Louis Sambon and the Clash of Pellagra Etiologies in Italy and the United States, 1905–14

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ABSTRACT

This article explores the extent to which the bacterial concept of disease acted as an obstacle to the understanding of deficiency diseases, by focusing on explorations into the cause of pellagra in the early twentieth century. In 1900, pellagra had been epidemic in Italy for 150 years and was soon to become so in the United States, yet the responses of medical investigators differed substantially. To account for these, the article reconstructs the sharply contrasting reactions to a provocative theory proposed by Louis Sambon. Applying a tropical diseases approach to pellagra, Sambon argued that pellagra had nothing at all to do with maize consumption, as the Italians had long thought, but was caused by the bite of a parasite-carrying insect. Italian pellagrologists, involved in a dogmatic quest for a toxin in maize, and with pellagra rates there on the decline, marginalized the Sambon hypothesis. By contrast, in the United States, with pellagra on the rise, the dominant infectious paradigm put Sambon center stage, his proposed etiology shaping the earliest American investigations. When the deficiency disease concept gained currency in 1913, the relatively closed world of Italian pellagrology was wrong-footed, while the more open-ended U.S. community was better able to follow up the new lead. The article discusses what these shifts and the resulting controversies reveal about the medical contexts. The actor-centered approach, with reaction to Sambon’s intervention as a kind of test-case, is the key to understanding these controversies and why they mattered.

KEYWORDS: pellagra, history, Louis Sambon, disease etiology

On 19 December 1910, Bergamo’s main newspaper, L’Eco di Bergamo, reported on a new theory claiming to explain the causes of pellagra.1 The disease was then epidemic

1 Anon. [Un medico condotto], “La riabilitazione della polenta. La legge sulla pellagra ed i mugnai,” L’Eco di Bergamo, 19–20 December 1910, (287). 3. All translations are my own unless indicated otherwise.
in much of northern Italy, with the Bergamo province being one of the worst affected. The news thus merited one-and-a-half columns of the four-page newspaper. Signed by “a district physician” (un medico condotto), the article was entitled “The Rehabilitation of Polenta,” for the theory asserted that maize and a maize-based diet had nothing whatsoever to do with pellagra. It meant that local people should be able to eat polenta to their heart’s content and that farmers and producers would be able to cultivate and trade maize unencumbered by the restrictive policies that Italy’s recent anti-pellagra laws had put in place. The new theory identified a small gnat as the culprit, complete with the Latin tag of Simulium, which spread a harmful protozoan from person to person. The originator of the theory, the article reported, was an English doctor named Louis Sambon, lecturer at London’s School of Tropical Medicine; most of the rest of the article was devoted to a Progress Report that Sambon had written on the subject, translating the Report’s conclusions.²

Although this newspaper article was signed by a physician, there was no attempt to evaluate the new theory from a medical point of view. Protecting Bergamo’s preeminence as a milling center, supplying five-eighths of Italy’s maize flour,³ seems to have been the author’s main concern. The medical reaction, when it came a few weeks later, was contemptuous. In a leading article in the Rivista pellagrologica italiana, one of that journal’s co-editors, Giuseppe Antonini, praised L’Eco di Bergamo for bringing Sambon’s theory to the attention of Italians.⁴ Antonini quoted extensively from the article’s translation of the Progress Report, even as he proceeded to dismiss its conclusions, point by point. Antonini objected most of all to Sambon’s confident assertion that Cesare Lombroso’s spoiled maize theory was wrong, that all the recent measures taken as a result were useless, and that even if every grain of spoiled maize were to disappear from Italy, pellagra would continue to exist as before. “I confess, I thought I was dreaming,” was Antonini’s reaction, before pointing out how the number of pellagra sufferers in Italy had declined steadily since 1902, so that in the period 1906–8, the number of pellagrins was half of what it had been in 1900–2.

Across the Atlantic Ocean, in the United States, the medical reaction to Sambon could not have been more different. As a professor of medicine in Atlanta, Georgia, put it: “With the publication of Sambon’s ‘Progress Report’ in 1910, the investigation of pellagra really began. Before that time, men studied a cereal [i.e., maize], and thought they were studying a disease.”⁵ In a full-page article in the New York Times Sunday magazine section, which announced a privately endowed study of the disease in the U.S. South, Sambon was credited with having “proved almost indubitably that pellagra is an insect-borne disease, and that probably corn or maize does not enter

² For the Report itself, see Louis Westenra Sambon, “Progress Report on the Investigation of Pellagra,” J. Trop. Med. Hyg., 1910, 13, 271–82, 287–300, 305–15, 319–21 (also printed in book form with the same title, London: Bale and Danielsson, 1910).
³ J. W. Babcock and W. B. Cutting, Pellagra (Washington, DC: Government Printing Office, 1911), 37.
⁴ Giuseppe Antonini, “La riabilitazione della polenta,” Rivista pellagrologica italiana, January 1911, 11(1), 2–6.
⁵ Stewart Roberts, “The Analogies of Pellagra and the Mosquito,” in Transactions of the National Association for the Study of Pellagra: Second Triennial Meeting, at Columbia, South Carolina, October 3 and 4, 1912 (Columbia, South Carolina: R.L. Bryan, 1914) [henceforth as Transactions], 291. Available online at: www.archive.org/transactions.
into the problem at all." Sambon’s theory “has been received with incredulity in some quarters, and is calculated, eventually, to create consternation in other medical circles when the news reaches them,” the article reported. Nonetheless, and despite continuing uncertainty about the causes of pellagra, it had won many “converts,” particularly in the United States.” The article hailed Sambon’s Progress Report as “probably the most important contribution to the literature of pellagra,” and it ended, like the earlier one in L’Eco di Bergamo, by quoting its conclusions verbatim, granting Sambon the final word on the matter.

For all this, and as Kenneth Carpenter noted several decades ago in an edited collection of readings dedicated to pellagra, “time has not been kind to Sambon”: his ideas, so “eloquently” expressed, “have proved quite baseless.” There are, however, several reasons why I think studying how one “wrong” theory of pellagra (Sambon’s) clashed with another equally wrong theory (Lombroso’s) can be both revealing and historically important. The first of these concerns the history of work on deficiency diseases. Pellagra would eventually be classified as a deficiency disease, but only after medical investigators were finally forced out of the bacterial rut into which they had dug themselves. As a theory of disease, the germ concept was “vastly more successful than any previous medical concept”; however, its very dominance in medical thought “proved a major barrier to the recognition and study of deficiency diseases.” Did bacterial theory effectively side-track research on pellagra and diet for many years, in the interests of medical investigators, public officials, and maize producers? The same claim has been made for the investigation of beriberi, that the deficiency concept would have been more readily accepted had there been no bacterial theory.

The philosopher Codell Carter has dismissed this contra-factual claim, by stressing “the variety of causes contemporary researchers were willing to entertain.” Rather, in Carter’s view, the deficiency concept and the bacterial theory belonged to the same “scientific research programme,” with its general focus on etiology and the search for causes. This unity is certainly evident when we survey the range of work then being done at an international level, across the different diseases, as Carter does. But when we shift our gaze to the controversies and power structures as they played out at a strictly national level, the picture seems much more divided and confrontational. Sambon’s intervention into the world of Italian pellagrology brought the question of pellagra’s etiology back on to the table, in a way it had not been for twenty-five

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6 “Commission to Be Sent South to Fight Pellagra,” New York Times, Sunday magazine section, 24 September 1911, 11.
7 Kenneth Carpenter, ed., Pellagra (Stroudsburg, Pennsylvania: Hutchinson Ross, 1981), 5.
8 Aaron Ihde and Stanley Becker, “Conflict of Concepts in Early Vitamin Studies,” J. Hist. Biol., 1971, 4(1), 16.
9 Elmer McCollum, A History of Nutrition (Boston, Massachusetts: Houghton Mifflin, 1957), 225. Kenneth Carpenter offers a more nuanced interpretation, in Beriberi, White Rice, and Vitamin B: A Disease, a Cause, and a Cure (Berkeley: University of California Press, 2000), 196–98. The most recent, and most socially contextualized study, will be discussed below: Alexander Bay, Beriberi in Modern Japan: The Making of a National Disease (Rochester, New York: University of Rochester Press, 2012).
10 K. Codell Carter, The Rise of Causal Concepts of Disease (Aldershot: Ashgate, 2003), 179–80, 193–94.
years. It is no accident that this decade (1900–10) saw more pellagra studies published in Italy than any other.\textsuperscript{11} Established interests were challenged—not just in the production of medical knowledge, but in the more materially lucrative production of maize itself—in ways that paralleled the understanding of the role of husked rice in the etiology of beriberi in Japan.

If, as we shall see, the Italian reaction to Sambon’s provocations says much about the continuing dominance of the Lombrosian theory there, to the virtual exclusion of alternative explanations, its enthusiastic reception in the United States is suggestive of the very different medical context there. In the one country, pellagra had been known for centuries and a theory of its causation was well entrenched; in the other, pellagra had just appeared, sparking a flurry of interest and relative open-mindedness. The very different research agendas and medical power structures in the two countries determined both the reaction to Sambon and the direction of medical research that was carried on in its wake. The riddle of a disease that was on the wane in Italy just as it started to rage in the United States still awaits investigation today; the puzzle was that much greater a hundred years ago. It is worth remembering that at the beginning of the twentieth century, there was still no cure for pellagra, and there was still much about it that eluded understanding, regardless of the theory: its seasonal appearance and spontaneous remission, the severe insanity it could cause, and why it seemed predominantly to affect the poor.

