly confess, that with a becoming share of philosophic doubt where such authorities are ranged upon the opposite side, I believe the fungal theory to be the true one.[21]

Berkeley acknowledged that opposition to the "fungal theory" was common; he mentioned the commissions formed in Ireland, Brussels, and Groningen, as well as individual naturalists (including Félix-Archimède Pouchet in France and Friedrich Kützing in Germany), as examples of such opposition. Whether they attributed late blight to excessive rain, "peculiar electric phenomena," or fluctuations in heat and moisture, he noted, "they all tend necessarily to the same point:" that the disease was "independent of parasitic fungi, and that it arises from the peculiar atmospheric conditions of the season."[22]

Berkeley did not deny that environmental factors influenced the development of late blight, but he did deny that climate could be the *only* cause. Calling attention to the notoriously wet weather in the affected areas, he reasoned,

> Did the disease really arise from the water of vegetation existing in too great abundance, and hence inducing putrefaction of the cellular substance, it would be difficult to say why it has not existed to a considerable amount before...I think then it is at least plain that no supposed peculiarities of season are sufficient without some more specific cause to account for the general prevalence of this disease.[23]

Lindley challenged him on this very point by asking him to demonstrate that the fungus was *not* a normal consequence of putrefaction from

[20] Berkeley, 1846. "Murrain" and "late blight" were equivalent terms.

[21] Berkeley, 1846, p. 18.

[22] *ibid.*, p. 19

[23] *ibid.*, p. 22.

congestion by excess matter. If the fungus were indeed causal, Lindley argued, it must somehow come into contact with the plant *before* the symptoms of blight developed. He recognized that some fungi formed air-borne spores, but pointed out that even this did not explain how tubers buried underground could be infected. When pressed, Berkeley was unable to counter these objections.

This, then, was the gauntlet that de Bary picked up. By August 1853, he had already observed the symptoms of late blight on the surface of potato plants and described the morphology of both the mycelium within plant tissue and of resting and germinating spores.[24] He then mapped the symptomatic progression of late blight against the developmental states of *P. infestans* through a series of experiments in 1861 and 1862. He induced its growth on the same tissues – leaves, stems, and tubers from potato plants – and under the same variations of light and moisture in which it grew in the field, and found that the onset of the brownish-black lesions on the plants' leaves and stems corresponded to the initial entrance of fungal hyphae (thin tube-like structures) into the plant tissue (Figure 1). The spots and necrosis that characterized late blight followed the spread of the mycelium (or vegetative body of the fungus) deeper into the interior of the plant (Figure 2), and the appearance of conidiophores – the thin, delicate filaments on the leaves and stems – correlated with the maturation of the fungus (Figure 3). These fruiting stalks in turn produced conidia (Figure 4), or asexual spores that, when introduced to healthy tissue under the right conditions, either released a swarm of zoospores (motile spores) or germinated on their own (Figure 5). When the zoospores or conidia germinated, they punctured the epidermis of their new host and began a new cycle.

The experiments de Bary conducted to illustrate the correlation between the life cycle of *P. infestans* and stages of disease progression, also served as powerful ammunition against doctrines of spontaneous generation.[25] Proponents of spontaneous generation shared the conviction that direct parentage was not necessary, but the material from which organisms emerged and the mechanism of generation could differ

[24] SBPK De Bary Nachlass, Mpe 15, Bl. 108 and unpaginated; de Bary, 1861. Although de Bary used the term "conidium" (pl. -a) in his 1863 paper, he used "sporangium" in his 1861 book on late blight. "Sporangium" refers to the structure that contains the asexual spore and is the term currently in use. De Bary may have regarded the two as synonymous.

[25] Farley, 1977; Farley, 1992, pp. 133–149.

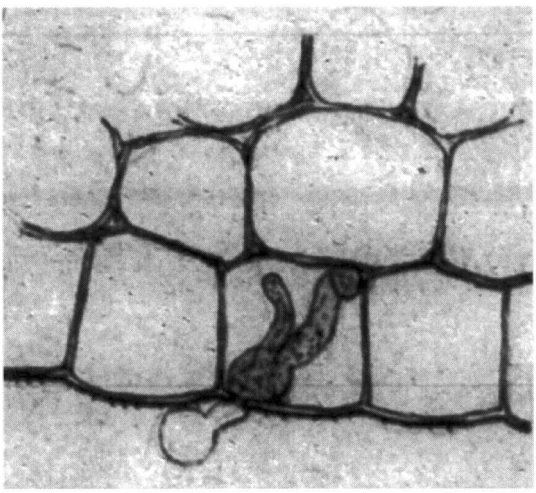

Figure 1. Initial entry of *P. infestans* into host leaf tissue. De Bary's experiments showed that upon germinating, the conidia (now referred to as "sporangia") of *P. infestans* produced slender filaments that penetrated the host tissue. From de Bary, Nachlass, Mpe 15 Bl. 80, undated.

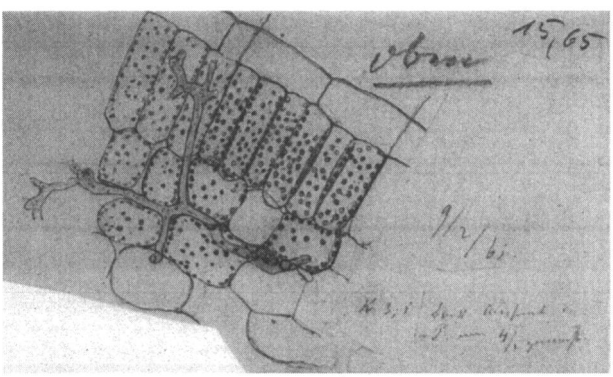

Figure 2. P. infestans present in leaf tissue. De Bary's experiments with *P. infestans* in 1860 and 1861 showed that the mycelium, or vegetative structure, of the organism spreads vertically through the cells in the host tissue and horizontally through intracellular space. This sketch is dated February 9, 1861. From de Bary, Nachlass, Mpe 15 Bl. 80.

