On 19 December 1910, Bergamo’s main newspaper, L’Eco di Bergamo, reported on a new theory claiming to explain the causes of pellagra. The disease was then epidemic in much of northern Italy, with the Bergamo province being one of the worst affected, bringing dermatitis, dizziness, diarrhea, extreme weakness, insanity and death in its wake. The news of a new theory 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 farmers and producers 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 author 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 Sambon had written on the subject, translating the Report’s conclusions.

Sambon was scathing towards Italy’s 1902 anti-pellagra law, based as it was on what he considered an erroneous etiology of the disease. According to L’Eco di Bergamo’s unnamed author, “the Italian government drew up, debated and passed a law based on a theory, already opposed and denied at the time, pushed for and forced through by Lombroso and his school”.

The reference was to Cesare Lombroso, who first developed the theory that pellagra was due to the eating of spoiled maize; a theory which the 1902 law made official. The law was in tatters now, the newspaper argued; “however, there remain the heavy and numerous fines unfairly enforced against millers and farmers; there remains the harm to industry, flourishing even here in Bergamo, to trade and to agriculture itself”.

Although signed by a “medico condotto”, the article’s author had a pronounced free-trade axe to grind. Protecting Bergamo’s pre-eminence as a milling centre, supplying five-eighths of Italy’s maize flour, seems to be the author’s main concern. There was no attempt to evaluate the new theory from a medical point of view. That came a few weeks later; but the reaction could not have been more different. 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. Antonini quoted extensively from the article’s translation of the Progress Report, even as he proceeded to rubbish its conclusions, point by point.

Antonini objected most of all to Sambon’s confident assertion that Lombroso’s spoiled maize theory was wrong, that all the 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 on reading this, before pointing out how the numbers 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 1900-02.

As one of Italy’s leading pellagrologists, Antonini did not take kindly to Sambon’s claim that the spoilt 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, arguing that who better to diagnose pellagra than 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 sleeping sickness, Antonini admitted; but his view of pellagra-like disease had been shaped too much by his observance of it 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.

Across the Atlantic Ocean, in the United States, the reaction 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”. 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 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. None the less, 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
collection 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.

There are several reasons why studying how one “wrong” theory of pellagra sought to
displace another equally wrong theory is revealing. First of all, whatever Sambon had to say to
the contrary, 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 was identified (in 1924). From the
time of the earliest Italian explorations of pellagra in the mid eighteenth century, dating from a
time 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 is what these shifts and the resulting controversies reveal
about the medical context. Secondly, 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 years. Italian pellagrologists had to question their most firmly held and
cherished beliefs. This was the decade (1900-10) that saw more pellagra studies published in
Italy than any other.8 Thirdly, because reactions to Sambon’s provocative theory were very
different in the U.S., where pellagra had just appeared, we need to account for the differences
in reaction in Italy and the U.S., in terms of research agendas and medical power structures. This allows us to explore the reality of endemic pellagra, and the medical response to it, in both countries.

*Pellagra in Italy and the Toxicozeist Hegemony*

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 a third to one half of insane asylum patients in affected areas were victims of pellagra.9 Those who survived suffered regular outbreaks of the disease each spring, the symptoms getting worse each 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”.10 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 theory” 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 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 town’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 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 his laboratory analyses, toxic substances formed within poorly dried or unripened maize, Lombroso argued, and this “diseased” or spoiled maize, when ingested, led to the symptoms of pellagra.

The spoilt maize or toxicozeist 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. The search was on for the responsible micro-organism in spoiled maize. The toxicozeist 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 law of 21 July 1902 enabled a more systematic response, focused around the Lombroso-inspired battle “alle muffe e alle truffe” (against molds and frauds). It consisted of nineteen articles, targeting unripe or spoiled maize and the products made from 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. It also gave new impetus to preventive and therapeutic initiatives, and led to the setting up of new 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. Such measures were considered too radical. Nor did it fund all the proposed initiatives, whose costs had to be met by local authorities.