Paradoxically (and somewhat tragically), pellagra is an example of a disease for which there were more or less effective preventive measures available—though not necessarily enacted—long before there was a complete understanding of the disease and an exact cause identified (in 1924). From the time of the earliest Italian explorations of pellagra in the mid-eighteenth century, when disease categories were still symptomatic (based on evident symptoms), through to the rise of etiology as an explanatory concept in the late nineteenth century and attempts to identify specific causal agents, most theories about pellagra’s causation were at best partial and at worst wrong. What is at issue in this study is what these shifts and the resulting controversies reveal about the medical context. The actor-centered approach I adopt here, with reaction to Sambon’s intervention as a kind of test-case, is the key to understanding these controversies and why they mattered.

PELLAGRA IN ITALY AND THE TOXICOZEIST HEGEMONY

As one of Italy’s leading pellagrologists, Antonini did not take kindly to Sambon’s claim that the spoiled maize theory was “the official one, accepted by statisticians who have never seen a pellagra case, laboratory investigators who have studied only damaged maize, and doctors in insane asylums and pathologists who have never visited the places where pellagra is rampant.” Antonini rebutted with reference to Lombroso’s own life-long efforts to study and eradicate pellagra, before turning to the efforts of local doctors. Antonini argued that who better to diagnose pellagra than

\textsuperscript{11} Figures based on Costanza Bertolotti, \textit{La pellagra: bibliografia degli studi, dal 1776 al 2005} (Mantua: Istituto mantovano di storia contemporanea, 2009), 12.
the person “who has lived in the place where pellagra has been endemic for centuries, who has, because of his profession, to follow the sufferer in all his phases and who knows his family background.” Antonini refuted Sambon’s claim that district physicians, closest to the epidemic, never subscribed to the spoiled maize theory, describing their efforts over the past two decades to combat it, and asking rhetorically what measures had been taken against the *Simulium* in recent years to account for pellagra’s decline. Sambon may have been justly famous for his work on malaria and sleeping sickness, Antonini admitted; but his view of pellagra had been shaped too much by his work in tropical climes. Pellagra was linked to the consumption of spoiled maize and no “fortuitous coincidence” would demonstrate otherwise, canceling out the many thousands of observations made over the years.

To understand the widely divergent views of Sambon and Antonini with regard to the Lombrosian etiology of pellagra, and why Antonini perceived Sambon as such a threat, we must take a step back. Pellagra had a long history in Italy. The earliest detailed descriptions of the disease called “rough skin” (*pelle agra*) by the peasants who suffered it, date from the 1770s. Pellagra manifested itself by a reddening of the skin like sunburn, which progressed to symmetric scaly eruptions on both sides of the body. A variety of symptoms followed—stomach upset, diarrhea, debility, and vertigo—culminating in chronic cases in severe mental illness. It has been estimated that something like one-third to one half of insane asylum patients in affected areas were victims of pellagra. Those who survived suffered regular outbreaks of the disease each spring, the symptoms getting worse every year, and often perished after years of repeated incidents.

And yet the causes of pellagra remained the matter of conjecture for some time to come; so much so that even in 1911, the U.S. vice-consul in Milan, Bayard Cutting, could refer to it as (quoting an unreferenced French study) “des malades sans une maladie.” Ascertaining the exact etiology of the disease was the main stumbling block from a public health point of view. By the end of the nineteenth century, the “Lombrosian hypothesis” had come to dominate Italian medicine, exercising control over the provincial and national pellagrological conferences and the discipline’s journal, the *Rivista pellagrologica italiana*. The theory proved to be flexible enough to adapt itself to the latest science, attracting followers from different specialisms, and so survive till the 1920s. The author of the theory was Cesare Lombroso, who would achieve international renown (and later, infamy) as the originator of criminal anthropology, but was a visiting lecturer in mental illness at Pavia University when he first undertook his study of pellagra. In Pavia, Lombroso was also director of the insane ward at the city’s hospital, and most of his experiences regarded pellagra sufferers made mad by the disease. Between 1863, when he first noted a link between the consumption of maize and the spread of pellagra in the Italian region of Lombardy, and his death in 1904, Lombroso published over sixty articles and books on the

12 Simone Botti and Egidio Priani, “Il trattamento della follia nel morocomio veneziano di San Servolo (1840–1860): cura fisica e cura morale,” in *Lo sguardo psichiatrico: studi e materiali dalle cartelle cliniche tra Otto e Novecento*, ed. R. Panattoni (Milan: Bruno Mondadori, 2009), 258–68.

13 Babcock and Cutting, *Pellagra*, 15.
disease, although he was best known for his 1869 monograph relating his own “clinical and experimental studies of the nature, cause and therapy of pellagra.”

The association with maize was clear enough to many observers, a theory known generically in medical circles as “zeism,” from the botanical name for maize, *Zea mays*. The poor peasants of Lombardy and the Veneto, especially landless day-laborers and tenant farmers, were reduced to a diet of maize, in the form of maize polenta and maize bread, and little else, especially during the winter months. Zeism was a broad church. But for Lombroso, it was not a question of maize being deficient at a dietary level when it was used as a subsistence food, as other investigators, beginning with Filippo Lussana, had suggested; rather it was the quality of the maize consumed, since much of it was actually harmful. According to Lombroso’s laboratory analyses, toxic substances formed within poorly dried or unripened maize, and this “diseased” or spoiled maize, when ingested, was responsible for pellagra.

The spoiled maize or toxicozoeist theory was derided at first, but soon came to dominate medical discussions. It was firmly within the still evolving microbiological paradigm, more interested in examining the properties of invading agents under the microscope than actual patients. This was even truer of Lombroso’s followers and successors, who focused on the search for the micro-organism responsible. The toxicozoeist theory had laboratory science on its side, allowing it to occupy a hegemonic position. Just as importantly, the theory would become official, adopted by the state in its belated response to pellagra. It suited the medicine of the period, which was becoming increasingly successful at finding external agents for diseases. And it suited government, which sought a clearly identifiable enemy and practicable solutions. But it came up against the interests of landowners, millers, and merchants dependent on maize production. In the end, the national government did nothing, as a series of modest bills proposed in the 1880s by the agriculture ministry failed to meet with parliamentary assent, despite the presence of an estimated 100,000 pellagra cases.

It would take until 1902 before vested interests were overcome and a pellagra bill became law. By this time, new forces were active, in the form of an increasingly organized agricultural workforce, less likely to be paid in kind (that is, in maize), and a Permanent Interprovincial Pellagrological Committee. The political climate had also changed. A new liberal-left government, under Giuseppe Zanardelli, with the moderate Giovanni Giolitti first as interior minister, was more receptive to social issues. The legislation of 21 July 1902 enabled a more systematic response, focused around the Lombroso-inspired battle “alle muffe e alle truffe” (against molds and frauds). The legislation consisted of nineteen articles, targeting unripe or spoiled maize and the products made from

14 Cesare Lombroso, *Studi clinici ed experimentalis sulla natura, causa e terapia della pellagra* (Bologna: Fava and Garagnani, 1869).
15 Edward Jenner Wood opted for “zeitoxic” as a translation of the Italian tossicozeista: in Wood, *A Treatise on Pellagra for the General Practitioner* (New York and London: Appleton, 1912), 62.
16 Claudio Pogliano, “L’utopia igienista (1870–1920),” in *Storia d’Italia, Annali 7, Malattia e medicina*, ed. Franco Della Peruta (Turin: Einaudi, 1984), 611.
17 Ministero di Agricoltura, Industria e Commercio, *La pellagra in Italia, 1879*, in the *Annali di agricoltura*, Rome, 1879, (18), 324–25.
them, recognizing the provincial pellagrological commissions, obliging towns to keep regularly updated lists of pellagrins, and ensuring the hospitalization of the most serious and needy cases.18 It also gave new impetus to preventive and therapeutic initiatives, and led to the setting up of soup kitchens, health stations, and pellagra hospitals. However, with the area of land dedicated to growing maize never higher, the 1902 law stopped short of interfering with maize cultivation, a measure considered too radical. Nor did it fund all of the proposed initiatives, whose costs had to be met by local authorities.