depending upon the organism in question and the philosophical preferences of the observer. Heterogenesis may have appealed especially to cryptogamic botanists, who studied organisms with reproductive structures that ranged from carefully hidden to downright impossible to identify. The sheer frustration of searching for (and not always finding)

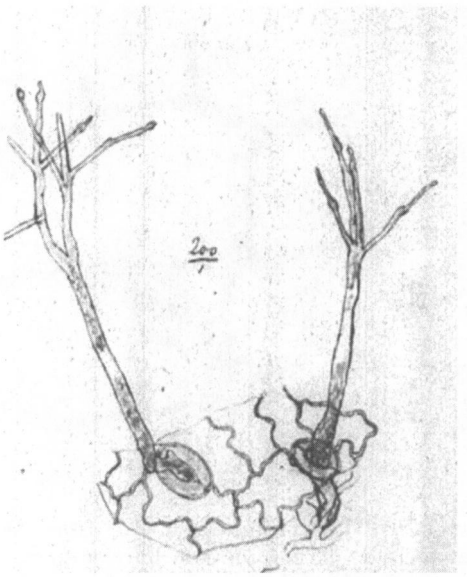

Figure 3. Conidiophores emerging from stomates on leaf tissue. The conidiophores of *P. infestans* emerge from leaf tissue either through the stomates or by raising the epidermis and puncturing individual cells. De Bary observed these conidiophores on August 1, 1853. From de Bary, Nachlass, Mpe 15 Bl. 109.

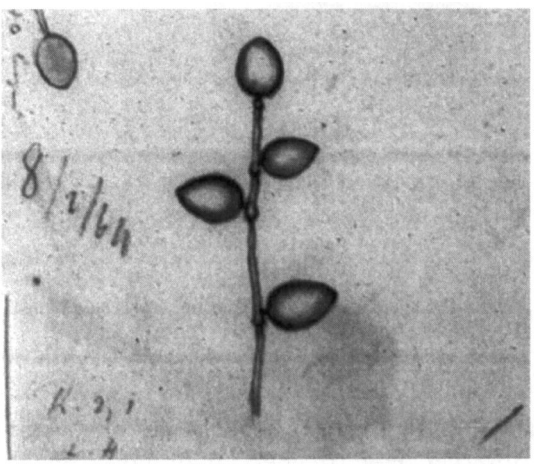

Figure 4. Conidiophore with conidia. As conidia at the tip of the conidiophore develop, they are "pushed" to one side and the tip continues to grow. From de Bary, Nachlass, Mpe 15 Bl. 79.

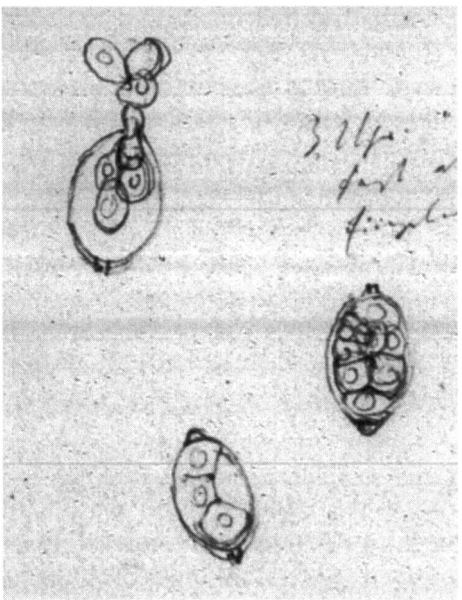

Figure 5. Conidia with zoospores contained therein. De Bary observed that the conidia of *P. infestans* can either germinate singly on their own or release multiple zoospores, which then germinate on the host tissue. This sketch is dated February 11, 1861. From de Bary, Nachlass, Mpe 15 Bl. 79.

cryptogams' means of reproduction may have prompted cryptogamists to embrace spontaneous generation as conforming most closely to their observations – again echoing earlier discussions of parasitic worms.[26]

Whether spontaneous generation was possible was therefore the linchpin for debates about the cause of plant disease. For those who insisted that the conidiophores of *P. infestans* seen on blighted leaves were the material product of a host plant's response to external stress, explaining late blight depended on nothing more than bad weather and weak plant stock. Such explanations did not require clouds of toxic air as did miasmatic theories; the presence of an organic or chemical toxin as in zymotic theories; or a mechanism for transferring living organisms from one host to another as did contagionist explanations. By invoking host weakness and poor growing conditions, those who defended spontaneous generation could neatly explain the appearance of *P. infestans* on blighted leaves.

Such claims carried with them a specific set of practical recommendations for dealing with the disease. In Ireland, Lindley was especially adamant that the disease was a form of plant "dropsy" resulting from

[26] Farley, 1982.

the unusually damp soil, and he argued that the tubers and plants had putrefied because they were unable to absorb excessive rainfall.[27] Accordingly, the commission's recommendations for coping with late blight focused on keeping the fields well-drained and the stored tubers dry. Farmers were instructed to dry their seed potatoes in the sun and store them in shallow dugouts lined with soil treated with either sawdust or lime rather than in untreated pits. To the dismay of farmers and botanists, these efforts showed only limited, short-term efficacy. As Woodham-Smith noted, "Whether ventilated, desiccated, salted, or gassed, the potatoes melted into a slimy, decaying mass; and pits, on being opened, were found to be filled with diseased potatoes."[28]

De Bary was not inclined to give environmental arguments any credence. He had dedicated his early career to meticulously following the developmental cycles of various fungi, and was therefore well-versed in cryptogams' ability to reproduce via sporulation and germination. He had been impatient with spontaneous generation from his first foray into plant disease; in 1853, he published *Untersuchungen über die Brandpilze und die durch sie verursachten Krankheiten der Pflanzen*.[29] There, de Bary summarized the current knowledge of the "Brand fungi" (those fungi associated with the bunt and rust diseases of wheat) with an eye towards challenging existing classificatory schema that relied heavily upon morphological characteristics. He instead proposed that examining the processes of fertilization and reproduction would lead botanists to more accurate taxonomies than morphology alone.