The steady decline in pellagra numbers in the wake of the 1902 law appeared to vindicate the Lombrosian theory.16 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.17) It 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”.18 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”.19 If the 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 ready made, and not in the form of a bacterium”.20

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 actually prosecuted— 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. But even if it became
enshrined in law, it would “be necessary to remain vigilant and attentive, and that action
resulting from the law be full and complete, and that its enforcement is not frustrated by
crippling compromises”. 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. Antonini 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 cured it
was explained.24

As Antonini told the delegates to 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”.25
Antonini countered each of the new theories in turn, which if taken together cancelled one
another out.26 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
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 “[eliminate] 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.27 From the
organization of his defence 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,
exaggerating 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 bacteriological theory was being challenged by the deficiency theory, and meeting with the same sort of establishment opposition as in Italy. 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 deficiency theory. The reference here is to the tobacco industry’s deliberate and longstanding policy of scientific misinformation regarding the links of cigarette smoking to cancer, as described by Robert Proctor. Antonini was not an ally of maize-producing interests in Italy; indeed the 1902 anti-pellagra law had put curbs on the production and sale of maize. That said, he did have his own professional interests to protect. Self-righteous wrongheadedness, most probably; but deliberate obfuscation, no.

*Louis Sambon: a Man and his Theory*

If the leading Italian pellagrologists were a self-righteous lot in 1910, so too was Louis Westenra Sambon. Sambon had grown up in Italy, mostly in Milan, the son of a Franco-Italian antiquarian father and an English mother. Sambon sidestepped family tradition in order to study medicine, choosing the University of Naples, whose medical faculty was then Italy’s largest and one of its most prestigious, particularly in fields like pathology and medical hygiene. “Luigi Sambon di Giulio, Milano” matriculated in 1884, the same year a cholera epidemic struck
the city, and the young medical student was called upon to provide assistance.\textsuperscript{31} The experience may have sparked Sambon’s interest in epidemiology. After receiving his MD in 1891, Sambon served briefly as an assistant surgeon in the Italian army in Africa long enough to write a study of the Ethiopian army.\textsuperscript{32} He then returned to live in Rome, where he practiced gynecology, studied natural history and collected and wrote on medical antiquities.\textsuperscript{33} Still in Rome, Sambon contributed to the “white men in the Tropics” debate, writing an article for the \textit{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 was microbial in nature, including not just malaria and tuberculosis, but also sunstroke, which he called “siriasis”.\textsuperscript{34}

Sambon attracted 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.\textsuperscript{35} 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 Sambon went to England, “against the strongly expressed wishes of his father”.\textsuperscript{36} London was then the medical centre of the British empire, where hitherto unfamiliar diseases were encountered and investigated, and knowledge about them generated.\textsuperscript{37}

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 autumn of 1900, Manson sent Sambon, together with George Low, lecturer at the School, and the artist Amadeo Terzi, who had done many of the illustrations for Sambon’s Ethiopia book, 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 “Roman school” of Grassi and Celli were working on similar lines.

The first point of the British expedition, of which Sambon regarded himself as leader, was to demonstrate how to prevent mosquito transmission of the *Plasmodium vivax* strain of malaria. 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 intermediating amicably between British and Italian colleagues, Sambon sided with Ross in the priority dispute against the Italian zoologist Giovanni Battista Grassi and the “Rome school” over the discovery of the role of mosquitoes in the transmission of malaria. This included Grassi’s claims about prevention, based on a trial conducted in 1899, which had involved “mechanical prophylaxis” (metal window and door screens). Reviewing the history of filarial [parasite] disease research, Sambon argued that Grassi and his assistant Noè claimed far more priority for themselves than
was warranted by the facts.\textsuperscript{42} Ross, not surprisingly, replied with a letter supporting Sambon’s take on things.\textsuperscript{43} At this point Sambon was translating Grassi’s letters and articles for Manson, who specifically requested him to include the “lies and insinuations”.\textsuperscript{44}

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).\textsuperscript{45} 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 over the next fifteen years. By 1915, the number of deaths had declined from 600 per million inhabitants to less than 50.\textsuperscript{46} 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.

