FROM MEDICAL GEOGRAPHY TO GERM THEORY IN COLOMBIA, 1860-1900

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ABSTRACT

Before the consolidation of the germ theory of human diseases at the end of the nineteenth century, medical explanations about disease causation were dominated by the environmental notions of medical geography. This dissertation explores how nineteenth-century Colombian physicians transformed the medical geographical approach using the early concepts and technologies of the emerging Pasteurian germ theory. I follow this transformation in the cases of periodic fevers (yellow fever and malaria), continuous fevers (typhoid fever and typhus) and leprosy. The analysis reveals that by mid century physicians had incorporated neo-Hippocratic versions of disease causation and French medical geographical ideas in order to make sense of disease of the warm, temperate and cold lands. Their conceptual network revolved around the specific, predisposing and occasional causes in which climate and geography played a determinant role. Evidence indicates that this was the case of periodic fevers of the warm lands (yellow fever and malaria). I argue that the "parasitic" hypothesis of yellow fever was accepted during the controversy around the prophylactic inoculations inspired by Pasteurism that were applied in Colombia in 1887. However, doctors struggled to reconcile the medical geographical and the bacteriological perspective of both yellow fever and malaria. Continuous fevers, on the other hand, were also framed within the medical geography scheme of disease causation. I show how during the debates about typhoid fever and typhus happening in the Colombian highlands during the 70s, 80s and 90s, doctors used medical geographical notions and developed anti-pasteurian arguments, while the international scientific community had identified the specific bacilli for typhoid fever. Finally, I argue that the strong interest of Colombian doctors on leprosy –also understood in neo-Hippocratic terms- that foster the search for local treatments based on Pasteurism (antiseptics in the 1880s and serotherapy in the 1890s) also prompted the extension of the bacteriological model and techniques to other diseases in those decades.

CONTENTS

MAP OF COLOMBIA (AND PART OF VENEZUELA) WITH THE MAIN LOCATIONS AND REGIONS MENTIONED IN THIS WORK

ABREVIATIONS

ANM	*Academia Nacional de Medicina* (National Academy of Medicine)
JCH	*Junta Central de Higiene* (the Central Hygiene Bureau)
SMCN	*Sociedad de Medicina y Ciencias Naturales* (the Medical and Natural Sciences Society of Bogotá).

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INTRODUCTION

Before the consolidation of the germ theory of human diseases at the end of the nineteenth century, medical explanations of disease causation were dominated by neo-Hippocratic and climatic notions. In Colombia these notions took the form of medical geography, that is, the study of the climatic and geographical conditions that were causally associated with the development and distribution of diseases. Colombian physicians first discussed germ theory during the anthrax epizootic of 1869, but it would not be until the late 1880s that physicians would fully incorporate Pasteurian notions into their medical understanding of human diseases. Thus, the purpose of this dissertation is to explore how and why nineteenth-century Colombian physicians transformed the medical geographical causal approach to human diseases by the use of concepts and practices related to the Pasteurian germ theory of diseases.

I chose to follow this transformation in the cases of periodic fevers (yellow fever and malaria), continuous fevers (typhoid fever and typhus) and leprosy. Because of the focus on these diseases, this dissertation can also be seen as a history of the transformation of the identity of fevers and leprosy in the early years of Pasteurism in a particular locale, Colombia.

In this introduction I will discuss the way that scholars have set up the historical relationship between medical geography – or pre-bacteriological notions of disease causation - and the germ theory, and the way that they have presented that process in the Colombian case. I will then explain the problems of this investigation and the theoretical approach, and finally I will refer to the sources and the content of this dissertation.

The historiography of the relationship between medical geography and bacteriology

The germ theory of disease has appealed more to historians of medicine and science than medical geography. This is probably because to physicians, the public, and historians of bacteriology, germ theory represents a watershed between traditional

and modern science, an irreversible turning point in the history of disease concepts.[1] As the history of medical geography has faded under the power of the uncontested product of the bacteriological revolution, histories of the negotiations about the meanings of disease during the transition from medical geography to germ theory have become almost non-existent.

Scholarship on germ theory seldom refers to medical geography.[2] Moreover, there is no consensus on how to frame the pre-bacteriological definitions of contagious disease. For example, in the chapter on "Contagion, Germ Theory/Specificity" published in the *Companion Encyclopedia of the History of Medicine* (1997), Margaret Pelling contrasts the nineteenth-century use of the "traditional multifactorial structure of natural causation" of classical philosophy in explanations of disease (the primary, the immediate or environmental, the predisposing, and the proximate causes) with the reductionism of early germ theory (which tended to ignore all but the second of these causes).[3] Both William Bulloch (*History of Bacteriology*, 1938 [1960]), one of the early historians of bacteriology, and John Andrew Mendelsohn (*Cultures of Bacteriology*, 1996), among the most recent examples, use an even narrower framework to refer to pre-bacteriological notions of communicable diseases: the contagion or miasma theories.[4]

Scholarship on medical geography, on the other hand, either only covers the period before the establishment of the germ theory, like Caroline Hannaway's chapter "Environment and Miasmata" in the *Companion Encyclopedia of the History of Medicine* (1997),[5] or highlights the continuity of that medical discipline to the

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[1] See for example William Bulloch, *The History of Bacteriology* (London, Oxford University Press, 1960 [1938]); W.A. Foster, *A History of Medical Bacteriology and Immunology* (London: William Heinemann Medical Books, London, 1970); Andrew Cunningham, "Transforming Plague. The Laboratory and the Identity of Infectious Disease" in Andrew Cunningham and Perry Williams (eds.), *The Laboratory Revolution in Medicine* (Cambridge: Cambridge University Press, 1992), pp. 209-244.