Studying development could also help characterize the interactions between fungi and the plants upon which they were found. In the second half of the *Untersuchungen*, de Bary argued that bunt and rust must be caused by an external parasite. He noted that some of his colleagues still favored symptomatic theories, and showed no restraint in accusing them of "relying upon the negative results of their often quite scanty experiments." He deemed their theories "unsupportable," and announced, "the facts on which the proponents of this view support their argument turn out to be in part the products of delusions and in part to have been the products occasionally of a truly deplorable mania for theories and analogies."[30] De Bary left no doubt as to his stance on proper investigative technique, cryptogamic reproduction, or the cause of plant disease.

[27] Woodham-Smith, 1962, p. 47.

[28] Woodham-Smith, 1962, p. 74.

[29] de Bary, 1853, p. 54; Arny et al., 1969.

[30] Arny et al., 1969, pp. 68–70.

De Bary's "Recherches" as Experimental Evidence

As Antonio Gálvez has noted, the Académie's 1859 Alhumbert prize question on spontaneous generation was only one in a series concerned with the generation of living things that began in the 1840s.[31] The Académie's interest in spontaneous generation was long-standing, and although the prize question was its most direct expression thereof, the question was not itself either unusual or unexpected. The immediate impetus behind the Alhumbert question was the publication of Félix-Archimède Pouchet's *Hétérogenie ou la Traité de la génération spontanée* in 1859, in which Pouchet, then director of the Natural History Museum at Rouen, reversed his previous denial of spontaneous generation and defended heterogenesis as the source of "the eggs of infinitely small organisms."[32]

Pouchet's treatise followed a series of publications by Louis Pasteur, whose chemical investigations had led him to declare that fermentation as a process required the presence of microorganisms. The resulting debate between Pasteur and Pouchet has been well-documented elsewhere and needs only brief explanation here.[33] Shortly after the publication of Pouchet's *Hétérogenie,* Pasteur recounted some of his own researches on fermentation and lactic acid in a short exchange of letters with Pouchet. Pasteur then presented to the Académie a series of short notes that demonstrated the presence of living organisms in the air, thus discounting Pouchet's experimental results as contaminated. The Académie awarded Pasteur the Alhumbert prize in 1862 (over de Bary's submission) for these pieces, which were published in *Annales des sciences naturelles* under the title "Mémoire sur les corpuscles organisés qui existent dans l'atmosphere."[34] The Académie's decision inflamed Pouchet, who reportedly withdrew his submission, citing bias on the part of the committee.[35]

While Pasteur and Pouchet were engaged in their public tussle de Bary prepared and presented "Recherches sur le développement des quelques champignons parisites." Again, he had initially intended to submit this paper to the 1856 Grand Prix competition on fungal

[31] Gálvez, 1988. Gálvez consistently translates "*Champignons*" as "mushrooms," but the word was (and still is) used to include *all* fungi.

[32] Pouchet, 1859; Farley and Geison, 1974, p. 168.

[33] Farley and Geison, 1974; Farley, 1977; Roll-Hansen, 1979; Geison, 1995.

[34] Pasteur, 1861; Académie des Sciences, 1862, pp. 977–979.

[35] Farley and Geison, 1974, p. 181.

reproduction, but was unable to complete his research before the question's withdrawal. The Académie's stipulation that all entries include experimental evidence may have caused this delay; the "Recherches" included accounts of experiments de Bary had carried out with *Cystopus* in early 1862, and it is likely that he had been in the middle of his investigations when the question was cancelled. De Bary even noted that the experiments he designed were time-consuming and could only proceed very slowly precisely because they depended on observing the complete development of the organism. He wrote, "It is evident that to answer these questions, there is no other way than a very careful study of the entire development of the parasite, along with well focused experiments under the external conditions by which development is governed...One can therefore only advance very slowly."[36]

These experiments went slowly in part because de Bary, true to the conditions of the prize question, meticulously followed the developmental cycle of *P. infestans* from germination through maturation and reproduction. This, in turn, allowed him to chart growth and nutrition as physiological processes against the progression of symptoms seen in host tissue. He accomplished in his experiments what neither Berkeley nor any other botanist had been able to: he demonstrated that the fungi associated with late blight were distinct organisms, confirmed that they generated exclusively from spores of their own kind, and established that they were the cause, not the symptom, of plant disease.

De Bary was fortunate that the similarities between the 1856 Grand Prix and 1859 Alhumbert questions reflected the Académie's broader interest in spontaneous generation, thus allowing him to apply his expertise to both competitions. Again, the criteria the Académie set for the Grand Prix asked entrants to examine the development and germination of spores either within or on the surface of other organisms. In effect, the Académie had posed a question that could not be answered to its satisfaction by any assertion of spontaneous generation. The experimental techniques the question demanded could not support heterogenesis, much less abiogenesis. The practical requirements of conclusive experimental evidence, if carried out by observing fungal development in its entirety as the Académie required, simply could not support spontaneous generation.

The committee's specific interest in parasitic fungi gave additional preference to those who denied the possibility of spontaneous generation in general and heterogenesis in particular. De Bary's equation of

[36] De Bary, 1863, p. 6.

"parasitic" with "pathogenic" was therefore especially meaningful; parasites, then as now, were organisms known to draw their sustenance from a living host. This, in turn, resulted in pathogenesis, or a set of specific symptoms seen in the host tissue, as the parasite completed its life cycle. To de Bary and other botanists who recognized fungi as causes of plant disease, parasites and pathogens were equivalent; even in modern plant pathology, any difference between the two terms is negligible. In order to be a specific – that is to say, single – cause, a parasitic fungus must come into contact with the plant *before* the symptoms of that disease appear, just as Lindley had insisted to Berkeley in 1846; that required that the parasites originate independently from the host tissue in order to infect it. Or, in short, the organism could not simultaneously be a cause *and* a symptom of disease.