In 1902 Manson asked Sambon if he would like to accompany Low and Cuthbert Christy to Equatorial Africa and Uganda on a Royal Society expedition to study an epidemic of sleeping sickness. Sambon declined for family reasons; in his place, he recommended a young doctoral student at the London School, Aldo Castellani. The trip was the beginning of Castellani’s career and fame, but in his autobiography of many years later he could not bring himself to name Sambon.\textsuperscript{47} Perhaps Castellani was still resentful that Sambon, from his London study, claimed a role in the discovery of the etiology of sleeping sickness, while Castellani was risking his life in the field. Sambon collated the available literature on the disease, studied its topographical
distribution and possible determinants, and related these to known parasites.48 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 73rd meeting of the British Medical Association, held in Leicester that year, Sambon voiced his hypothesis on the etiology and spread of pellagra, employing his now standard methodology, using a detailed survey of pellagra’s spread to identify a possible vector or agent.49 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”.50 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”.51 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 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”, a gifted theorist and investigator. 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”.52 Manson was even more impressed. In a letter to Ross, Manson characterized Sambon as “a magnificent worker, with ideas ahead of the time”; indeed this was why “so many affect to sneer him”.53 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”.54 Sambon could also count on the support of the successful medical entrepreneur and collector Henry Wellcome.55 By this time Sambon had identified a single insect for investigation, the Simulium reptans or sandfly, a small blood-feeding fly. Sambon’s proposed visit to Italy was intended to determine whether a sandfly was the carrier of pellagra and whether pellagra could be defined as belonging to the group of protozoal diseases.56
Sambon departed for Italy on 20 March 1910, even though the committee had thus far raised only a fifth of the target amount. Sambon’s twelve-week fact-finding trip took him throughout the areas of Italy worst affected by pellagra, from Bergamo in the north to Perugia in the centre. Sambon “made it a point of getting in touch with those who had devoted special attention to the study of pellagra”, as he would later write in his *Progress Report*, meeting medical experts, provincial and municipal health officers, district doctors, pellagra commission members and inspectors, hospital and health station directors, and mayors, wherever he stayed.

Sambon’s *Progress Report* resembles his 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. He 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 dealing with pellagra but also noting how their frontline 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”.63 It explained the unchanging boundaries of pellagra’s “endemic centres”, 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 of cases following flooding, the seasonal eruption of the disease (spring and autumn, when sand-flies were active), 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”.64 If the protozoan responsible for the disease has not yet been identified, the role of the Simulium in the transmission of pellagra was “almost a certainty”.65

The Italian Reaction

Sambon may have thought his Pellagra Report “to be a model of its kind”, as he wrote in a letter to Wellcome, but not all of his British colleagues agreed.66 Ross, admittedly never easy to please, 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 that “produces the worst possible impression. It is not science”.67 Sambon had written to the Italian press in similar fashion, beginning with newspapers like L’Eco di Bergamo. The result, according to 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 amongst 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 a 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 is anything to go by. “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 was still up in the air did not necessarily bother contemporaries, at least not the toxicozeists. After all, Robert Koch had discovered the tuberculosis 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). What Sambon did was to bring the issue of etiology back into the pellagrological debate, whatever Antonini may have had to say about it. Other pellagrologists, without necessarily agreeing with Sambon, welcomed his ideas as a breath of fresh air. Thus 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.
It 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. The Committee was chaired by the professor of pathology (and, from 1911, senator) Alessandro Lustig. 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. He was uncharacteristically undogmatic, conceding that an insect other than the Simulium might be responsible. This was Sambon’s second research trip to Italy following the publication of his 1910 Progress Report and he particularly wanted to investigate pellagra in children. Sambon concluded by hoping the Commission would collaborate in his research to establish the role of the Simulium in pellagra infection.

Chairman Lustig then thanked Sambon and told how him pleased he was that Sambon was carrying out further research in Italy and wished to impress on his the unbiased nature of the Commission, which represented all “currents” (tendenze) of thought on the subject, united in their aim to clarify the etiology of pellagra. Commission members asked Sambon about pellagra numbers in children, the distribution of pellagra and Simulium in Italy, on any autopsies performed on pellagrins, and other matters. After Sambon had left, Lustig asked members to undertake a detailed examination of his research program 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. 77 For the Umbria sub-commission, the bacteriologist Gosio reported on its investigations into pellagra amongst 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”. 78 Whilst the Lombardy-Veneto sub-commission reported on recent bacteriological studies of tainted maize by one of its members (Tizzoni), without making any mention whatever of Sambon. The Commission’s conclusion? “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 at 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 had hitherto the support and justification of practice”. 79 And that is exactly what happened.