[2] See Bulloch, *The History of Bacteriology*; Foster, *A History of Medical Bacteriology*; Margaret Pelling, "Contagion, Germ Theory/Specificity", W.F. Bynum and Roy Porter, *Companion Encyclopedia of the History of Medicine*, 1 (London/New York: Routledge, 1997), pp. 309-334; John Andrew Mendelsohn, *Cultures of Bacteriology: Formation and Transformation of a Science in France and Germany, 1870-1914*, PhD Dissertation, Princeton University, June, 1996.

[3] Pelling, "Contagion, Germ Theory/Specificity", p. 312.

[4] Bulloch, *The History of Bacteriology*, p. 165; Mendelsohn, *Cultures of Bacteriology*, pp. 90, 5.

[5] Caroline Hannaway, "Environment and Miasmata", in W.F. Bynum and Roy Porter (ed.), *Companion Encyclopedia of the History of Medicine* (London and New York: Routledge, 1993), pp. 293-308.

twentieth century without detailing its transformation by germ theory.[6] Although little attention is paid to the changes and conflicts generated in the practice of medical-geographical-oriented doctors by the arrival of germ theory, it is recognized that the theory was responsible for the decline of medical geography around the turn of the twentieth century. There are only a few works that analyze this process in particular contexts, as I will show later, and they barely take into account the negotiations surrounding the germ theory's introduction of new meanings and concepts of disease.[7]

Thus, there are more works on the history of germ theory and on the history of medical geography than on their common history. In the following section, I will discuss some of the more significant books and articles in these two fields, their theoretical and methodological approach, and their main conclusions regarding the meaning of germs and the theories of disease. I will then look into the few works which specifically address the problem of the transition from medical geography to germ theory in general and in Colombia.

Trends in the scholarship of the history of the germ theory

In the introduction to the special issue on the reception of the germ theory of disease published by the *Journal of the History of Science* in 1997, Nancy J. Tomes and John Harley Warner clearly establish a watershed between those who treat "the germ theory of disease" as "if it were a real entity, an historical actor with agency of its own", and those who treat it as a construction, the product of many communities engaged in vigorous debates.[8] The first group is comprised of those who have created an internalist history of germ theory, that is, those who use the epistemic authority of scientific knowledge as the only explanation of the emergence and spread of

[6] Conevery Bolton Valencius, "Histories of Medical Geography" in Nicolaas A. Rupke (ed.), "Medical Geography in Historical Perspective", *Medical History*, Supplement No. 20 (London: The Wellcome Trust Centre For the History of Medicine at UCL, 2000), pp. 3-28.
[7] See Michael A. Osborne, "The Geographical Imperative in Nineteenth Century French Medicine" in Nicolaas A. Rupke, "Medical Geography in Historical Perspective", *Medical History*, Supplement No. 20 (London: The Wellcome Trust Centre For the History of Medicine at UCL, 2000), pp. 31-50; and Marcos Cueto, "Nationalism, Carrión's Disease and Medical Geography in the Peruvian Andes", *History and Philosophy of the Life Sciences*, 25 (2003): 319-335.
[8] Tomes and Warner, "Introduction to Special Issue" on Rethinking the Reception of Germ Theory of Disease: Comparative Perspectives", *Journal of the History of Medicine*, 52, January (1997), pp. 7-16.

scientific theories. This would be the case with William Bulloch's book *The History of Bacteriology* (1938), and W.D. Foster's *A History of Medical Bacteriology and Immunology* (1970). Bulloch and Foster, both bacteriologists committed to the documentation of the development of their discipline, account for the main achievements in acceptable knowledge within their field; hence, their histories focus on early believers in germ theory struggling against opposite points of view until the theory imposed itself by its rational force (Bulloch), and the degree of resemblance between modern knowledge of parasites and their description in the past (Foster).[9] A similar approach can be found in a more recent book, K. Codell Carter's *The Rise of the Causal Concepts of Disease* (2005). Convinced that the search for universal necessary causes was the most important medical project of the nineteenth century, Carter offers an historical account of the etiological standpoint established by Koch and Pasteur that draws on the ideas about scientific research programmes developed by the philosopher of science, Imre Lakatos.[10] Carter says that Koch and Pasteur were at the center of a "progressive scientific programme" which acquired its progressive character from the adoption of the theoretical and empirical perspective of the etiological standpoint. For Carter, the historical driving force was theory. In terms of Tomes's and Warner's argument, Bulloch, Foster and Carter treat germ theory as if it had some ontological life of its own.