The steady decline in pellagra numbers in the years following the 1902 law appeared to vindicate the Lombrosian theory.19 And, with hindsight, there is no doubt that some aspects of the law—especially in the treatment of sufferers and those at risk—had a positive impact. (It would not be the only time in Italian medical history that the “right” actions would be taken for the “wrong” reasons.)20 This apparent success bolstered the hegemonic position occupied by toxicozeists in Italy. This was so even if, as vice-consul Cutting put it, “the doctrine is, in the present state of science, insusceptible of direct proof and of direct disproof.”21 This was “because it is impossible to show that any given patient whose food was corn ate only healthy corn, whereas it is easy to demonstrate the presence in spoiled corn of a toxic substance.” Moreover, “the symptoms of pellagra are so varied, even in Italy, that they impinge frequently on those of other maladies.”22 If the Lombrosian doctrine could not readily be disproved, it could not be proved either. “Experiments with the poison of spoiled corn have indeed induced serious, and even fatal, results on all kinds of animals, and on human beings; but they have not induced the precise disease, pellagra,” Cutting noted. “Nor has it been conclusively shown that the poison enters into the human system readymade, and not in the form of a bacterium.”23

Despite this serious structural limitation, from 1902, issue after issue of the Rivista pellagrologica italiana charted the successful effects of the law based on the doctrine. There was a regular column devoted to its application, reporting province by province on different initiatives in the implementation of the law, as well as regular articles devoted to studies of the chemical composition of spoiled maize. There were occasional expressions of doubt—that not enough was being done to improve peasants’ living conditions, to track down house-bound pellagrins unknown to the district physicians, to check on the activity of millers, or to ensure the sale of spoiled maize was

18 Antonini, Pellagra, 115–20; L. Perisutti, “La legge contro la pellagra,” in Atti del secondo congresso pellagrologico italiano: Bologna, 26–28 Maggio 1902, ed. G. Battisti Cantarutti (Udine: Fratelli Tosolini e G. Jacob, 1902), 303–19; Babcock and Cutting, Pellagra, appendices 1 and 2, 46–49.
19 Giovanni Battista Cantarutti, “La pellagra in Italia negli anni 1881–1899–1910,” in Atti del quinto congresso pellagrologico italiano: Bergamo, 9–11 settembre 1912, ed. Cantarutti (Udine: Tosolini, 1912), 471–74.
20 The same thing could be said with regard to early measures against typhus or malaria, effective even if done by following what turned out to be the “wrong” theories. Luigi Faccini, “Tifo, pensiero medico e infrastrutture igieniche nell’Italia liberale,” in Storia d’Italia, Annali 7, Malattia e medicina, ed. Franco Della Peruta (Turin: Einaudi, 1984), 716.
21 Babcock and Cutting, Pellagra, 19.
22 Ibid., 19.
23 Ibid., 20.
actually prosecuted—

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—but these were the exceptions to the rule. Even the author of these complaints pronounced himself “trusting” (fidente) in the local work being done. No voices were raised against the law, not least from the journal’s co-founder and co-editor, Antonini. Antonini had been a pupil of Lombroso’s and resolutely towed the line of “the great master,” “the true father of modern pellagrology,” throughout this period, and continued to do so well into the 1920s, long after most other Italian pellagrologists had abandoned it. Nevertheless, there is no doubting the commitment of someone like Antonini, who campaigned tirelessly against pellagra. Director of Pavia’s provincial insane asylum (manicomio) at Voghera, and from 1911 director of the insane asylum at Mombello, outside Milan (until 1932, two years before his death), Antonini was active in the provincial and national pellagrological conferences and the author of numerous books and pamphlets offering advice on how to understand and combat it.

Writing on the eve of the 1902 law, Antonini stressed that “if, one day, which I hope not far off, the entry on deaths due to pellagra is eliminated from cause of death statistics in Italy,” it will all have been due to Lombroso and the theory that bore his name. By the time of Sambon’s Progress Report eight years later, Antonini was seeing his wish fulfilled and he was not going to give up now. He must have wondered at the fuss over etiology that Sambon’s investigations had kicked up, given recent Italian “successes” in pellagra prevention. He must have considered pellagra to be “understood” as a disease—in the way scurvy was “understood” long before the reason why lemons successfully cured it was explained.

As Antonini told the delegates at the fifth Pellagrological Congress, meeting in Bergamo in 1912, new “doctrines” like Sambon’s “advance threateningly to show the work of the toxicozeists to be, as it were, absurd, and the struggles, sustained victoriously through so many years, to be superfluous.” The risk was that these theories would “undermine the foundations of the ancient edifice without presenting reliable elements for the construction of new ones.”

Antonini countered each of the new theories in turn, which if taken together canceled one another out. Sambon’s was the most hypothetical. In order to be “converted” to it—and the religious language is Antonini’s—we would need positive identification of the responsible parasite and proof of infection. Sambon’s confident tone will lead people into “acting as if a

24 Cesare Salvetat, “Commissione pellagrologica comunale di Asola (Mantova),” Rivista pellagrologica italiana, May 1907, 7(3), 154–59.

25 Giuseppe Antonini, “Il quinto congresso pellagrologico italiano,” Rivista pellagrologica italiana, May 1912, 12(3), 141; and idem, Pellagra: storia, etiologia, patogenesi, profilassi, (Milan: Hoepli, 1902), 35.

26 Antonini, Pellagra, 35, 115.

27 Carter, Causal Concepts, 182.

28 B. Gosio and G. Antonini, “Considerazioni sull’etiologia della pellagra,” in Atti del quinto congresso, ed. Cantarutti, 180–87.

29 Alongside Sambon’s, the new etiological “doctrines” discussed by Antonini and Gosio were Guido Tizzoni’s (which was not exactly new), posit ing a pellagra germ, the Streptobacillus pellagrat, the most complimentary to the Lombrosian theory; Giulio Alessandrini’s, which posited a parasite in drinking water as the cause, and was often paired with Sambon’s; Lussana’s notion of dietary deficiency or “maidism,” a theory whose simplistic appeal among district physicians the authors found worrying; and Raubitschek’s photodynamic theory, which argued for the complicating effects of the sun on people with a maize diet.
conclusion had already been reached in antagonism to what the [1902] law prescribes."
Perhaps with L’Eco di Bergamo’s conclusions in mind, Antonini remarked that the new
theories were “pernicious” in that they could be used by vested interests to argue
against the law. Continued research was necessary into pellagra, in order to “[elimi-
nate] those destructive critics whose only aim is an apparent desire to produce
discord.” Unity was required. The paper was met with “the hearty applause of the
assembly,” according to the proceedings.30 From the organization of his defense
down to the very language used, Antonini’s stance was the standard one in scientific
controversies: he stressed the uncertainties inherent in the opposing theories, exagger-
ating their weaknesses and inconsistencies, in order to counter his rivals and ensure the
continued predominance of the toxicozeist project.

Was Antonini consciously engaged in producing scientific uncertainty? There are
striking parallels with Japanese debates over the etiology of beriberi, taking place at
the same time. In Japan, according to Alexander Bay’s lucid analysis, the dominant bac-
teriological theory was being challenged by the deficiency theory, and meeting with the
same sort of establishment opposition as in Italy.31 But I think it would be inaccurate to
speak of “the construction of ignorance” in Antonini’s response to opponents of the
Lombrosian theory in Italy, as Bay does for the response of Tokyo doctors to the defi-
ciency theory. The reference here is to the tobacco industry’s deliberate and longstand-
ing policy of scientific misinformation regarding the links of cigarette smoking to
cancer, as described by Robert Proctor.32 Antonini was not an ally of maize-producing
interests in Italy; indeed the 1902 antipellagra law had put curbs on the production and
sale of maize. But he did strive to protect his own professional interests and status,
marginalizing those who questioned the established orthodoxy.

LOUIS SAMBON: A MAN AND HIS THEORY
If the leading Italian pellagrologists were a self-confident lot in 1910, so too was Louis
Westernra Sambon. Sambon had grown up in Italy, mostly in Milan, the son of a
Franco-Italian antiquarian father and an English mother. In 1884, he matriculated at
the University of Naples, the same year a cholera epidemic struck the city, and the
young medical student was called upon to provide assistance.33 The experience may
have sparked Sambon’s interest in epidemiology. In any case, he contributed to the
“white men in the Tropics” debate, writing an article for the British Medical Journal
on the acclimatization of Europeans. Sambon’s approach mixed geographical analysis
and epidemiology and the nascent fields of microbiology and parasitology: an approach
that we shall see again in his study of pellagra. He claimed that most illness in the Tropics

30 Cantarutti, ed., Atti del quinto congresso, 187.
31 Bay, Beriberi in Modern Japan, 106–10.
32 Robert Proctor, “Agnotology: A Missing Term to Describe the Cultural Production of Ignorance (and Its
Study),” in Agnotology: the Making and Unmaking of Ignorance, ed. R. Proctor and L. Schiebinger (Stanford,
California: Stanford University Press, 2008), 1–33.
33 Annuario della R. Università degli studi di Napoli. Anno scolastico 1885–86 (Naples: Tipografia della Acca-
demia reale delle scienze, 1886), 182; Gerald Holyroyde, “Obituary: Louis Sambon MD,” Br. Med. J., 12
September 1931, S14–15. On the epidemic, see Frank Snowden, Naples in the Time of Cholera, 1884–1911
(Cambridge: Cambridge University Press, 1995), 181–231.
was microbial in nature, including not just malaria and tuberculosis, but also sunstroke, which he called “siriasis.”

This work brought him to the attention of Patrick Manson, one of the key players in an expanding field grounded in microbiology and parasitology, that of tropical medicine, and who had just founded the School of Tropical Medicine in that same year (1899) in London. This specialism, so closely linked to the colonial enterprise, was intensely nationalistic and fiercely competitive. There was much at stake and contemporaries were aware of the great strides being (and yet to be) made, offering opportunities to gifted, ambitious young men. It was in this context and at this point that Sambon went to England. London was then the medical center of the British empire, where hitherto unfamiliar diseases were encountered and investigated, and knowledge about them generated.