Although Sambon himself could not attend,80 seven out of eighteen papers in the “medical section” (“parte medica”) of the meeting were specifically devoted to the “new etiologies”. This was in itself significant: Sambon had put etiology back on the table. That said, none of the papers which mentioned it found any support for Sambon’s theory. 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 none 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 survey did testify to the variety of opinion present amongst district physicians, as well as the ongoing—indeed perhaps rising—support for the Lussana theory. This indecision, or perhaps openness, was in stark contrast with the Lombrosian orthodoxy prevalent amongst full-time pellagrologists. But this was never going to worry the Italian medical elite, given the relatively low standing of the poorly paid and 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 Tizzoni, who claimed to have isolated a streptococcal bacillus in pellagrins and, in animals, was able to duplicate the skin lesions typical of pellagra. It is another indication of how the Lombrosian, toxicozetist orthodoxy was still able to dominate proceedings and claim the high moral 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 toxicozetist theory should be reaffirmed as the “correct basis” of the present law and as effective in the prevention of pellagra. 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 a physical monument to Lombroso in Verona.
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”. 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. 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. In fact, this was more or less the case, but not for want of trying on Sambon’s part. Wellcome agreed to finance another expedition to Italy, involving Sambon and Albert Chalmers, but the trip did not go well.

**Reaction to Sambon in the United States**

In 1913 Sambon’s wife, Hilda, wrote to Charles Thompson about her husband’s reception in the US:

You will see by these cuttings what a reputation my husband has in America, & I believe, that for a scientist, such an enthusiastic reception is unprecedented. He tells me that after his address in Spartanburg [South Carolina], old men kissed him and the young ones swarmed around him like bees. Also, all those whose invitations he was unable to accept sent him profusions of flowers.

The newspaper clippings Hilda Sambon enclosed with her letter have not survived. However an article from the *New York Times* bearing the headline “Prof. Louis Sambon here”,


referring to him as “the head of the Tropical School of Medicine in London and a recognized
authority on pellagra”, and describing his efforts at “exploding” the maize-based theory, gives
the idea. The newspaper also reported on Sambon’s triumphal 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 U.S. 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).

Pellagra seemed to appear from nothing in the U.S. 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? Because pellagra was new there was little of the zeist cultural baggage
present in Italy. It struck at a time when a whole 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 U.S. 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 travelled 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 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 in 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 Agriculturist* 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 (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 lack 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 toxicozeists. In a small laboratory at the South Carolina Hospital for the Insane, searching for what he supposed to be
the *streptobacillus pellagrace*, 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 South Carolina reported 30,000 cases of the disease and a
mortality rate of 40 per cent.

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, as announced in the *New York Times* article quoted at the start
of this essay. 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. 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.
Throughout 1912 medical 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 of that year. J. H. Taylor, which prefaced Sambon’s remarks. J. H. Taylor, a local physician, was an early convert, one of the first to write about pellagra in the U.S. and to espouse Sambon’s ideas. Another delegate, Edward Jenner Wood, had just published a treatise on pellagra which had “drawn freely” from the “great work” of Sambon. Entomologists, like Kansas’s S. J. Hunter, were in hot pursuit of the sand fly in their own States. Not everyone at the Columbia conference was so readily swayed, but Sambon was the focus of discussion nevertheless, effectively dividing opinion, even amongst 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”.

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”. Babcock need not have worried about U.S. pellagrologists 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, as reported by Hilda Sambon in
her proud letter to Thompson. Despite Sambon’s inspirational visit, the commission ruled out
the Simulium (locally, the buffalo gnat) as a carrier. Local conditions were different from those
in Italy. 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.117

Being at the centre of scientific debate, along with the fame and adulation, were
starting to go to Sambon’s head. In the summer of 1914 he wrote to Thompson, complaining of
the lack of support and scholarly freedom.