During the 1980s and 1990s, according to Tomes and Warner, historians have taken up the task of rewriting these kinds of mythic accounts of the germ theory. In contrast to Bulloch's and Foster's narratives, authors within this revisionist project consider germ theory to have had "many different meanings assigned to it in debates over specific diseases, at different points in time, across national boundaries, and among different classes and ethnic groups".[11] Works such as Bruno Latour's *The Pasteurization of France* (1988),[12] Margaret Pelling's paper, John Andrew Mendelsohn's thesis about bacteriology in France and Germany, and Michael

[9] Foster, *The History of Medical Bacteriology*, p. 1.
[10] That etiological standpoint would be that "diseases are best controlled and understood by means of causes and, in particular, by causes that are *natural* (as opposed to the wilful transgression of moral or social norms), *universal* (the same cause is common to every instance of a given disease), and *necessary* (disease does not occur in the absence of its cause)". See K. Codell Carter, *The Rise of Causal Concepts of Disease* (Aldershot: Ashgate, 2003), p. 1 and p. 2-3.
[11] Tomes and Harley, "Introduction to Special Issue", p. 12
[12] Bruno Latour, *The Pasteurization of France* (Cambridge/Massachusetts/London: Harvard University Press, 1988).

Worboys's book, *Spreading Germs* (2000), on the case of Britain,[13] can be seen as part of this new trend. Although all of them consider germ theory to be a construction, their theoretical and methodological approach is diverse.

In *The Pasteurization of France* (1988), Bruno Latour chooses the case of the Pasteurian revolution to illustrate his Actor-Network Theory. Using published journals and semiotics as his main research strategy, Latour depicts the multiple translations that Pasteur and Pasteurians made in order to build the long networks that would make their truth about microbes indisputable.[14] According to Latour, the Pasteurization of France would not have been possible if Pasteurians had not inserted themselves into the French hygienist movement. Thus, they had first to translate the interests of the hygienists, and then those of the physicians, in order to gain them as allies in the war against microbes; in the meantime, as Pasteurians became the only authorities on microbes by virtue of their laboratory techniques, they also became an obligatory point of passage for those allies as they introduced the laboratory into hygienic and medical practices. This network of humans and non-humans (microbes), continues Latour, guaranteed success in the trials of strength and the strategies that caused microbes to remain (cultures, sterilization procedures, etc.). In conclusion, the Pasteurization of France was the setting up of this network and thus the trials which made the enemy (microbes) visible.[15]

From the point of view of the germ theory of disease, Latour's Actor Network theory shows how the new general law of disease diminished the uncertainty of vague concepts such as spontaneous morbidity and allowed hygienists to focus and hence strengthen their actions. Though this conclusion expresses one of the general outcomes of the "Pasteurization of France", other historical accounts of the way that microbes came to be defined and set up as part of the process of disease show other complexities.

In *Cultures of Bacteriology* (1996), "the intellectual history of bacteriology", John Andrew Mendelsohn traces the shaping of two traditions of thought and experiment on pathogenic microorganisms from 1880-1914: the French tradition led

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[13] Michael Worboys, *Spreading Germs. Disease Theories and Medical Practice in Britain, 1865-1900* (Cambridge: Cambridge University Press, 2000).
[14] Latour, *The Pasteurization of France*, p. 7. See also Latour, "Pasteur on Lactic Acid Yeast: A Partial Semiotic Analysis, *Configurations*, 1, 1 (1993) pp. 129-130.
[15] Latour, *The Pasteurization of France*, pp. 18-34; 40, 93,126.

by Louis Pasteur (1822-1895) and his immediate followers,[16] and the German tradition led by the German physician Robert Koch (1843-1910). Through the analysis of published works, personal correspondence, course notebooks and official reports, Mendelsohn shows that the emergence of these two research traditions had its foundations in two different worlds of experience, the agricultural world of Pasteur and the medical world of Koch. As part of this experience, Mendelsohn includes not only professional experience (chemists' or physicians' knowledge, identity and interests), but also the uncontested, little considered circumstances of their daily life.[17]

According to Mendelsohn, the ideas developed via Pasteur's microbiology between 1857 and 1877 were part of an agricultural science, particularly that of vineyards and the fermentation process. This agricultural setting explains why Pasteur saw microorganisms in a positive way: for him, microbes were necessary for the production of human goods such as wine, beer and cheese and were also crucial actors in the cycle of life and death; they were even "agents of universal hygiene". Mendelsohn argues that when Pasteur moved from the study of fermentation to the study of disease, he brought with him not only his methods but also this non-medical and positive way of thinking about microorganisms. Mendelsohn shows how the discoveries in Pasteur's laboratory during the 1880s such as virulence, variability of virulence, and attenuated vaccines, as well as the Pasteurian ideas regarding aetiology, epidemiology and history of disease, were remarkably continuous with early agricultural Pasteurian microbiology.[18]

Mendelsohn contrasts this Pasteurian general theory with the atomistic, purely medical microbiology of the Koch school. In contrast to Pasteur, Koch's medical experience in the war and as a district medical official caused him to see microbes as killers, and thus he was only interested in studying the pathogenic microorganisms in order to destroy them.[19] This difference, according to Mendelsohn, meant that there were at least "two distinct germ theories of disease", the French and the German.[20]

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[16] They are Emile Duclaux (1840-1904), Emile Roux (1853-1933) and the Swiss, Alexandre Yersin (1863-1943).
[17] Mendelsohn, *Cultures of Bacteriology*, pp. 9, 34.
[18] Ibid., pp. 66, 81, 165-170.
[19] Ibid., pp. 17-18, 100.
[20] Ibid., p. 19.