Pasteur and Pouchet had addressed this distinction in their discussion of fermentation. As Farley and Geison explained,

> Implicit in the notion of the specificity of fermentative microorganisms is a commitment to an ordinary sort of generation for them. Only if they arise by an ordinary sort of reproduction, it would seem, could they retain the specific hereditary properties which must account for the specificity of their actions during fermentation.[37]

Specificity, or the claim that each unique cause produced its own unique set of products, was not the heated issue that it would later become in discussions of bacterial diseases, where questions of causality were complicated by debates about pleomorphism.[38] But the same principles involved in fermentation and specificity also applied to contagion and causality. Just as Pasteur understood that proving microorganisms' involvement in fermentation meant demonstrating their presence before the process began, de Bary recognized that he would need to show the presence of *P. infestans* in or on a plant before the symptoms of blight appeared. Only if *P. infestans* were the product of "an ordinary sort" of reproduction could it be the specific causal organism that produced the consistent set of symptoms that spread so rapidly throughout blighted fields.

Strict adherents to spontaneous generation objected to this argument by claiming that identical environmental stresses could cause plants to consistently generate the same fungal growths. But taking this stance could be problematic; Lindley, for example, found himself in the

[37] Farley and Geison, 1974, pp. 173–174.

[38] Mazumdar, 1995.

awkward position of explaining how climate could have caused the same symptoms in the 1845 and 1846 epidemics when the conditions during their respective growing seasons were completely different. In this way, at least, de Bary's insistence that late blight of potato (like the other plant diseases he had studied) was transmitted through natural reproduction was much neater. Because de Bary did not accept late blight as an individualized response to environmental pressures, his argument did not rely on weather remaining consistent across multiple growing seasons. That late blight was the result of fungal development might have posed some experimental challenge, but it did not leave de Bary fumbling to explain consistent symptoms caused by inconsistent conditions. He remained insistent that late blight of potato (like the other plant diseases he had studied) was transmitted through the normal reproduction of fungi, and was *not* an individualized response to environmental pressures.

De Bary noted early in his "Recherches" that reproduction and disease were entwined; he wrote, "Considering the assertion, made numerous times, that these parasites are both from the exact same substance as the organism that carries them, these investigations [of plant disease] are intimately linked to the question of spontaneous generation."[39] He then set forth the two main questions he sought to answer through his experiments. First, he asked, "What is the origin of parasitic fungi; what is the manner by which they reach the stage at which we observe their fruit; what is their causal relationship with the morbid state of the organisms on which they live?"[40]

His second question directly challenged arguments that these fungi could arise by heterogenesis:

> Can we note that, in a sick plant, the mycelium of an endophyte was born of the same diseased substance, not of a germ from an endophyte of the same species? Or do directly observed facts explain the appearance of an ordinary endophyte by the development of its germs that have penetrated the plant that carries it?[41]

Studying development under the same conditions in which the fungus grew naturally, he asserted, would provide conclusive answers to both sets of questions, and prove that parasitic fungi could be a direct cause of plant disease. Establishing these conditions carefully was essential;

[39] de Bary, 1863, p. 6.

[40] *ibid.*, p. 6.

[41] *ibid.*, p. 13.

the observations de Bary needed to understand the growth and physiology of *P. infestans* as it progressed through potato plants and tubers could only be made under a microscope in a laboratory, not in the field. But by regulating the temperature, moisture, and host tissue carefully, de Bary could reasonably replicate the conditions under which the fungus thrived in the field.

De Bary began his account of his experiments with *P. infestans* by reiterating the morphology of the *Peronospora* as a genus. Though each species showed its own unique attributes, the genus as a whole possessed a highly branched mycelium with hyphae that filled the intracellular channels of the host tissue. The conidiophores grew directly off the mycelium, raising the epidermis of the leaves by puncturing the cell walls of the epidermis or by pushing out through the stomates (Figure 3).[42] The conidiophores of *P. infestans* bore between two and five branches from its upper portion, with individual conidia growing directly upon them; other species of *Peronospora*, in contrast, showed multiple main branches narrowing into a series of smaller branches – each of which bore its own conidium.

Germination was central to disproving spontaneous generation, and de Bary recounted multiple experiments he had devised to demonstrate that *P. infestans* could develop only from its own conidia and not other organic material.[43] He explained that *P. infestans* can germinate in one of two ways, depending upon the temperature and moisture content of its environment: either the conidium itself will germinate and produce a germ tube that enters the host tissue, or it will release a swarm of motile zoospores that germinate and penetrate the plant tissue individually. De Bary's writings indicate that he observed both processes, but he did not always distinguish between which he had observed in a particular experiment - perhaps suggesting that he regarded them as physiologically equivalent. On February 4, 1860, for example, de Bary sowed conidia of *P. infestans* on a portion of potato leaf. By the following morning, the conidia had germinated and penetrated the tissue, and on February 8 de Bary observed "the eruption of fertile branches" – the conidiophores and their conidia.[44] A second experiment yielded similar results, thus showing that the conidiophores developed from their own species, and were distinct in form and origin from host tissues.

De Bary then demonstrated that *P. infestans* could be transmitted from plant to plant by transferring the new conidia to different host tissue. At 5:00 p.m. on February 9, he harvested the mature conidia

[42] *ibid.*, pp. 35–36.

[43] Speerschneider, 1857, pp. 81–87; Large, 1940, p. 96.

[44] de Bary, 1863, p. 47.

from his first experiment and distributed them across a set of glass slides with a bit of water and a slice of healthy potato stem.[45] Two hours and fifteen minutes later, de Bary noted, zoospores had been released from the conidia and elongated into tubules. By the next morning, these tubules had penetrated the stem tissue, and by February 11, the mycelium had spread through six layers of cells via intracellular space in the parenchyma. Three days later, conidiophores were visible on the surface of the stem tissue. De Bary noted that conidia of *P. infestans* showed the same rapid development when sown on a slice of tuber as when sown on leaf or stem tissue: they first pierced the corky outer layer of the tuber and then spread throughout "the walls of the superficial cells to scatter into the parenchyma located underneath them."[46] He interpreted these experiments, along with sowings of other species of *Peronospora*, as proof that the fungus did not appear spontaneously within the plant tissue, but developed from conidia of its own species.