I need liberty, full liberty to carry out the great work I feel capable of
accomplishing, and I am ready to sacrifice all for the love of science, as I
have done again and again. In the past I was alone, now my scientific
friends both in the Old and New Worlds have promised me every kind of
help and support.118
Although Sambon was not aware of it, his gnat-based notoriety had already peaked.

*Epilogue*

As early as 1856 the physiologist 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” (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 Kenneth 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 U.S., 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 U.S. Public 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 towards diet as a chief factor.

The situation 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 of the Lombrosian hegemony in Italy. 1914 also marked the end of 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 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” 129

Despite this, the Lombrosians hung on to their hegemony till the end. As late as 1921 the devoted Lombrosian Luigi Devoto admitted that the theory needed to be “amended”, both on practical level, by recognizing that sound maize could make pellagrins worse, and on a theoretical level, by acknowledging that the intense and prolonged consumption of maize could cause pellagra.130 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.131 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.132 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 this caused 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.133 Two years later, in more or less in a tacit admission of defeat, the Rivista pellagrologica italiana, a Lombrosian stronghold since its inception in 1901, ceased publication.

Did the infectious paradigm delay the defeat of pellagra, as has been argued?134 Did it effectively side-track research on pellagra and diet for many years, in the interests of public
officials, medical practitioners 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.  

135 Codell Carter has dismissed this contra-factual claim, by stressing “the variety of causes contemporary researchers were willing to entertain”. Rather, in his view, the deficiency concept and the bacterial theory belonged to the same “scientific research programme”, with its general focus on etiology.  

136 This is certainly evident when we survey the range of work then being done at an international level, 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. Here, the protection of legitimacy and reputation lead to the production of scientific doubt as well as scientific knowledge, as Bay has suggested for Japan.  

When it comes to U.S. investigations into pellagra’s etiology, there was no legitimacy to protect. The influence of the infectious paradigm contributed to Sambon’s short-lived notoriety there. However, a look at the conferences on pellagra shows just how much open-ended debate and argument was going on at the time. In Italy, by contrast, a Lombrosian hegemony in Italy still reigned, strong enough to marginalize Sambon and other alternative etiologies at the highest levels. That said, research in other areas went on, sometimes behind the scenes, sometimes quite openly. District doctors remained divided, or undecided, or even combined opposing theories into an original synthesis. Moreover, 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 micro-organism was conveyed via an insect or through maize consumption. Whilst the Lombrosians shared with the Lussanians a conviction that maize
was at the heart of the problem. 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 underlying social conditions if the cause was bacterial. In Italy, the gradual disappearance of pellagra took the sting out of the dispute, and my also explain why Italian investigators were not in the forefront after WWI, whereas in the U.S. the worsening of the epidemic made the hunt for a cause all the more pressing.

1 Acknowledgments removed.

2 Anon. [Un medico condotto], “La riabilitazione della polenta. La legge sulla pellagra ed i mugnai”, L’Eco di Bergamo, 19-20 December 1910, no. 287, 3. All translations are my own unless indicated otherwise.


10 Babcock and Cutting, *Pellagra*, p. 15.


17 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 the the “wrong” theories. Luigi Faccini, “Tifo, pensiero medico e infrastrutture igieniche nell’Italia liberale”, Franco Della Peruta, ed. *Storia d’Italia, Annali 7, Malattia e medicina* (Turin: Einaudi, 1984), 716.


19 Ibid., 19.

20 Ibid., 20.

21 Cesare Salvetat, “Commissione pellagrologica comunale di Asola (Mantova)”, *Rivista pellagrologica italiana*, vii, no. 3 (May 1907), 154-9.


Alongside Sambon’s, the new aetiological “doctrines” discussed by Antonini and Gosio were Guido Tizzoni’s (which was not exactly new), positing a pellagra germ, the *streptobacillus pellagrace*, 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 affects of the sun on people with a maize diet.