Mendelsohn also argues that the bacteriological revolution of the 1870s and 1880s was followed by a revolution in bacteriology itself: during the 1890s, microorganisms were increasingly found in healthy bodies where they had not induced illness, and it was also found that they could not survive in the environment, the breeding ground that had previously been thought of as their natural home. The idea that other microbes in the intestine had a relevant role to play in the production of disease, as well as the concept of healthy carriers, contributed to the final inversion of the original filth theory of germs: now, it was thought that the localization of epidemics was produced by the localization of the microbial flora in the body, not those in the environment as had been prescribed by the miasmatic theory and early germ theories. By the 1920s, concludes Mendelsohn, despite differences between diseases, institutional and national contexts, and research schools, bacteriological theory changed universally from the idea of infection as invasion to that of the intimate nature of infection.[21]

While Mendelsohn identifies two distinct germ theories of disease, the French and the German, Michael Worboys claims that there were many germ theories. In his book, *Spreading Germs* (2000), Worboys explores the negotiations surrounding the constructions, meanings and uses of germ theories and practices in Britain between 1865 and 1900.[22] On the basis of provincial and specialist literature and national journals, Worboys builds his history around four propositions: that there was not one but instead many germ theories of disease and of other phenomena; that practices and theories were equally important; that it is necessary to consider ideas about how the body reacted to germs; and finally, that germ theories and practices held new meanings for science and medicine. These four propositions have some methodological consequences: first, though centering his analysis on medical practitioners, Worboys extends it to veterinarians, surgeons and public health officers; second, he takes into account those who embraced chemical and non-bacterial germ theories. Thus, Worboys identifies two phases in the development and spread of germ theories of disease: from 1865 to 1882, the era of germs, and from 1882 to 1900 and beyond, the era of bacteria. During the first period, the contemporary references to the germ theory varied according to specific contexts and

[21] Ibid., pp. iv; 12; 447; 456-58; 479; 488; 594-623; 633-37; 776.
[22] Worboys, *Spreading Germs.*

different scopes: for example, surgeons borrowed Pasteurian germ theory to explain one process, sepsis, while maintaining the chemical theories for all other diseases. During the 1870s, disease germs were imagined to be many things and to act in various ways, but by the end of the decade physicians were increasingly discussing specific, observable microorganisms, especially bacteria, that were associated with specific diseases. Worboys finds that British physicians used the "seed and soil" analogy and its variants to refer to the relationship between bacteria and the body.[23]

After the 1880s, British germ theorists became bacteriologists by the spreading of Kochian theories of disease. Worboys argues that although British bacteriology is a synthesis of the Koch and Pasteur schools, it is better understood as an indigenous product: as they held weak institutional positions, physicians were more concerned with clinical and preventive interests and their work was disease-centered. This is the reason for the widespread use of the metaphor of "seed and soil", but also the reason why British scientists were so poor at staking discovery claims. Finally, Worboys shows how germ science and bacteriology were used to guide, legitimate and give meaning to practices old and new at every level.[24]

In conclusion, Mendelsohn, Worboys and Latour show that there is more than one history of the germ theory of disease. These representatives of the revisionist project reveal that there were multiple germ theories of disease, depending on the specific contexts. Their work also points out how deeply indebted particular understandings of germ theory were to pre-existing traditions of explaining disease. However, neither Mendelsohn nor Latour deal with medical geography, the medical field that developed in France and Germany – Pasteur's and Koch's home countries - as part of these pre-existing traditions. Let us then move to the historiography of medical geography.

Trends in the history of medical geography and the problem of the transition to germ theory

[23] Ibid., pp. 277-86.
[24] Ibidem.

Conevery Bolton Valencius has written a very good historiographical analysis of medical geography.[25] Valencius affirms that almost all historical accounts trace the genesis of the study of health and environment to the Hippocratic treatise *On Airs, Waters, and Places*. This consensus is clearly expressed in Hannaway's chapter "Environment and Miasmata" in the *Companion Encyclopedia of the History of Medicine* (1997). Hannaway shows how the environmental Hippocratic view was extended to medical topography and medical geography from the seventeenth century onwards. Sydenham's concept of epidemic constitution and the use of instruments such as the thermometer and barometer to measure the carrier of the miasma, the air, was part of that first effort. From the eighteenth century onwards, medical topography became more complex due to the introduction of many environmental, local and social considerations in the production of disease, which were much expanded beyond airs, waters, and places. First Italian medicine, and later that of the French and Germans, expanded this concept to national scales and produced studies known as medical geographies for a large part of the nineteenth century.[26]

According to Valencius, recent works on medical geography have analyzed this field as part of the efforts of early European states to assert control over territory, including their colonies. Thus, some historians have found ties between the emerging conceptions of localism, state-self definition and medical ideas. Others have focused on the use of tools of exact measurement within a set of practices centered on investigations into the way that environment determined human health. In that case, medical geography would have encompassed elements from geography, meteorology, medicine, cartography and geology. Other historians have also considered medical geography as a contribution to an environmental discourse on race and racial difference. Ideas about otherness, race, and environment, have been used to explain the way in which European and American explorers and settlers viewed land as different from that to which they were accustomed or essentially similar in degree rather than in kind. Finally, some historians have detailed how the very organization of medical geography was shaped by territorial expansion and the colonial context. In France, for example, the field of medical geography was created