These experiments supported de Bary's claims that the mycelium and conidiophores seen on blighted fields had developed through the germination of spores, not by spontaneous generation within the plant tissue. The delayed onset of symptoms that had baffled farmers became less puzzling when explained in terms of the germination of unseen spores, and in additional experiments, de Bary illustrated that the conidia could germinate up to 3 weeks after maturation as long as they were not completely desiccated.[47] Mature conidia could wait out the weather, so to speak – they could withstand short periods of dryness and germinate when rain set in, producing what appeared to be completely spontaneous symptoms.

The levels of moisture in which *P. infestans* could survive and grow to reproductive maturity fell within distinct parameters – an observation that provided additional counterevidence to environmental interpretations that attributed disease symptoms to a plant's individual response to oversaturation. If the appearance of symptoms in the host corresponded to the life cycle of an externally introduced parasite, and if that parasite could not grow, mature, and produce under the particular conditions that the symptomatists claimed produced it, then the symptoms of late blight could not be a stress-induced plant response. De Bary acknowledged that moisture was a critical component in the onset of late blight, but his experiments disproved claims that the disease was the host plant's internal response to excessive rain.

[45] *ibid.*, pp. 46–48.
[46] *ibid.*, pp. 47–48.
[47] *ibid.*, pp. 42–43.

Indeed, de Bary identified water as the greatest factor that influenced the growth of *Peronospora*: it, more than any other environmental condition, determined whether the organism grew prolifically and reached reproductive maturity or grew irregularly and died prematurely. Excessive moisture was detrimental; conidia exposed to high levels of moisture either did not germinate or produced stunted, deformed mycelia that did not mature.[48] He observed that when a section of plant tissue infected with *Peronospora* was submerged in water, the resulting growth was spindly and leggy and died quickly. In contrast, if the section of host tissue was sprayed with water or placed in a moist environment, the fungus developed normally – in fact, such environments were highly favorable.[49]

De Bary believed the correlation he had established between the life cycle of *Peronospora infestans,* its growing conditions, and the stages of late blight was sufficient to conclusively dismiss environmental or noncontagionist arguments. Yet he wanted to be quite clear that the appearance of this parasite on living plant tissue was not the result of the plant's predisposition, and asserted that he had never found one individual plant to show a greater susceptibility than another plant of the same species, as long as the environmental conditions in which they lived were identical.[50] Even arguments that blighted plants must have been inherently weak to begin with could not hold up under de Bary's developmental observations. It may have been logically tempting to conclude that a weak host was more susceptible to disease, but de Bary showed that weakened or dying plants were inhospitable to *P. infestans* and that the parasite thrived best in the healthiest, most robust plants. In fact, de Bary continued, his experiments showed that *Peronospora* could only survive in healthy hosts, and would not grow on weakened or rotting tissue (a distinction that would later figure into de Bary's creation of a new genus for *P. infestans*.)[51] As the mycelium of *P. infestans* progressed through the plant tissue and necrosis set in, its growth slowed, then ceased. He found the same phenomenon in the tubers – as long as the surrounding tissue remained alive, the mycelium would grow to reproductive maturity. Because *Peronospora* required healthy host tissue, weak plants could not adequately support its maturation and production of conidia. Any growths found on dead tissue,

[48] *ibid.*, pp. 53–54.
[49] *ibid.*, pp. 53–54.
[50] *ibid.*, p. 66.
[51] *ibid.*, p. 54.

de Bary concluded, had been introduced to the tissue secondarily, and were not a species of *Peronospora*.

For the most part, the experiments upon which de Bary based his claims of causality and his attack on spontaneous generation were short and uncomplicated. They required little more than microscope slides (or other glass receptacle), a ready supply of fresh, healthy host tissue and implements for making sections, a microscope for observing the progress of the fungus, and a sample of the fungus itself. This equipment was not particularly specialized, especially in contrast to that required for investigations in phanerogamic physiology – for example, Julius Sachs's self-recording auxanometer, a delicate instrument he designed to measure plant growth.[52] Furthermore, the rapid maturation of *P. infestans* meant that de Bary's microscopic observations could be conducted over a matter of days, rather than the weeks or even months that physiological investigations of phanerogams (more complex plants with specialized organ systems) often entailed. For all the burden of proof he placed upon them, de Bary's experiments were remarkably elegant in their simplicity.

The Missing Link: Sexuality in *P. infestans*

Despite this research, one crucial detail about *P. infestans* eluded de Bary: in his experiments of the 1860s, he had been unable to identify its sexual spores – a potentially crucial factor in explaining how the organism overwintered and reappeared in subsequent growing seasons.[53] De Bary had observed that the conidia of *P. infestans* could germinate up to 3 weeks after maturation, but were structurally delicate and highly dependent upon moisture. He also knew that, because the mycelium of *P. infestans* died with its host, the fungus could not overwinter in dead or decaying tissue left in the fields. The presence of conidia and mycelia alone, therefore, could not explain the reappearance of the fungus at the start of the growing season on plants grown from healthy tubers. De Bary had observed sexual spores in other plant pathogenic fungi, including *Puccinia, Pythium,* and *Ustilago*, and known them to be hardier spores capable of remaining dormant and viable during the off-season. Arguing by analogy, de Bary concluded that if he could find the same resting structures in *P. infestans*, he could explain the recurrence of late blight in fields year after year.

[52] de Chadarevian, 1993, 1996 .

[53] Turner, 1980.