By early 1900, Sambon was hired as an occasional lecturer in the London School, lecturing on epidemiology and parasitology. He proceeded to organize a meeting of British investigators (including Manson and James Cantlie) with their Italian counterparts (including Giovanni Battista Grassi and Angelo Celli) in Rome. In the autumn of 1900, Manson sent Sambon, together with George Low, lecturer at the School, to the Roman Campagna. In the ongoing search for the means of transmission of malaria, Low had just outlined the life-cycle of a vector-borne nematode infection. Malaria and its transmission was then medical science’s most engrossing problem. Ronald Ross, working in India, had written about the mosquito transmission of avian malaria, although clear proof of transmission in humans had not yet been found. The “Rome school” of Grassi and Celli were working on similar lines.

Sambon straddled two worlds. For British researchers, malaria was “over there”: a colonial problem, a tropical disease. For Italian investigators, it was a disease long endemic to their own country, a tragic reality encountered only seven kilometers from the gates of Rome. Sambon evidently cast his lot in with the British, writing exclusively in English and taking part in a British-sponsored expedition. In 1902, two years after mediating amicably between British and Italian colleagues, Sambon sided with Ross in the priority dispute against the Italian zoologist Grassi and the “Rome school” over the discovery of the role of mosquitoes in the transmission of malaria.

34 Luigi Sambon, “Remarks on the Possibility of the Acclimatisation of Europeans in Tropical Regions,” Br. Med. J., 9 January 1897, 61–66; L. Westenra Sambon, “Remarks on the Etiology of Sunstroke (Siriasis),” Br. Med. J., 19 March 1898, 744–48.
35 W. F. Bynum, *Science and the Practice of Medicine in the Nineteenth Century* (Cambridge: Cambridge University Press, 1994), 145–52; Maryinez Lyons, *The Colonial Disease: A Social History of Sleeping Sickness in Northern Zaire, 1900–1940* (Cambridge: Cambridge University Press, 1992), 64–67.
36 Holyroyde, “Obituary”; J. H. Taylor, “Sambon the Man, and His Later Investigations of Pellagra,” South Carolina Med. J., 1913, 6, 599–605, and Transactions, 69–80.
37 Douglas Haynes, “Framing Tropical Disease in London: Patrick Manson, *Filaria perstans* and the Uganda Sleeping Sickness Epidemic, 1891–1902,” Soc. Hist. Med., 2000, 13, 467–93.
38 “A Malaria Conference in Rome,” Br. Med. J., 10 February 1900, 323–25.
39 Ernesto Capanna, “Grassi versus Ross: Who Solved the Riddle of Malaria?” *Perspect. Int. Microbiol.*, 2006, 9, 69; Frank Snowden, *The Conquest of Malaria: Italy, 1900–1962* (New Haven and London: Yale University Press, 2006), 39.
Amidst claim and counter-claim, personal ambition and national pride, the Grassi–Ross dispute intensified when Ross was awarded the Nobel prize that same year (1902). The dispute to one side, the discovery of a vector, combined with a national campaign against mosquito infestation and the free distribution of quinine (a truly effective medicine) helped reduce malaria deaths in Italy. By 1915, the number of deaths had declined from six hundred per million inhabitants to less than fifty. It was one of the great success stories of pre-World War I Italian medicine. The movement to eradicate both malaria and pellagra would follow similar trajectories, with major learned associations, journals, public health campaigns, and national laws launched within a few years of one another at the turn of the century. Pellagra, as we have seen, also declined; but the role of medicine in this would be quite different.

As an epidemiologist, Sambon could position himself as being just ahead of the other sciences, paving the way for them, as it were. Sambon’s first foray into pellagra came in 1905, hot on the heels of his studies of sleeping sickness and malaria. At the seventy-third meeting of the British Medical Association, held in Leicester that year, Sambon voiced his hypothesis on the etiology and spread of pellagra, using a detailed survey of pellagra’s spread to identify a possible vector or agent. Sambon’s “Remarks” on pellagra came in the section devoted to tropical medicine. “[N]o other department of medical science can show such activity as this,” in the words of the BMA’s president, Rupert Bryce, even if its progress was “hindered through want of the necessary knowledge” of “the life-history of parasites and insect hosts.” Mind you, Sambon did not consider himself “hindered” in any way; rather, he bravely stormed ahead with a novel hypothesis relating just these sorts of “parasites and insect hosts” to a disease about which much was already known. Sambon’s “Remarks” were intended to stamp his name on this theory before anyone else got to it, using his now standard methodology. The style is that of an essay: laying out the problem historically and geographically, discussing the dominant zeist etiological theories, then demolishing them in turn, before concluding with what appears to be the only logical solution to the problem—a solution which although glaringly obvious had not occurred to anyone else before.

Maize might come into it, but only indirectly, Sambon remarked: “Probably, therefore, it is in the maize field that the peasant comes in touch with the specific agent of pellagra, and possibly through the agency of some biting fly.” Sambon seems to toss this suggestion out casually, but this is in fact what he has been leading up to. Sambon’s conclusion is circumspect and understated, whilst at the same time self-serving: “If I were asked to suggest a new theory of pellagra, merely as a working hypothesis, I should feel inclined to draw some attention to the many analogies between pellagra and some of the

40 Capanna, “Grassi versus Ross”; Gordon Harrison, Mosquitoes, Malaria and Man: A History of the Hostilities since 1880 (New York: E.P. Dutton, 1978), 102–8.
41 Capanna, “Grassi versus Ross,” 73.
42 Louis Sambon, “Remarks on the Geographical Distribution and Etiology of Pellagra,” Br. Med. J., 11 November 1905, 73rd Annual Meeting of the British Medical Association, 1272–75.
43 Rupert Bryce, Introduction, Br. Med. J., 11 November 1905, 1258.
44 Sambon, “Remarks,” 1275.
protozoal diseases which have been recently worked out.” There is no doubt that for Sambon, the “Remarks” marked the start of a project.

Sambon had gained himself a reputation as an “ideas man.” According to Cantlie, writing in 1908 about the field of tropical medicine, “men of his [Sambon’s] type are rare. We have many observers but few thinkers.”45 Manson was even more impressed, characterizing Sambon as “a magnificent worker, with ideas ahead of the time.”46 Manson was suggesting to Ross that Sambon be given a chance to investigate pellagra, with which “I am sure he would struggle till he got a solution.” With a letter of reference like that, from such an influential and well-connected man as Manson, now knighted and a fellow of the Royal Society, it was no surprise that a “Pellagra Investigation Committee” was soon set up, with the aim of raising a fund of £1,000 to pay for Sambon’s travel “to a pellagrous area to study the topographical distribution and epidemiology of the disease.”47 Sambon could also count on the support of the successful medical entrepreneur and collector Henry Wellcome.48 By this time, Sambon had identified a single insect for investigation, the Simulium reptans, a small blood-feeding sand-fly. Sambon’s proposed visit to Italy was intended to determine whether a sand-fly was the carrier of pellagra and whether pellagra could be defined as belonging to the group of protozoal diseases.49

Sambon departed for Italy on 20 March 1910, even though the committee had thus far raised only one-fifth of the target amount.50 His twelve-week fact-finding trip took him throughout the areas of Italy worst affected, from Bergamo in the north to Perugia in the center. The resulting Progress Report resembles Sambon’s 1905 “Remarks,” albeit on a much grander, and more detailed, scale. Much more could now be based on his own personal findings and “encounters” in areas where the disease was rife. Sambon makes much of the efforts of the “hundreds” of district physicians who he had met with during his investigations, praising their “energy, skill, integrity, and devotion” in

45 James Cantlie, “The Importance of Rational Inductive Methods in Advancing Knowledge,” editorial, J. Trop. Med. Hgy., 1 February 1908, cit. in Taylor, “Sambon the Man,” 599. Sambon had used some of these “ideas” to support a line of clothing designed to prevent climatic disease in the Tropics, in apparent contradiction to his belief that microbes were the sole cause of tropical illness. The clothing, called “Solaro” and manufactured by Ellis and Johns, was first advertised in the journal Climate, in April 1905. Ryan Johnson, “European Cloth and ‘Tropical’ Skin: Clothing Material and British Ideas of Health and Hygiene in Tropical Climates,” Bull. Hist. Med., 2009, 83, 530–60.
46 Letter of 5 November 1909 from Manson to Ross, in Bynum and Overy, Beast in the Mosquito, 473.
47 London School of Tropical Medicine and Hygiene, “Pellagra Investigation Committee,” 1910, GB 0809 Pellagra; “Pellagra Investigation Committee,” J. Trop. Med. Hgy., 1910, 13, 60–63.
48 Wellcome was probably genuinely interested in the results of the research. He had just published a lecture on ergotism and its history, which was also a tie-in to Burroughs Wellcome products: Henry Wellcome, From Ergot to “Ernutin”: A Historical Sketch (Chicago: American Medical Association, 1908).
49 “The Nature and Causation of Pellagra,” The Times (London), 4 February 1910, 4. In 1903, William Leishman and Charles Donovan had both identified a sand fly as the possible carrier of the parasite responsible for the deadly disease kala-azar (visceral leishmaniasis).
50 In the end, Wellcome agreed to pick up most of Sambon’s bill for the expedition. This included “44 bound books & 190 pamphlets, reports & loose articles from papers, etc,” “of very little to anyone but [Sambon] himself” (and to the author of this article). Memorandum from Charles Thompson to Wellcome, 18 July 1911, Wellcome Archives, London, Historical Medical Museum, CO/Ear/842 (hereafter WA/HMM/CO/).
dealing with pellagra, but also noting how their front-line duties had made most of them skeptical of the official stance. One district physician admitted that he did not believe in the maize theory, "but everybody does and there is nothing I can suggest to replace it." And a local health officer "had given up reading Lombroso to take up Manson’s classical text-book of tropical diseases, of which he had an Italian translation."