[25] Valencius, "Histories of Medical Geography".
[26] Hannaway, "Environment and Miasmata", pp. 295-7; 300-302.

primarily by military physicians and those connected with militarily-enforced settlement.[27]

In relation to the meaning of disease during the transition from medical geography to germ theory, Valencius's analysis suggests a consensus among historians on the idea that in the early part of the twentieth century the older language and concerns of medical geography were taken up by those interested in the diseases of hot climates, but with a fundamentally different epistemic basis. Tropical medicine, rather than medical geography, would become the rubric by which to investigate disease in different lands. The concept of environment would have lost much of its holistic embrace, becoming a narrow harborer of pathogens instead of a unified set of influences. However, Valencius continues, other accounts point toward the co-existence of the concepts of environmental determinism and medical geography and newer ideas of laboratory pathology and germ theory.[28]

Michael Osborne is one of the few historians who address the problem of the transition from medical geography to germ theory, in his history of medical geography in France.[29] Osborne describes how medical geography was closely aligned with military and expeditionary hygiene on the basis of Hippocratic ideas. According to him, though Pasteur's microbes provided a target for action and an etiological focus for many hygienists, traditions of medical geography persisted into the twentieth century; those critical of laboratory medicine either resisted the all-sufficient germ theory of disease or struggled to accommodate germs into their medical cosmos. Others saw the work of Pasteur as "not the destruction of the work of Hippocrates, but its complement, development and provisional coronation" or tried to present a medical geography that was integrated with the most recent scientific findings and ready for application. In France's case, according to Osborne, the germ theory of disease was not the only reason for the decline of medical geographical activity; the success of the medical specialty of hygiene was also instrumental because medical geography failed to attain the status of a recognized medical specialty, as did hygiene.[30]

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[27] Valencius "Histories of Medical Geography", pp. 14-16.
[28] Ibid., pp. 20-21.
[29] Osborne, "The Geographical Imperative".
[30] Ibid., pp. 46-50.

Marcos Cueto's paper "Nationalism, Carrión's Disease and Medical Geography in the Peruvian Andes" (2003) is one of the few whose main concern is the transition from medical geography to germ theory; it follows the case of Carrion's disease, today known as bartonellosis.[31] Cueto shows how, during the turn of the twentieth century, the medical community in Peru reinforced the uniqueness of the disease environment of the Andes by studying this native ailment. The Peruvian physician Ernesto Odriozola (1862-1921), who wrote on Carrion's disease in 1898, asserted that the rare combination of moderate high altitude, hot temperature and poor air circulation explained why the disease only existed in the Peruvian Andes. Cueto shows how this work was the basis of a national medicine which helped not only to reinforce the geographical and ethnical division of the country, but also to enhance the special and rich geographical features of the country, awaken international medical interest in Carrion's disease, and show that Peruvians could make original international contributions. During the first decades of the twentieth century, some laboratory studies on Carrion's disease took up a great deal of the energy in Lima's Medical School until the bacteria and the mosquito related to the disease were found and fully accepted by scientific communities in Peru and abroad. Cueto concluded that, as a result, the visibility of medical geography diminished; only provincial doctors, and studies of the social dimensions and popular treatments, continued to use the geographical descriptions.[32]

With regards to Colombia, the history of medical geography and the germ theory have rarely been the focus of scholarly works. I have previously explored how and why physicians built and consolidated the notion of Magdalena fevers (the periodic fevers of the Magdalena River valley) between 1860 and 1886, and the place of medical geography in that process.[33] The question of the destruction of the Magdalena fever notion by the new germ theory of disease was actually the origin of the idea for this dissertation. Jorge Márquez Valderrama's book on the assimilation of Pasteurian science in the Antioquia region (2005), on the other hand, analyses medical discourses during the medicalization of the city of Medellín, that is, during

[31] Cueto, "Nationalism, Carrión's Disease".
[32] Ibid., pp. 322-31.
[33] Claudia Mónica García, "Las 'fiebres del Magdalena': medicina y sociedad en la construcción de una noción médica colombiana, 1859-1886", *Historia, Ciencias, Saude-Manguinhos* (Rio de Janeiro), 14, 1, (2007), pp. 63-89;

the years of consolidation of the medical profession and its growing intervention in the hygiene of the city.[34] Márquez follows this process through the lens of Gaston Bachelard's notion of "epistemological obstacles" to the development of knowledge, and thus finds that the old neo-Hippocratic ("air-related" or miasmatic) theories were both a receptacle and an obstacle to Pasteurism in Medellín.[35] According to Márquez, doctors in Antioquia, working with the miasmatic framework, either identified germs as synonymous with miasmas or used them to displace old contagion notions. He also finds that despite Pasteurism, hygienic practices continued to be based on the old miasmatic and "aerist" model until at least the end of the century.[36] Finally, Diana Obregón has identified the local actors involved in the diffusion and practice of bacteriology in Colombia. She points to the main issues in the transition of what she calls "the miasmatic medicine" to the bacteriological: the debates around anthrax in the 1860s and leprosy in the 1880s. Her analysis is based on the socio-constructivist perspective, with the intention to show the social aspects involved in the knowledge and practices associated with Pasteurism in Colombia and also to contest the idea that scientific knowledge is passively transmitted from centers to peripheries.[37] Obregón develops the former argument in her book on leprosy in Colombia. There she shows evidence of a kind of syncretism between miasmatic and Pasteurian notions of leprosy in the 1880s, and also highlights physicians' use of Pasteurism in their rhetoric to gain social legitimation.[38]