Although de Bary's focus shifted away from spontaneous generation after the mid-1860s, his determination to find sexual spores in *P. infestans* led him to accept £100 from the Royal Agricultural Society (Great Britain) to renew his investigations.[54] Locating oospores was a central goal of the two questions he sought to answer through his new experiments; when he published his results in 1876, he asked, "Where does [*P. infestans*] remain in the period between its disappearance and its reappearance?" and "How and where does it winter, and how does it pass from its winter-quarters to the foliage of the potato?"[55] He chose not to address either spontaneous generation or disease causation, explaining,

> That the species of the Peronosporae, as parasites, do more or less interfere with the life of their host and produce disease in it must be obvious; and the circumstances under which this occurs is too generally known to require detailed explanation here.[56]

By the publication of de Bary's paper, Pasteur's conclusions, along with Ferdinand Cohn's demonstration of heat-resistant spores in *Bacillus subtilis*, had weighed in heavily against spontaneous generation, and research on plant disease and cryptogamic reproduction had made such theories much less common in cryptogamic botany than they had been earlier in the century.[57] Spontaneous generation debates were ongoing in Britain; Jim Strick, for example, has detailed the heated debates between Henry Charlton Bastian and John Tyndall in Britain.[58] But although de Bary was writing for a predominantly British readership, he may have regarded his responsibility to the Royal Agricultural Society as explaining only the how the organism overwintered and not where it originally came from. Since explaining both overwintering and the missing sexual cycle assumed generation from its own spores, de Bary may have assumed that spontaneous generation was of no interest to growers despite any perceived threat from these continued debates.

De Bary's extensive knowledge of other fungi (and other *Peronosporae* in particular) gave him a firm basis for his conviction that

[54] J.D. Hooker, then director of Kew Gardens, wrote to Charles Darwin on March 3, 1874, that the Society's request of de Bary was an insult to Berkeley and his investigations of late blight of potato. Cambridge University Library, DAR 103: 189–192.

[55] de Bary, 1876.

[56] de Bary, 1876, p. 244.

[57] Cohn, 1875; Pasteur, 1861; Strick, 2000; Farley, 1977.

[58] Strick, 2000; Farley, 1977.

P. infestans produced oospores. Whereas other *Peronosporae* formed asexual conidia and oospores by sexual fertilization, de Bary had known *P. infestans* to produce conidia exclusively.[59] This was crucial to understanding how the fungus often reappeared in the same fields season after season. Unlike conidia, oospores could survive the winter and germinate upon the arrival of warmer weather. De Bary argued that if the conidia of *P. infestans* could not overwinter, then there were only two plausible explanations as to how the organism itself did: through the survival of the mycelium in the tubers or by the production of oospores. He had observed the survival of the mycelium in tubers contaminated with *P. infestans*, but had never found sexual spores – those were limited to *Cystopus* and all other *Peronospora* except for *P. infestans*. This apparent lack of oospores was one of the main reasons de Bary proposed creating a new genus – *Phytophthora* – for *P. infestans,* yet he still remained optimistic that careful research would ultimately locate its oospores. He noted,

> Oospores have not been observed in the *Phytophthora*; but from analogy, it may be taken as certain that they may somewhere occur. The discovery of them would at once fill up the gap, both in the morphology of the fungus and in the practically important question of how it hibernates.[60]

The experiments de Bary undertook in search of oospores expanded upon those he had conducted in the 1860s and presented in "Recherches" as evidence against spontaneous generation. In much the same way as he had collected and sown conidia on dampened slides and plant tissue to illustrate that the mycelium of *P. infestans* could only grow from spores of its own species, De Bary designed his experiments of 1875 and 1876 to determine under what conditions, if any, *P. infestans* might produce oospores. In one experiment, he placed a number of tubers showing the presence of mycelium in spring water.[61] The resulting growth resembled that of mycelia exposed to moist air, except that the conidia germinated before detaching from their branches. But when the tuber and mycelium died, de Bary could find no trace of oospores on the surface of the tubers. He obtained the same results after placing a series of infected tubers in moist soil. These experiments ruled out high exposure to water as a potential factor that might induce the

[59] de Bary, 1876, pp. 242–244.

[60] *ibid.*, p. 248.

[61] *ibid.*, p. 249.

production of oospores, reaffirming that *P. infestans* could not mature in excessively moist environments.

De Bary's next set of experiments was more complex. He hypothesized that *P. infestans* produced its oospores in the interior of tubers rather than on surfaces that were exposed to air. To test this, he drew upon the knowledge that tubers infected with *P. infestans* could still produce healthy shoots. As the shoots grew, the stores of starch within the tuber were depleted, leaving behind cells filled with "watery liquid." Ultimately, the interior of the tuber collapsed and decayed.[62] In infected, non-sprouting tubers, the mycelium was predominantly present in the "periphery or outer portion" of the tissue and did not permeate deeply into its interior. But in sprouting tubers, de Bary observed, new hyphae invaded the watery interior tissue, where they grew "very luxuriantly." When this growth did not result in the tissue discoloration indicative of infection in potato tubers, de Bary suggested that "the luxuriant branching of the fungus in the sprouting potato was for the purpose of forming oospores," not normal vegetative growth.[63] Because the hyphae were reproductive structures, not the vegetative filaments of the mycelium that drew nutrition from the plant cells, they did not induce the same blackened spots indicative of necrosis in the tissue. If de Bary were correct, the germination of these oospores, formed in the interior tissue of the collapsing tuber, would coincide with the maturation of the potato plant itself.[64] This would identify the missing stage in the development of *P. infestans,* and offered a concrete explanation as to how *P. infestans* survived from one growing season to the next.

With this in mind, in early 1875 de Bary inoculated a sampling of potatoes by sowing fresh conidia on the eyes of potato tubers and placing them in moist air under a glass bell.[65] He observed the deterioration of the tubers, then planted those tubers in which he had confirmed via microscopic examination the presence of *P. infestans*. When the tubers sprouted, he noted that some of the shoots were healthy and some exhibited symptoms of infection. He sampled the tubers at various stages of decay and examined them, but still found no oospores in their tissue.