Sambon is able to present the work of Lombroso as the “old science” against which he must wage his battle. If the label of “Lombrosian theory” is suitable, Sambon argues, it is “not that Lombroso suggested the theory, but because he and his school imposed it with a dogmatism and intolerance inconsistent with the spirit of modern science.” The “school” has marginalized evidence against the theory, like pellagra in infants and children. Considered a rarity by Lombrosians, the presence of such cases served to support Sambon’s own views. Pride of place is given to a further issue: the precise geographical spread of pellagra. In Sambon’s mind, this made pellagra similar to malaria, sleeping sickness, Rocky Mountain fever, “and other diseases known to be transmitted by mosquito-, fly-, or tick-carriers, presenting well-defined habitats.” It explained the unchanging boundaries of pellagra’s “endemic centers,” the predominance of field laborers (and their infants and children) as sufferers (most exposed to the infective agent), the exemption of towns (since sand-flies had a very limited range), the increase in cases following flooding, the seasonal eruption of the disease (spring and autumn, when sand-flies were active), and the areas of the skin particularly affected. In Sambon’s words, “there is not a single fact which the Simulium theory does not satisfactorily cover.” If the protozoan responsible for the disease had not yet been identified, the role of the Simulium in the transmission of pellagra was “almost a certainty.”

THE ITALIAN REACTION
Sambon may have thought his Pellagra Report “to be a model of its kind,” but not all of his colleagues agreed. Ross complained of Sambon’s “insufficient random sampling” in positing the correlation between pellagra and sand-flies. It certainly did not give Sambon the “authority to write to the lay press stating positively that Simulium carries the disease,” an approach Ross believed “produces the worst possible impression. It is not science.” Sambon had written to the Italian press in similar fashion, beginning with newspapers like L’Eco di Bergamo. The result, according to

51 Ibid., 282, 290.
52 Ibid., 290.
53 Ibid., 281. Patrick Manson’s Tropical Diseases: A Manual of the Diseases of Warm Climates was first published in 1898 (London: Cassell); the Italian translation, Manuale della malattie dei climi caldi (Milan: Società Editrice Libraria, 1909–11), was based on the fourth edition, printed in 1907.
54 Sambon, “Progress Report,” 290.
55 Ibid., 297.
56 Ibid., 321.
57 Ibid., 271.
58 Letter of 12 August 1910 from Sambon to Wellcome, WA/HMM/CO/Ear/843.
59 Letter of 11 November 1910 from Ross to Manson, in Bynum and Overy, Beast in the Mosquito, 478–79. On 15 October 1910, The Times (p. 12) had carried a lengthy article entitled “Pellagra: Some Recent Discoveries (From a Correspondent),” the latter no doubt Sambon himself.
M. V. Carletti, lecturer in medical pathology at the University of Padua, was that it “stirred up lively comments and impassioned discussions in the newspapers and among Italian doctors.” For instance, the chairman of Belluno’s provincial pellagrological commission, Luigi Alpago Novello, wrote a spirited criticism of Sambon’s theory for the Venetian newspaper L’Adriatico. Worse still, objected local doctors like Cesare Ceresoli from Brescia, Sambon’s press barrage gave the wrong idea to “local farmers, administrators, industrialists, and merchants,” who were all hoping for the abolition of the 1902 laws regulating “the cultivation, consumption and trade in maize.”

We have already discussed the views of Antonini, enforcer of the official dogma. Many Italian doctors shared Antonini’s reaction, if Carletti’s views were at all representative: “It was impossible for many people to call into question or indeed abandon the whole rich heritage of investigations, knowledge and hypotheses, often brilliant, which from the time of Ballardini and Lombroso, down till our own times, Italian scholars have been accumulating in regards to the relations between pellagra and maize.” The fact that so much about pellagra was still up in the air did not necessarily bother contemporaries, at least not the toxicozeists. After all, Robert Koch had discovered the tuberculous bacillus in 1882, widely accepted as the cause, even if there was still no cure (and would not be until the discovery of streptomycin in 1944). Other Italian pellagrologists, without necessarily agreeing with Sambon, welcomed his ideas as a breath of fresh air. The professor of general pathology at the University of Perugia, Gustavo Pisenti, could find little to support Sambon’s theory in his native Umbria; but he criticized the “Lombrosian orthodoxy” as misguided, creating the ineffectual 1902 law. Pisenti concluded that more epidemiological studies were necessary.

Sambon’s theory was a provocation the Italian state could not ignore. In November 1910, the Italian parliament set up a Ministerial Commission for the Study of Pellagra (Commissione ministeriale per lo studio della pellagra) charged with examining the different etiological theories then being proposed and how they affected the 1902 law. At the Commission’s meeting in Rome on the morning of 12 February 1912—to which Antonini mysteriously sent his apologies—Sambon was given the chance to air his views. For over two hours, Sambon presented his findings, expressing the hope that the Commission would collaborate in his research to establish the role of the Simulium in pellagra infection.

60 M. V. Carletti, “Etiologia della pellagra. Le critiche di L. W. Sambon alle teorie maidiche,” Gazzetta degli ospedali e delle cliniche, 1911, 64, 5.
61 Luigi Alpago Novello, “Discussioni pellagrologiche,” reprinted in the Rivista pellagrologica italiana, 1911, 11 (3), 35–37.
62 Cesare Ceresoli, “I nuovi orizzonti della genesi della pellagra,” Rivista pellagrologica italiana, 1911, 11(2), 19–23.
63 Carletti, “Etiologia della pellagra,” 5.
64 Gustavo Pisenti, L’ortodossia pellagrosa e il semplicismo delle scuole maidiche (Perugia: Tip. Perugina, 1912).
65 As reported in the Rivista pellagrologica italiana, 1912, 12(3), 144–45.
66 The first, undertaken in the summer of 1911, had yielded mixed results. An unsigned letter to Wellcome complained of Sambon’s “lack of staying power and no method whatever.” (Letter of 5 July 1911, WA/HMM/CO/Ear/844.)
The Commission’s chair responded by asking members to undertake a detailed examination of Sambon’s theory and report back on it by spring. From Friuli, Antonini had already reported that “none of the people specifically asked about insect bites could cite factors substantiating the Sambonian hypothesis.” The remaining two sub commissions reported nothing new. For the Umbria subcommission, the bacteriologist Gosio reported on its investigations into pellagra among children and into the Simulium as an agent of infection, “but from the observations made hitherto I could not produce sufficient arguments in favor of this hypothesis.” While the Lombardy–Veneto subcommission reported on recent bacteriological studies of tainted maize by one of its members (Tizzoni) without making any mention whatever of Sambon. The Commission concluded that “it was clear that in the present state of knowledge the strict enforcement of the current pellagra laws must continue.”

This declaration set the tone for the fifth Italian Pellagrological Congress, held in Bergamo in September 1912. Announcing the conference program, the editors of the Rivista pellagrologica italiana were certain that the discussion of the new etiological theories would be of “very notable importance”; but they remained in no doubt that the meeting would have “the merit of removing any doubts raised against the Lombrosian theories, which, even for those not expert in pellagrology, have hitherto had the support and justification of practice.” And that is exactly what happened.