In conclusion, the scarce literature on medical geography and Pasteurism in Colombia shows that before the arrival of any germ theory in Colombia, physicians relied on miasmatic, neo-Hippocratic or medical geographical explanations for disease. It also shows that there was a period of coexistence and/or synthesis of aspects between both models. However, there is no deep analysis of the pre-bacteriological model of disease causation in Colombia; there is no scholarship that questions the changing meanings of germs (or "parasites" as contemporaries called

[34] Jorge Márquez Valderrama, *Ciudad, miasmas y microbios. La irrupción de la ciencia pasteuriana en Antioquia* (Medellín: Universidad de Antioquia, 2005).
[35] Ibid., pp. 75-76.
[36] Ibid., 26, 41-43.
[37] Diana Obregón, "Sobre epidemias, endemias y epizootias: algunos aspectos del desarrollo de la bacteriología en Colombia", *Biomédica*, 18 (2), 1998: 110-121.
[38] Diana Obregón, *Batallas contra la lepra: Estado, Medicina y Ciencia en Colombia* (Medellín: Eafit, 2002).

them), nor any that analyzes how doctors fit that new notion into the medical geographical scheme. As such, the purpose of this dissertation is to offer a thorough description and explanation of the conceptual choices that Colombian doctors made with regards to those problems, according to their particular context.

The subject of this investigation and the theoretical approach

It is possible to put forward answers to the question of how the transition from pre-bacteriological to bacteriological theories might have occurred along three different lines: at one end there are those who defend the idea that bacteriology represented a natural or logical development in the history of medicine, and therefore see foreshadowing of the modern understanding of germs in the ideas of the past (e.g. Bulloch, Foster and Carter); at the other end there are those who defend a radical incommensurability between pre-bacteriological and bacteriological identity of diseases. Indeed, Andrew Cunningham considers that given the fact that plague and other infectious diseases were defined by different criteria (mainly by symptoms) before laboratory medicine, pre-laboratory and laboratory definitions of diseases are incommensurably different.[39] Between these two positions, there are scholars such as Mendelsohn, Osborne, Cueto, Marquez and Obregón, who claim that there was a kind of coexistence and/or synthesis between the pre-bacteriological and bacteriological models of disease causation that lasted until or beyond the end of the century.[40] Historians of the first group search past documents for aspects that are familiar to their own understanding of germs, leaving aside some of the complexities of contemporaries' causal notions of diseases; historians of the second group do the opposite and point out the differences between pre-bacteriological and bacteriological notions, assuming that they were separated in contemporaries' minds.

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[39] Andrew Cunningham, "Transforming Plague", pp. 213-223; 242.

[40] Other works by Andrew Mendelsohn and Michael Osborne are: Andrew Mendelsohn, "'Like All That Lives': Biology, Medicine and Bacteria in the Age of Pasteur and Koch", *Hist. Phil. Life Sci.*, 24 (2002), pp. 3-36. Michael Osborne, "'Resurrecting Hippocrates": Hygienic Sciences and the French Scientific Expeditions to Egypt, Morea and Algeria", in David Arnold (ed.), *Warm Climates and Western Medicine : The Emergence of Tropical Medicine, 1500-1900* (Amsterdam/Atlanta: Rodopi, 1996), pp. 80-95.

Finally, historians of the third group discuss how and to what extent climatic notions coexisted or synthesized with the new germ theory, and question the extent to which they were radically different. In this research, I have followed this latter position with a new focus.

Following the constructivist perspective of the recent historiography of the germ theory, I assume that nineteenth-century Colombian doctors may have held multiple meanings of germs, and that those meanings were indebted to pre-existing traditions for explaining disease. I also assume that, as Conevery Bolton Valencius says, the transformations brought about by germ theory varied according to fundamentally disparate influences derived from the particularity of each national context.[41] But I have also found in the sociology of scientific knowledge (SSK), ideas that contribute to the building of my approach to the problem of this research, in particular the Strong Program.