This dealt a temporary blow to de Bary's hypothesis. He had previously noted the presence in these tubers of a fungus "with oogonia,

[62] *ibid.*, p. 250.

[63] *ibid.*

[64] Oospores *could* overwinter, but this was not a necessary stage of the life cycle; they would also germinate immediately if exposed to healthy plant tissue.

[65] de Bary, 1876, p. 251.

antheridia, and oospores."[66] Because these structures were morphologically similar to and found in tissue that had been discolored by *P. infestans,* de Bary thought that perhaps he had at last identified his "winter spores." Yet de Bary did not take these morphological similarities as absolute proof; instead, he conducted another set of experiments to confirm that he had isolated oospores of *P. infestans.* He began by sectioning a tuber in which he had found numerous oospores of this second fungus, then placed the sections in water on glass plates and waited until they germinated. As he explained, "The proof here required could only be obtained by ascertaining if the young plants ("germ-tubes") and zoospores would grow on a suitable nidus or substratum into undoubted *Phytophthora.*"[67] After sowing his germinated oospores on fresh tissue from both the potato plants and tubers, however, he saw that,

> The formation of the zoospores was easily confirmed in these sowings. But nowhere did the young plants ("germ-tubes") advance beyond the stage of development which they reached on the glass, nowhere did they penetrate into the interior of the living parts of the plants, and nowhere did they develop mycelium. This result, repeatedly confirmed with certainty, could mean nothing else than that these oospores did *not* belong to *Phytophthora,* but to another fungus, which apparently had entered into the already dead tissues of the tuber while it was still in the ground.[68]

De Bary identified this fungus as a species of *Pythium,* and confirmed this by sowing the germinated tubules on dead tissue taken from insects. *Pythium* produced a "splendid mycelium" and later formed oospores in the interior of the tissue that were morphologically identical to those de Bary had found earlier in the potato tissue. He concluded that the *Pythium* had entered the tuber as a secondary infection. Because this particular species had not been previously described, de Bary named it *Pythium vexans* in honor of the 2 years he had spent working through its puzzling characteristics.[69] Despite his best efforts, de Bary never did find oospores, and the sexual stage of *P. infestans* was not identified until the mid-1950s.[70]

[66] *ibid.*, p. Oogonia are female gametes, while antheridia are organs containing male gametes.

[67] *ibid.*, p. 254.

[68] *ibid.*

[69] *ibid.*, p. 255.

[70] Gallegly and Galindo, 1958.

What's In A Name? Physiology and Its Taxonomic Implications

De Bary's efforts to find sexual stage of *P. infestans* illustrate the importance of physiology in establishing taxonomic categories. Through his experiments, he identified a set of morphological and reproductive differences that could be used to distinguish *P. infestans* from species of *Peronospora* and other genera. The conidiophores of *P. infestans,* for example, were distinguishable from those of other *Peronosporae*. De Bary noted that, while the conidiophores of *Peronospora* bore "a solitary conidium" that was "never followed by a second," the conidiophores associated with late blight of potato had

> not one but several conidia successfully formed at the end of each branch of the tree-like conidiophore....such swellings do not occur in the stems of the true *Peronospora*, but are a sure empirical character of the *Phytophthora*. Among the well-known forms of the family, the potato-fungus, *Peronospora infestans*, Mont., is the only one which has this peculiarity.[71]

From the morphological and developmental evidence he had amassed, de Bary concluded that *P. infestans* was significantly different from the other members of *Peronospora* so as to warrant reclassifying the organism. He wrote, "It may therefore be distinguished as *Phytophthora infestans*," a name he selected for its origins in the Greek "*phtheíro*," with its connotations of death and destruction.[72] De Bary also created the genus specifically for this organism, though other species of *Peronospora* were later moved into *Phytophthora* as well.[73]

Again, the organism bore at least five names in the 44 years between Fries's diagnosis in 1832 and de Bary's creation of the genus *Phytophthora* in 1876. That de Bary saw fit to reclassify it based on its physiology *and* morphology – and that his name has remained in use for over a century – reflects the larger philosophical and methodological changes that German academic botany underwent in the nineteenth century. Until Schleiden's physiological approach began to take root in the late 1830s and early 1840s, taxonomic criteria had reflected botany's emphasis on comparative anatomy and biogeography. As botanists' understanding of plant physiology grew, however, the means by which botanists established taxonomic relationships expanded to include matters such as nutrition, reproduction, and environmental require-

[71] de Bary, 1876, p. 240.

[72] *ibid.*

[73] Waterhouse, 1970.

ments as well as their form and development. Rather than classifying organisms on the basis of their physical characteristics, botanists began to consider differences in processes – matters such as how organisms reproduced or obtained nutrients became important factors in determining the most appropriate taxonomic schema. De Bary's own reclassification of *P. infestans* from *Peronospora* to *Phytophthora* was part of this gradual shift in criteria.

The early genus name of *Botrytis*, for example, was assigned for the organism's (supposed) physical similarity to other members of the genus. The species name (which changed even more frequently than did the genus) allowed for more individualized description of its properties – Marie-Anne Libert, a Belgian mycologist, proposed *Botrytis vasatratix* in August 1845 to acknowledge the devastation the fungus caused. A few weeks later, this name was mistakenly given as *Botrytis devastatrix*, and later that fall Charles Montagne dubbed it *B. infestans*. In 1852, however, Robert Caspary insisted that the organism was rightly included among the *Peronosporae*. Although he acknowledged that the two genera were morphologically similar, he recognized one crucial difference: *Botrytis,* as a genus, are facultative parasites – that is, they can grow on either dead or living tissue. The *Peronosporae*, as Caspary and, later, de Bary, had observed, are obligate parasites – meaning, they are incapable of growing on dead tissue and require a living host. Caspary regarded this as reason enough to reclassify the organism.