Although Sambon himself could not attend, seven out of eighteen papers in the “medical section” (parte medica) of the meeting were nevertheless specifically devoted to the “new etiologies.” None of the papers which mentioned Sambon’s theory found any support for it. Even a survey of Italian medici condotti (district physicians), so warmly praised by Sambon for their openness to his ideas and condemnation of the zeist position, found no one who favored the sand-fly as a cause. Of the 247 district physicians treating pellagrins at the time of the survey, 87 stated that pellagra was due to a dietary insufficiency (the Lussana theory), 79 that it was due to maize intoxication (the Lombroso theory), 66 that it was due to dietary insufficiency and occasional maize intoxication (the “mixed theory”), 8 that it was due to an infection, 2 that it was related to alcoholism, and 5 various other causes. The variety of opinions held by district doctors, whether due to openness or indecision, was in stark contrast to the Lombrosian orthodoxy prevalent among full-time pellagrologists. But this was never going to worry the Italian medical elite, given the relatively low standing of the poorly paid and

67 Telegram of 17 February 1912 to Thompson, WA/HMM/CO/Ear/844.
68 Giuseppe Antonini, “Escursioni pellagrologiche nel Friuli,” Rivista pellagrologica italiana, 1912, 12(2), 129–32.
69 “Commissione ministeriale per lo studio della pellagra,” Rivista pellagrologica italiana, 1912, 12(4), 162.
70 Bartolomeo Gosio was employed at the public health laboratories in Rome. Gianfranco Donelli and Valeria Di Carlo, I laboratori della sanità pubblica. L’amministrazione sanitaria italiana tra il 1887 e il 1912 (Rome-Bari: Laterza, 2002), 267.
71 “V° Congresso Pellagrologico Italiano,” Rivista pellagrologica italiana, 1912, 12(3), 141–44.
72 Sambon pleaded previous commitments and “a most painful neuritis.” Cantarutti, Atti del quinto congresso pellagrologico, 179.
73 V. L. Camurri, “L’etologia della pellagra nel giudizio dei medici condotti in Italia,” Atti del quinto congresso, 226–27.
generally demoralized condotti. Either way, the link between pellagra, poverty, and maize consumption was evident to almost everyone.

By far the most debated “new” findings at the 1912 meeting were those of Guido Tizzoni, who claimed to have isolated a streptococcal bacillus in pellagrins and, in animals, was able to duplicate the skin lesions typical of pellagra.\(^{74}\) It is another indication of how the Lombrosian, toxicozeist orthodoxy was still able to dominate proceedings and claim the moral high ground. Indeed, at the end of the conference’s “medical section,” a motion was proposed and approved that, in light of the “many theories put forth recently against the Lombrosian concept,” the toxicozeist theory should be reaffirmed as the “correct basis” of the present law and as effective in the prevention of pellagra.\(^{75}\) The 1912 meeting was a monument to Lombroso, which is perhaps fitting since this was the first meeting held after Lombroso’s death (on 19 October 1909). It also announced the imminent completion of an actual stone monument to Lombroso in Verona.\(^{76}\)

In the meantime, the ministerial Commission continued to meet from time to time. It was certainly more open to novelty than the Rivista pellagrologica italiana, which continued to prop up the “Lombrosian edifice.”\(^{77}\) A 1913 report on the Commission’s activities from its inception in 1910 noted how Sambon’s Simulium hypothesis had been the first to be seriously considered and how investigations were still ongoing.\(^{78}\) This was despite not having heard anything further from Sambon and his team, who had requested the Commission’s collaboration in the first place. From this the Commission had to assume that Sambon had no further findings to report.\(^{79}\) In fact, this was more or less the case, but not for want of trying on Sambon’s part.\(^{80}\)

**REACTION TO SAMBON IN THE UNITED STATES**

Pellagra seemed to appear from nothing in the United States in the early 1900s, so fast indeed that it was regarded as an infectious disease. What else could explain its sudden appearance and quick spread?\(^{81}\) Because pellagra was new, there was little of the zeist cultural baggage which weighed so heavily in Italy. It struck at a time when a whole

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74 Guido Tizzoni, untitled presentation, *Atti quinto congresso*, 204–18. Tizzoni was the professor of general pathology at the University of Bologna and specialist in the study of infections and immunology, famous for pioneering the antitetanus serum in 1889–90. His 1913 work, *La pellagra studiata nelle sue cause* (Turin: UTET), was all about the (phantom) *Streptobacillus pellagraceae*.

75 *Atti quinto congresso*, 286–87; “V° Congresso Pellagrologico Italiano,” 189–92.

76 *Atti quinto congresso*, 515–16. The bronze sculpture, by Leonardo Bistolfi, was eventually unveiled in 1921.

77 Antonini, editorial, *Rivista pellagrologica italiana*, 1913, 13(1), 1–2.

78 “Resoconto della Commissione Ministeriale per lo studio della pellagra. Breve riassunto esposto dal Presidente della Commissione,” *Rivista pellagrologica italiana*, 1913, 13(2), 17–24.

79 Ibid., 19.

80 Wellcome agreed to finance another expedition to Italy, involving Sambon and Albert Chalmers, but the trip did not go well. Letters of 15 April and 27 May 1912 from Sambon to Wellcome, WA/HMM/CO/Ear/844.

81 Chris Leslie, “‘Fighting an Unseen Enemy’: The Infectious Paradigm in the Conquest of Pellagra,” *J. Med. Humanit.*, 2002, 23, 187–202; Alfred Jay Bollet, *Politics and Pellagra: The Epidemic of Pellagra in the U.S. in the Early Twentieth Century*, *Yale J. Biol. Med.*, 1992, 65, 211–21; Harry Marks, “Epidemiologists Explain Pellagra: Gender, Race, and Political Economy in the Work of Edgar Sydenstricker,” *J. Hist. Med. Allied Sci.*, 2003, 58, 34–55. Much of the background to developments in the United States can be
range of diseases—anthrax, tuberculosis, diphtheria, typhoid, tetanus, bubonic plague, syphilis—were found to be caused by micro-organisms. It seemed self-evident that all epidemic disease would be found to have a microbial cause. If long experience with pellagra had taught the local, family doctor in Italy that maize and poor diets had to be linked in some way, the average U.S. practitioner thought of pellagra as a disease one “caught”—from family, neighbors, or ancestors.

Pellagra in the United States also coincided with the rise of tropical medicine as a discipline. Successes in the field were palpable. Within the space of a few years, by targeting mosquito populations, according to the latest theories developed by the likes of Koch, Grassi, and Ross, the U.S. authorities had managed to make the Panama Canal area relatively safe for human populations. In 1907, the mastermind behind this success, the U.S. army surgeon William Gorgas, predicted the total elimination of yellow fever and malaria within a few years. “Life in the tropics for the Anglo-Saxon,” Gorgas went on, “will be more healthful than in the temperate zone.” The influence of the “infectious paradigm” thus conditioned early U.S. medical responses to pellagra. What is striking is the way in which attention eventually focused on finding a micro-organism external to maize.

The disease was not recognized at first. As the South Carolina doctor James Babcock, superintendent of the State Hospital for the Insane, put it: “the authors of English and American textbooks . . . have told us, if they told us anything at all about it, that pellagra is an Italian disease that does not occur in our country.” In 1908, a concerned Babcock traveled to Italy to study it in the company of a local senator, Benjamin Tillman. Babcock visited several pellagra hospitals, the better to diagnose and treat the increasing number of cases back home, whilst the senator requested the U.S. vice-consul in Milan, Bayard Cutting, to write a report on pellagra in Italy (which has been quoted on several occasions). Later in the year, the South Carolina State Board of Health held a public conference on the subject, which became something of a scare story in the local press. In 1909, partly because of his expertise, Babcock was elected president of the newly founded National Association for the Study of Pellagra. In 1910, Babcock and Claude Lavinder translated a version of Lombroso’s monumental Trattato profilattico e clinico della pellagra (1892), heavily abridged and published in French by Armand Marie in 1908, with extensive additions and revisions to “to include the latest opinions regarding the possible parasitic origin of pellagra.” But Babcock’s warnings about pellagra’s spread in South Carolina and

found in Elizabeth Etheridge, The Butterfly Caste: A Social History of Pellagra in the South (Westport, Connecticut: Greenwood, 1972).
82 Bollet, “Politics and Pellagra,” 214; Etheridge, Butterfly Caste, 25–26.
83 Leslie, “Infectious Paradigm,” 190.
84 H. B. Allen, Report on Health Conditions at Panama (Melbourne: Commonwealth of Australia, 1913), no pg., in Harrison, Mosquitoes, Malaria and Man, 167.
85 The expression is Leslie’s, “Infectious Paradigm.”
86 J. W. Babcock, “The Prevalence of Pellagra in the United States,” J S C Med. Assoc., September, 1910, 445–49; reprinted in Babcock and Cutting, Pellagra, 10–11.
87 Etheridge, Butterfly Caste, 12–13; Bollet, “Politics and Pellagra,” 213.
elsewhere; his focus on the relationship between poverty, diet, and pellagra; and the threat to the maize business posed by the threat of a maize toxin, made him extremely unpopular. As in northern Italy, vested interests were at stake. The Chicago Post worried that if millers or their customers in the U.S. South ever “get it into their heads that Illinois corn is the cause of pellagra, we shall feel the economic effect of it in this state, world wide though our markets be.”

And when Sambon came along, The American Agriculturalist was relieved to relate the news that pellagra might be insect-borne.

With threats to the maize business acting as a push factor and Sambon’s theory as a pull factor, the State of Illinois set up a commission to study pellagra in 1910. The Illinois Commission’s report, issued the following year, adopted Sambon as a constant point of reference. The latest word in pellagrology offered a scientific foundation for discussions of the transmission of pellagra by U.S. doctors, even if evidence in its support was wanting. Maize was let off the hook. The Illinois Commission’s report began by summarizing Sambon’s unashamedly self-serving discussion of etiologies (in his Progress Report), which it praised as “one of the best critical reviews of previous work upon pellagra.” It identified Sambon’s own theory as “attractive and plausible in many respects,” whilst singling out its main weakness: that it “so far lacks much more evidence that simulia are the carriers than the fact that in many places simulia and pellagra are found in the same locality.” However, the report was at one with Sambon in his battle against “the maize hypothesis,” arguments in favor of which were “extremely slender.”