In an effort to demonstrate the social character of scientific knowledge, the scholars behind the Strong Program have developed an explanation for the conventional character of knowledge. According to this theoretical perspective, we cannot assume that the explanation for the cognitive decisions of scientists resides either in natural reality or in the logical structures of individual cognition; it must be found in the contingent judgment involved in scientific work.[42] Thus, I follow the scholars working within SSK and the Strong Program in supposing that the content of knowledge is radically under-determined by the forms and structures of natural reality; that the application of scientific concepts to the natural world is ultimately a matter of inductive judgment, not deductive logic; and that the meaning and use of such concepts is therefore capable of changing incrementally over time.[43] This is important to my study of changing concepts of disease theories in Colombia in a number of ways. It means that I do not attribute any fixed or essential character to any of the theories that I address. I do not assume that either medical geography or germ theory have any specific meaning or content, nor that they imply any particular ideas or practices, nor – importantly – that they are incommensurable with one

[41] Valencius, "Histories of Medical Geography", p. 24.
[42] See Barry Barnes, David Bloor and John Henry, *Scientific Knowledge. A Sociological Analysis* (London: Althlone, 1996), particularly chapters 2 and 3; see also Barry Barnes, "On the Conventional Character of Knowledge and Cognition", *Philosophy of the Social Sciences*, 11, 3 (1981), p. 309.
[43] Barnes, Bloor and Henry, *Scientific Knowledge*, pp. 54-6.

another. Rather, adopting this perspective means that I can pay close attention to the particular ways in which theoretical concepts were mobilized in specific debates and in relation to specific practices, and can observe the ways that such usage changed over time and combined different ideas and different elements in different settings.

Secondly, I follow SSK in also supposing that while the use and meaning of scientific concepts is not determined by natural reality, it is subject to social surveillance and regulation by relevant communities of actors, and as such is shaped by the practical, professional and social interests of those communities.[44] This provides me with a means to understand why different actors used medical ideas and theories in the ways they did, and enables me to root my analysis of changing ideas about disease in the specific social and professional setting of late nineteenth-century Colombia. Thus, I intend to give a thorough account of what I have called the transition from medical geography to germ theory in Colombia.

As I said earlier, in exploring the transition from medical geography to germ theory by following specific diseases (periodic and continuous fevers and leprosy), I will also be addressing the question of the transformation of the identity of diseases. I will approach this problem in the same way as with my treatment of the transition from medical geography to germ theory; that is, following the central tenets of the SSK and the Strong Program. I do not assume that there is an over-arching or core set of ideas about what disease is. Rather, I assume the possibility of a diversity of disease theories being deployed in different settings or relating to different diseases. Historians of medicine such as Ludmila Jordanova, Andrew Wright and Ivan Crozier have debated how a similar approach (called by some the "constructivist" perspective) would benefit history of disease and disease concepts.[45] Peter Wright and Andrew Treacher, for example, have pointed out that traditional historians have approached diseases as natural entities, thus limiting their history to the problem of

[44] Ibid., pp. 190-191.
[45] Ludmila Jordanova, "The Social Construction of Medical Knowledge", *Social History of Medicine*, 8, 3 (1995), pp. 361-381; Ivan Crozier, "Social Construction in a Cold Climate". Discussion Point. *The Society for the Social History of Medicine*, 13, 3, 2000: 535-546; and finally Peter Wright and Andrew Treacher, "Introduction" in Ibid. (ed.), *The problem of Medical Knowledge: Examining in Social construction of Medicine* (Edinburgh: Edinburgh University Press, 1982).

the time of emergence or the social cause of some diseases. On the contrary, Wright and Treacher say, the constructivist perspective allows historians to see that diseases are not just natural entities but also conditions that do not necessarily have trans-historical and universal form.[46] One of the most quoted definitions that embrace this idea is Charles Rosenberg's:

> …disease is at once a biological event, a generation-specific repertoire of verbal constructs reflecting medicine's intellectual and institutional history, an occasion for and potential legitimation of public policy, an aspect of social role and individual – intrapsychic- identity, a sanction for cultural values, and a structuring element in doctor-patient interaction. In some ways disease does not exist until we have agreed that it does, by perceiving, naming, and responding to it.[47]

With regards to medical knowledge in particular, Wright and Treacher in 1982 and Adrian Wilson in 2000 have denounced an asymmetry in the history of medical knowledge. They say that when historians make retrospective diagnoses, not only do they obliterate the radical historicity of medical knowledge; they also assume that current medical notions are correct and therefore do not need to be explained whereas past knowledge is judged false and therefore explainable through social causes.[48] As I have explained, in examining the transformation of the identity of fevers and leprosy in this research, I am not using any pre-determined definition of disease; references to modern knowledge would therefore be redundant.

The content of this dissertation

In accordance with my general purpose, and following the theoretical premises mentioned above, in Chapter 1 I will explore the content of the Hippocratic causal framework of diseases in Colombian medicine that consolidated around the 1830s, explain how it transformed into the medical geography perspective by late 1850, and also explain how this transformation reflected on the case of periodic fevers. Having

[46] Peter Wright and Andrew Treacher, "Introduction", p. 4 and Adrian Wilson, "On the History of Disease-Concepts : the Case of Pleurisy", *History of Science*, No. 38, 2000, pp. 273-276.
[47] Charles Rosenberg, "Framing Disease: Illness, Society, and History" en *Explaining Epidemics* (New York: Cambridge University Press, 1992), p. 305.
[48] Wright and Treacher, "Introduction", pp. 4-5.

set up the complexities of the pre-bacteriological notions of disease causation that existed in Colombia around mid-century, the other four chapters will detail the transformation by the germ theory by focusing on three cases: periodic fevers (yellow fever and malaria) in Chapters 2 and 3; continuous fevers (typhoid fever and typhus) in Chapter 4; and finally leprosy in Chapter 5.