De Bary's experiments in fungal development and physiology led him to question and ultimately revise the taxonomic categories to which *P. infestans* belonged. His research identified new characteristics of the organism that were not dependent upon anatomy or morphology, but rather on physiological requirements such as exposure to moisture, reproduction, and the health of the host. The environmental parameters within which an organism grew optimally, the source of its nutrition, and how it reproduced offered insight into the place of *P. infestans* in taxonomic systems, and de Bary identified multiple characteristics that suggested that his "potato-fungus" was not, in fact, rightly included among the *Peronosporae*. Rather, he concluded, it was more accurately included within its own genus.[74]

Admittedly, de Bary cited morphological factors as his first reason for creating *Phytophthora* as a new genus. "In *Peronospora,*" he noted, "the conidiophores occur singly, or in small bunches:" the conidiophores were branched, and on each branch "a solitary conidium is formed, and this is never followed by a second." But in *Phytophthora*, the conidio-

[74] de Bary, 1876.

phores produced "not one but several conidia successively formed at the end of each branch." As mature conidia dropped off, they left behind a series of swellings along the branches – the tell-tale knobbles that distinguished the potato-fungus from other fungi. Because this organism was "the only species that showed this peculiarity," de Bary stated, it "may therefore be distinguished as *Phytophthora infestans*."[75]

Both Caspary's and de Bary's reclassifications were the direct result their emphasis on physiology over anatomy and appearance. The widespread adoption of "*Phytophthora infestans*" therefore carried with it an implicit acceptance of de Bary's conclusions and the investigative methods through which he arrived at them. It is important to note that the two names that this organism bore the longest, Caspary's *Peronospora* and de Bary's *Phytophthora*, were those based primarily upon physiological, not morphological, characteristics. Indeed, de Bary's taxonomy remains unchanged.[76] In this way, tracing the accepted nomenclature of *Phytophthora infestans* also traces the accepted methods for understanding the organism.

Conclusion

The Académie did recognize de Bary's "Recherches" as having helped clarify the question of spontaneous generation by presenting "new facts about the generation of parasitic fungi, and particularly in understanding the manner in which they are introduced onto the plants on which they live."[77] Although the Commission awarded the Alhumbert medal to Pasteur, it agreed that de Bary's experiments were still a substantial contribution to research on reproduction in lower plants (*végétaux inférieurs*) and awarded him an honorable mention and 1000 *francs* for his efforts.

Though de Bary is widely known for identifying *P. infestans* as the causal organism of late blight of potato, the content of his experiments have attracted little historical attention. Yet when considered as an argument about the generation of living things, de Bary's interest in *P. infestans* seems less exclusive to the practical problem of plant disease and more derivative of the philosophical interests embodied in the Académie des Sciences. The combined criteria of the 1856 Grand Prix and 1859 Alhumbert questions lent themselves well to de Bary's

[75] *ibid.*, p. 240.

[76] Gallegly and Galindo, 1958, p. 274.

[77] Académie des Sciences, 1862, p. 979.

meticulous observations of the complete life cycle of *P. infestans*; by the completion of his initial experiments in the 1860s, he was able to answer many of the philosophical and questions that had been under debate: he knew what the organism was and how it behaved on host tissue, its origin and how it developed, and, most important, how it caused disease symptoms.

These experiments garnered him a second-place award from the Academie des Sciences in 1862, a reputation as an eminent mycologist and experimentalist, and, later, the title of the "father of plant pathology" from many enthusiastic (if somewhat triumphalist) practitioner histories. But he was not the first to observe the progression of late blight or to experiment with its cause; he drew heavily upon (and gave full credit to) the work of Berkeley, Montagne, Speerschneider, and many others who had hypothesized causal relationships between fungi and plant disease. Nor was he the first to show the infiltration and colonization of plant tissue by cryptogams, the germination of fungi from spores, or the spread of disease through an experimental plot – these honors belong to other botanists, some of whom preceded de Bary by a half-century in their endeavours. His innovation was in mapping the life cycle of *Peronospora infestans* against the symptoms of late blight in a way that allowed him to identify the mechanism of infection and proliferation of the organism in a healthy host – and, in doing so, to offer the most complete explanation for the onset and spread of the most destructive plant epidemic known to Europe while also providing experimental evidence against spontaneous generation. He did, in short, exactly with the Académie des Sciences had expected; that he placed second to Pasteur is no denigration of his experimental technique nor his argument.

Just as de Bary's research shows the influence of institutions in articulating research questions, the multiple efforts he and other botanists made to accurately classify *P. infestans* are indicative of an overall change in the focus of nineteenth-century German botany. Whereas the organism's early designations owed much to the traditions of natural history (specifically, comparative anatomy), Caspary and de Bary considered traits that acknowledged the organisms' life processes – notably, how and where it obtained its nutrition, its environmental requirements, and its means of reproduction. De Bary did use anatomical traits in trying to classify *P. infestans*, but comparative anatomy was only one component of his overall approach. Only in the light of de Bary's experimental evidence of the germination, growth, and reproductive maturation of *P. infestans* did these differences warrant the

creation of a new genus. It was physiology, not anatomy, that provided the deciding criteria; de Bary's insistence that such characteristics could distinguish between visually similar organisms was an indication of the rising authority of plant physiology in determining the boundaries between genera and species.

De Bary's use of plant pathogenic fungi to put to rest doctrines of spontaneous generation (and his portrayal of the impossibility of spontaneous generation as support for the fungal theory) illustrates the strong implications that seemingly philosophical questions could hold for practical problems. His research also shows the extent to which physiology and experiment – not simply observing an organism, but manipulating its conditions as well – could contribute to understanding both problems simultaneously. This philosophical and methodological overlapping of spontaneous generation and plant disease also suggests that the disciplines we now regard as applied (plant pathology among them) in origin and goal may have been highly influenced by the broader philosophical questions of nineteenth-century biology. The interest scientific institutions showed in these questions – as seen through prize questions posed by the Académie des Sciences, for example – may well have much more to the delineation and shaping of the applied sciences than previously recognized.

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