Not everyone was in agreement. The chief investigator for the newly founded U.S. Public Health Service, the epidemiologist Claude Lavinder, although impressed by Sambon’s hypothesis, nevertheless followed procedures originated by Italian toxicozoists. In a small laboratory at the South Carolina Hospital for the Insane, searching for what he supposed to be the streptobacillus pellagrae, following Tizzoni’s lead, Lavinder injected the blood, spinal fluid, and spleen pulp from pellagrins into rabbits, chickens, and guinea pigs. His results were negative. In 1911, Lavinder set up a larger laboratory at the Marine Hospital in Savannah, where he attempted to transmit the causative agent to monkeys. Once again he had no success. Despite this, Lavinder replaced Babcock as president of the National Association for the Study of Pellagra in 1912—a year in which

88 Armand Marie, Pellagra, trans. C. H. Lavinder and J. W. Babcock (Columbia, South Carolina: The State Co., 1910), 3–4.
89 Chicago Post, undated, quoted in “Notebook,” James Woods Babcock Papers, Library of the Medical University of South Carolina, Charleston, cit. in Etheridge, Butterfly Caste, 21.
90 “Pellagra, Insect-Borne?,” The American Agriculturalist, as reprinted in The Literary Digest, 1 October 1910, 789–90, in Etheridge, Butterfly Caste, 26.
91 Leslie, “Infectious Paradigm,” 192.
92 Douglas Singer, “Current Views upon Pellagra,” in Report of the Pellagra Commission of the State of Illinois (President Frank Billings), November 1911 (Springfield: Illinois State Journal Co., 1912), 6.
93 Report Pellagra Commission, 8.
94 Ibid., 247.
95 Etheridge, Butterfly Caste, 16–17; Bollet, “Politics and Pellagra,” 214–15.
South Carolina reported thirty thousand cases of the disease and a mortality rate of 40 percent.96

By coincidence, Lavinder had spent much of the summer of 1910 in Italy investigating pellagra, occasionally encountering another American investigator, Joseph Siler of the Army Medical Corps. But whereas Lavinder was attracted by the zeist line of enquiry (maize, spoiled or unspoiled), Siler was working with Sambon, under the aegis of the Pellagra Investigation Committee, and was an enthusiastic supporter of Sambon’s theory. Siler would lead a privately funded investigation of pellagra. The work of the Thompson–McFadden Pellagra Commission, funded by philanthropists Robert Thompson and J. H. McFadden, was undertaken by the New York Graduate School of Medicine. When the County Medical Society of Spartanburg, South Carolina, voted to co-operate, field studies were set up in the town (June 1912) with laboratory work done back in New York. The Commission proceeded to make a detailed epidemiological study of the area and its pellagrins, considering factors like housing, sanitation, diet, employment, and income.97 In their report of the first summer’s work, the Commission thought the cause might be an infection, perhaps transmitted by the bite of the stable fly (a slight variation to Sambon’s Simulium) or to the intestine by contaminated food.98

Throughout 1912–13, discussion of pellagra could not avoid reference to Sambon. His name crops up in almost every paper presented at the second meeting of the National Association for the Study of Pellagra, which was held in Columbia, South Carolina, in October 1912. J. H. Taylor, a local physician, was an early convert, one of the first to write about pellagra in the United States and to espouse Sambon’s ideas.99 Taylor’s was the glowing preface to the paper Sambon sent.100 Another delegate, Edward Jenner Wood, had just published a treatise on pellagra which had “drawn freely” from the “great work” of Sambon.101 Entomologists, like Kansas’s S. J. Hunter, were in hot pursuit of the sand-fly in their own States.102 Not everyone at the Columbia conference was so readily swayed, but Sambon was the focus of discussion nevertheless, effectively dividing opinion, even among the undecided. “Most of us,” according to F. M. Sandwith, Sambon’s colleague at the London School, “are still sitting on the fence, waiting till workers prove whether the disease is caused by the ingestion of bad maize or whether Dr. Sambon’s theory is correct.”103

Babcock warned those present at the conference that “like our European confreres [by which Babcock really means ‘Italian’] we appear to be dividing into the two camps of zeist and antizeist.”104 Babcock need not have worried about U.S. pellagrologists

96 “Dr. Joseph Goldberger and the War on Pellagra,” National Institutes of Health, Office of History, http://history.nih.gov/exhibits/goldberger/index.html.
97 Etheridge, Butterfly Caste, 49–50.
98 J. F. Siler and P. E. Garrison, Pellagra, First Progress Report of the Thompson–McFadden Pellagra Commission (New York: no publisher, 1913), 5–26.
99 J. H. Taylor, “The Protozoan Theory of Pellagra,” J S C Med. Assoc., November 1908.
100 Taylor, “Sambon the Man,” 599–605; and Transactions, 69–80.
101 Wood, Treatise on Pellagra, ix.
102 S. J. Hunter, “Pellagra and the Sand-Fly,” Transactions, 63.
103 F. M. Sandwith, “Can Pellagra Be a Disease due to Deficiency in Nutrition?,” Transactions, 97–99.
dividing into two camps; in fact, they were divided into many more, and the situation was very fluid. Of the sixteen papers that addressed the matter of pellagra’s etiology, either directly or indirectly, at the conference, seven sided with Sambon’s insect cause, or some variant of it (counting Sambon himself); seven opted for diet as the main factor; and three were undecided. The fact that those who attributed pellagra to diet-related causes were themselves divided—from an excess of cotton-seed oil to one of unspecified carbohydrates, from an intoxication caused by tainted maize to a nutritional deficiency along the lines of beriberi—suggests how much was still up in the air. The variety of viewpoints expressed at the Columbia conference is in contrast to the more limited range of opinions voiced at the Italian Pellagrological Congress held in Bergamo that year.

Meanwhile, the Thompson–McFadden Commission continued its investigations into the source of what it believed to be the pellagra “infection” during the summer of 1913, visiting the home of every mill worker in Spartanburg County. The highlight of their work that summer was Sambon’s visit, the excuse for a special conference in his honor. This was reported in an article in the New York Times bearing the headline “Prof. Louis Sambon here.” It described Sambon (mistakenly) as “the head of the Tropical School of Medicine in London and a recognized authority on pellagra,” and related his efforts at “exploding” the maize-based theory. The newspaper also reported on Sambon’s triumphant return to New York following the conference, where “it was agreed that pellagra was an infectious disease, the germ carried by an insect.”

A general interest newsmagazine, The Literary Digest, looked to Sambon and his insect theory to solve a disease now plaguing the United States. It blamed pellagra on the “hordes of Italian immigrants who have arrived in the last 30 or 40 years,” bringing their infection with them (evidently unaware of Sambon’s own origins).

Despite Sambon’s inspirational visit, the Commission ruled out the Simulium—locally, the buffalo gnat—as a carrier. Conditions were different. Whereas in northern Italy, pellagra was a disease of field workers, in Spartanburg County, it was a disease of mill workers in factory villages. It agreed with Sambon that maize was probably not a factor; however, it concluded that the real culprit seemed to be poor sanitary conditions and waste disposal, resulting in intestinal infection. Although Sambon was not aware of it, his fly-based notoriety had already peaked.

104  J. W. Babcock, “Presidential Address—How Long Has Pellagra Existed in the United States?” Transactions, 20–21.
105  “Prof. Louis Sambon Here: Comes from London to Address Southern Pellagra Conference,” The New York Times, 1 September 1913, 2.
106  “Insect Carries Pellagra: English Scientist Says Spartanburg Conference Established This,” The New York Times, 13 September 1913, 20.
107  “Another Fly to Swat,” The Literary Digest, 22 November 1913, 1003, in Leslie, “Infectious Paradigm,” 194.
108  J. F. Siler, P. E. Garrison, and W. J. MacNeal, “Second Progress Report of the Thompson–McFadden Pellagra Commission,” Arch. Intern. Med., 1914, 14, 289–93; J. F. Siler, P. E. Garrison, and W. J. MacNeal, “The Relation of Methods of Disposal of Sewage to the Spread of Pellagra,” Arch. Intern. Med., 1914, 14, 453–66.
CONCLUSION

As early as 1856, the physiologist Filippo Lussana had theorized that a diet poor in “albuminoid foods,” or protein, was the cause of endemic pellagra. Although it had substantial support among district physicians, the Lombrosian hegemony meant it was marginalized among pellagrologists for the best part of fifty years—that is, until investigators began to explore what specific nutritional deficiency resulted from a maize diet. In 1911, the Italian Aristide Stefani, a student of Lussana’s, wrote of certain “imponderables” necessary to health but which the body could not manufacture by itself and needed to take on whole. Stefani argued that whilst maize was probably adequate in proteic terms, this did not mean it could be considered a complete foodstuff, containing all the dietary “principles” necessary to man, in the way of mother’s milk or wheat. The following year, in June 1912, the Polish biochemist Kasimierz (Casimir) Funk published his findings on the disease beriberi, linked to a diet of husked rice, a process that, Funk suggested, removed a vital substance from the rice which he called a “vitamine” (from vital amine). He related beriberi to scurvy and pellagra, calling them “deficiency diseases,” even if the exact nature of the maize deficiency remained unclear. Funk’s real contribution to an understanding of the etiology of beriberi, pellagra, and scurvy were relatively limited. However, according to Carpenter, “his introduction of the term ‘vitamine’ undoubtedly drew attention to this new field of research as something of much broader importance than the prevention of one particular tropical disease.”