Ernesto Capanna, “Grassi versus Ross: Who Solved the Riddle of Malaria?”,

Discussed in Snowden, *Conquest of Malaria*, pp. 44-5, who however does not mention the British “expedition” of the following year.

L. W. Sambon, “Remarks on the Life History of Filaria bancrofti and Filaria immitis”, 


Sambon, “Remarks”, 1275.

James Cantlie, “The Importance of Rational Inductive Methods in Advancing Knowledge”, editorial, J Trop Med Hyg, 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 of Med, 83 (2009), 530-60.

Letter of 5 November 1909 from Manson to Ross, in Bynum and Overy, Beast in the Mosquito, p. 473.


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).

“The Nature and Causation of Pellagra”, The Times (London), 4 February 1910, 4. In 1903 William Leishman and Charles Donovan had both identified a sandfly as the possible carrier of the parasite responsible for the deadly disease kala-azar (visceral leishmaniasis).
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/).


Ibid., 282, 290.

Ibid., 290.


Ibid., 297.

Ibid., 321.

Ibid., 271.


68 M. V. Carletti, “Etiologia della pellagra. Le critiche di L. W. Sambon alle teorie maidiche”, Gazzetta degli ospedali e delle cliniche, no. 64 (1911), 5.


71 Carletti, “Etiologia della pellagra”, 5.

72 Gustavo Pisenti, L’ortodossia pellagrosa e il semplicismo delle scuole maidiche (Perugia: Tip. Perugina, 1912).

73 As reported in the Rivista pellagrologica italiana, vol. 12 (1912), no. 3, 144-5.

74 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.).

75 Telegram of 17 February 1912 to Thompson, WA/HMM/CO/Ear/844.


77 “Commissione ministeriale per lo studio della pellagra”, Rivista pellagrologica italiana, vol. 12 (1912), no. 4, 162.

78 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.
43


80 Sambon pleaded previous commitments and “a most painful neuritis”. Cantarutti, *Atti del quinto congresso pellagrologico*, 179.


82 Guido Tizzoni, untitled presentation, *Atti quinto congresso*, pp. 204-18. Tizzoni was professor of general pathology at the University of Bologna and specialist in the study of infections and immunology, famous for pioneering the anti-tetanus sierum in 1889-90. His 1913 work, *La pellagra studiata nelle sue cause* (Turin: UTET), was all about the (phantomatic) *streptobacillus pellagrace*.


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


87 Ibid., p. 19.

88 Letters of 15 April and 27 May 1912, WA/HMM/CO/Ear/844.

89 Letter of 19 September 1913, WA/HMM/CO/Ear/845. Thompson was a collecting agent for Wellcome and responsible for employing Sambon. Frances Larson, *An Infinity of*
“Prof. Louis Sambon Here: Comes from London to Address Southern Pellagra Conference”, *The New York Times*, 1 September 1913, 2.


Leslie, “Infectious Paradigm”, 190.


The expression is Leslie’s, “Infectious Paradigm”.


103 Leslie, “Infectious Paradigm”, 192.


106 Ibid., 247.


115 F. M. Sandwith, “Can Pellagra be a Disease Due to Deficiency in Nutrition?”, Transactions, 97-9.


118 Letter of 27 June 1914, WA/HMM/CO/Ear/845.

119 Filippo Lussana and Carlo Frua, Su la pellagra: memoria (Milan: Giuseppe Bernardoni, 1856).

120 Aristide Stefani, Relazione sull’opera della Commissione pellagrologica provinciale di Padova nell’anno 1910 (Padova: Penada, 1911), 10-11.

121 Casimir Funk, “The Etiology of Deficiency Diseases”, Journal of State Medicine, vol. 20 (1912), 341-68.

122 Kenneth Carpenter, Beriberi, White Rice, and Vitamin B: A Disease, a Cause, and a Cure (Berkeley: University of California Press, 2000), 100.


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).


Luigi Devoto, “La pellagra in Lombardia e nella Venezia Tridentina dal 1900 in poi”, *Rendiconti del R. Istituto lombardo di scienze e lettere*, vol. 54 (1921), 239-42.


Ibid., 255.

134 Leslie, “Infectious Paradigm”, 201.