In the United States, this attention was immediate. The 1912 Columbia conference had seen the first reference to this new hypothesis in terms of pellagra. Rupert Blue, Surgeon General to the Health Service, identified Funk’s theory as one promising “important developments in the future.” And Sandwith, who identified himself as one of the “fence-sitters,” asked the question: “Is pellagra, too, a deficiency disease, waiting for a ‘vitamine’ to be discovered?” And he pointed to the work of Frederick Hopkins on the benefits of adding tryptophan to a maize-based diet in laboratory mice, a precursor to some “substance essential to the processes of the body.” Sandwith did not know how right he was; but it would be another two years before Funk’s hypothesis began to receive serious attention in the United States with the investigations of Joseph Goldberger. Goldberger’s were the investigations that now seemed to have “science” on their side, which is not to say that he did not meet with opposition, even hostility, at the local level. In an early paper, Goldberger made an obligatory reference to Sambon, but his sympathies already tended toward

109 Filippo Lussana and Carlo Frua, Su la pellagra: memoria (Milan: Giuseppe Bernardoni, 1856).
110 Aristide Stefani, Relazione sull’opera della Commissione pellagrologica provinciale di Padova nell’anno 1910 (Padova: Penada, 1911), 10–11.
111 Casimir Funk, “The Etiology of Deficiency Diseases,” J. State Med., 1912, 20, 341–68.
112 Carpenter, Beriberi, 100.
113 Rupert Blue, “The Problem of Pellagra in the United States,” Transactions, 1–7.
114 Sandwith, “Can Pellagra?, “99.
115 One local pellagrologist not convinced was Henry Harris, the first U.S. doctor to report a case of pellagra, in 1902, as health officer in Georgia, who remained a committed toxicozesit. H. F. Harris, Pellagra (New York: Macmillan, 1919).
diet as a chief factor. There was an urgency and practicality about Goldberger’s work that made it quite different from Sambon’s ideas-based approach, and which suited the times, with over one hundred thousand pellagra cases a year during the agricultural depression of the 1920s. As is now well known, Goldberger’s tireless work would eventually pave the way to our modern understanding of pellagra.

The mainstream medical reaction in Italy could not have differed more. Lussana, not to mention Funk, was not even referred to at the 1912 Bergamo congress. Stefani, listed as one of the representatives from the province of Padua, did not present a paper. The *Rivista pellagrologica italiana* finally allowed space for the Funk hypothesis in 1914, albeit reluctantly. This was the beginning of the end for the reign of the Lombrosian theory in Italy. 1914 also marked the end for Sambon’s hypothesis, destined to become a minor footnote in standard accounts of pellagra. And by this time, pellagra was clearly on the wane in Italy, making the etiological question less pressing—or at least less relevant. As the pellagrologist and historian Luigi Messedaglia put it: “The peasant eats better; and pellagra declines.” World War I, far from increasing pellagra, assisted in its decline, as the Italian government imported massive amounts of wheat and sold it at subsidized prices. By the 1920s, pellagra had all but disappeared, without the Italian public being much aware of it: a “silent victory.”

Despite this, the Lombrosians hung on to their hegemony until the very end. As late as 1921, the devoted Lombrosian Luigi Devoto admitted that the theory needed to be “amended,” both on a practical level, by recognizing that sound maize could make pelagrins worse, and on a theoretical level, by acknowledging that the intense and prolonged consumption of maize could cause pellagra. But what, then, remained of Lombroso’s original theory to amend? The following year, at the sixth—and, as it turned out, last—Italian Pellagrological Congress, the toxicozeist theory came in for serious criticism, to the dismay of its proponents. Gosio and Antonini were forced to admit that pellagra’s etiology was problematic, as they sought belatedly to reconcile the two positions. Their paper was followed by a series of speakers criticizing it. Antonini still had the gall to want to entitle the conference session “Reconfirming the Toxicozeist Theory,” but was forced to change his mind following an outcry.

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116 Joseph Goldberger, “The Etiology of Pellagra: The Significance of Certain Epidemiological Observations with Respect Thereto,” *Public Health Rep.*, 1914, 29(26), 1685–87.
117 Bollet, “Politics and Pellagra,” 211.
118 Etheridge, *Butterfly Caste*, 65–190, and Alan Kraut, *Goldberger’s War: The Life and Work of a Public Health Crusader* (New York: Hill and Wang, 2003), especially 97–255.
119 G. Volpino, “Il monofagismo ed i suoi rapporti con le malattie popolari della pellagra, dello scorbuto e del beri-beri,” *Rivista pellagrologica italiana*, 1914, 14(2), 17–18.
120 Luigi Messedaglia, “Mais e pellagra: un dramma di vita rurale” (1927), in idem, *La gloria del mais e altri scritti sull’alimentazione Veneta*, ed. Corrado Barberis and Ulderico Bernardi (Castabissara, Vicenza: Angelo Colla, 2008), 252.
121 Francesco Coletti, “Una vittoria nel silenzio,” *Corriere della Sera*, 11 July 1922, 1, in Messedaglia, “Mais e pellagra,” 251.
122 Luigi Devoto, “La pellagra in Lombardia e nella Venezia Tridentina dal 1900 in poi,” *Rendiconti del R. Istituto lombardo di scienze e lettere*, 1921, 54, 239–42.
123 Messedaglia, “Mais e pellagra,” 249–67.
124 Ibid., 255.
It was small comfort for the Lombrosians that maize consumption was still deemed to be the cause—even if it was now the result of a specific dietary deficiency rather than in the form of some sort of toxin. The toxicozeists could at least claim that if sound maize was lacking in some “antipellagrous” substance or principle, then spoilt maize had to be even worse.125 Two years later, in a more or less tacit admission of defeat, the Rivista pellagrologica italiana, a Lombrosian stronghold since its inception in 1901, ceased publication.

It will be evident by now that research into the causes of pellagra was a vital enterprise at the start of the twentieth century, with as much competition as cooperation, where new knowledge claims did battle with established interests. Did the “infectious paradigm” delay the defeat of pellagra, as has been argued, by getting in the way of the deficiency concept of disease?126 Or is it more useful to regard all of the investigations as part of the same research program, united as they were in a quest for causes, as Carter suggests? It is, clearly, a bit of both. On the one hand, and in support of the former, there is no doubt that, in Italy, the protection of legitimacy and reputation lead to the production of scientific doubt as well as scientific knowledge, similar to what Bay has suggested for beriberi in Japan.127 Neither Sambon nor Antonini had any time for the notion that pellagra might be due to a dietary deficiency. Sambon’s brief foray into Italian pellagrology demonstrated just how entrenched the Lombrosian hegemony was (as represented by Antonini). Sambon may have forced Italian pellagrologists to question their most firmly held and cherished beliefs, but the toxicozeists were strong enough to marginalize not only Sambon but all other alternative etiologies at the highest levels of medicine and government.

On the other hand, when it came to U.S. investigations into pellagra’s etiology, there was no prevailing legitimacy to protect. The influence of the infectious paradigm certainly contributed to Sambon’s short-lived notoriety there; but a look at the conferences on pellagra shows just how much open-ended debate and argument was going on throughout this period. This is much more redolent of Carter’s “single coherent research programme” (which would pave the way for Goldberger’s successful investigations). Evidence for this view can be found in Italy, too, where research in other areas did go on, sometimes behind the scenes, sometimes quite openly. District doctors were less swayed by the Lombrosian hegemony; they remained divided, or undecided, or even combined opposing theories into an original synthesis. Moreover, as we have suggested, Sambon and toxicozeists like Antonini had in common a belief that the cause of pellagra was bacterial; what they differed on was the means of transmission, whether the microorganism was conveyed via an insect or through spoiled maize consumption. As Michael Warboys reminds us, the germ theory encompassed a broad spectrum of factors, ranging “from simple chemical poisons through to worms.”128 Indeed, the Sambon–Antonini dispute

125 Alessandro Lustig and Augusto Franchetti, Studi ed osservazioni sulla pellagra: relazione (Florence: Ariani, 1921).
126 Leslie, “Infectious Paradigm,” 201.
127 Bay, Beriberi, 8.
128 Michael Warboys, Spreading Germs: Disease Theories and Medical Practice in Britain, 1865–1900 (Cambridge: Cambridge University Press, 2000), 2.
was so fierce because it was a clash between estranged cousins, as it were, although they themselves might not have recognized the relationship. Sambon and Antonini shared something else, too: their concern with a bacterial explanation effectively left society off the hook. There was no need to reform the underlying social conditions that led to a reliance on maize, if the cause was bacterial. In Italy, the gradual disappearance of pellagra took the sting out of the dispute, and may also explain why Italian investigators were not in the forefront after World War I, whereas in the United States, the worsening of the epidemic made the hunt for a cause all the more pressing.

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