In Chapter 2 I will explore the controversy surrounding a germ theory practice, the preventive inoculations against yellow fever, performed in 1887 in the village of Cúcuta in the northeastern part of the country. The controversy involved the local and national communities and had practical and conceptual consequences for the management and understanding of yellow fever and malaria, hitherto considered variations of one and the same disease (periodic or intermittent fevers).

In Chapter 3 I will analyze how this controversy, and the changing epidemiological behavior of fevers, helped to develop different ideas about the possible role of germs or microorganisms in the development of both diseases, and how doctors then reconciled the medical geographical and bacteriological perspectives with regards to those fevers. I will show that, in the process, doctors finally consolidated the emerging view that yellow fever and malaria were distinct diseases.

In Chapter 4 I will analyze how Colombian physicians mobilized medical, geographical and bacteriological arguments in their debates on the nature of typhoid and typhus fever. I will explain what typhoid and typhus meant to mid-century Colombian doctors and how they used neo-Hippocratic, medical geographical and clinical thermometry ideas and techniques to shape those meanings. I will explore arguments in favor of, and against, the bacteriological identity of typhoid fever, and look at how clinicians, physicians-hygienists and local authorities used bacteriology in discussions of and practices related to the typhoid epidemics of 1887-1890 in Bogotá.

In Chapter 5 I will explore the case of leprosy. I will show that the identity of leprosy revolved around its contagious or hereditary character as well as around the neo-Hippocratic scheme of diseases. I will also show that although a medical geographical approach to leprosy was not dominant, doctors still used the climatic approach to leprosy even when they accepted germs as the primary cause. I will also

explain the role of therapeutic practices such as antiseptics and serotherapy in the acceptance of the germ cause of leprosy.

In the conclusions, I will return to the general problem, highlight the main periods that characterize the transition from medical geography to germ theory in Colombia and discuss some points relevant to the Colombian case.

The reader may expect me to cover the history of antiseptic surgery or the debates on anthrax in Colombia, since they are usually critical to the history of the germ theory in other contexts. The evidence suggests that Listerism was indeed included in the Colombian medical literature from the mid-1870s and that it was presumably incorporated into surgical practices during that decade.[49] I also know that doctors discussed the germ cause of anthrax during the 1869 and 1886 epidemics in the savannah of Bogotá and other regions.[50] When I designed the project, I decided to be open to these two subjects even if they were at the periphery of the problem that I will explore in this dissertation. The fact that I found no sound debates regarding either topic among physicians, at least to the level of those generated by the diseases I analyze in this dissertation, and considering that, as I said, they were at the periphery of the subject of this research, I thus decided not to include them in this dissertation. I will briefly return to this point in the conclusions.

About the sources

Considering that the focus of this investigation is the cognitive decisions that Colombian doctors made before and during the transformation of the causal framework of diseases by the arrival of the Pasteurian germ theory, I chose to follow discussions within the medical and hygiene societies created in Colombia in the

[49] For example, eight papers of the *Revista Medica* deal with Joseph Lister's work in antiseptic surgery, between 1875 and 1884.

[50] Manuel Ancízar, "Documentos Relativos a la *epizootia* reinante en el ganado de la sabana de Bogotá", *Diario Oficial*, Año V, No. 1593, 23 de abril (1869), pp. 464-5; Antonio Ospina and Andrés María Pardo, "Memoria sobre la epidemia epizootia reinante en el ganado vacuno i lanar de la sabana de Bogotá", *Anales de la Universidad Nacional de los Estados Unidos de Colombia*, Tomo II, Bogotá, 1869, pp. 28-38; Antonino Gómez C., "La pústula maligna. Observada en los Estados de Santander y Boyacá", *Revista Médica*, Serie X, No. 103, 20 de junio, 1886, pp.: 55-66; SMCN, "Acta de las secciones ordinarias de los días 7, 14, 21 y 28 de septiembre y 5, 12, 19 y 20 de octubre de 1886", *Revista Médica*, Serie X, No. 108, 20 de noviembre, 1886, pp. 289-304.

nineteenth century (the *Sociedad de Medicina y Ciencias Naturales de Bogotá* – SMCN - 1873, called *Academia Nacional de Medicina* – ANM - from 1891 onwards; *Junta Central de Higiene* – JCH - 1886; *Academia de Medicina de Medellín*, 1887; *Sociedad de Medicina del Cauca*, 1888; *Sociedad de Ciencias Médicas*-Bucaramanga, 1893). The proceedings of their debates on clinical cases, diseases, problems with hygiene policy and international medical knowledge appeared in the societies' own journals (see bibliography). Note that the first medical society was only created in 1873 in Bogotá, and the other by the end of the 1880s. Note also that medical journals – and medical literature in general - were not very abundant in nineteenth-century Colombia: the first medical journal was published in 1852 (*La Lanceta*) and only lasted a few months. The second was published from 1864-1867 (*Gaceta Médica*), and the third from 1873 (*Revista Médica*). This last, and the *Revista de Higiene*, published since 1887, survived the end of the century.

Medical books by Colombian doctors, though scarce, were a good source for some of the issues that I will deal with in my dissertation; theses by medical students were also valuable because they tend to show conflicting views on diseases during the years of the arrival of the germ theory. Official press such as *Diario Oficial*, which sometimes recorded medical and hygiene issues, and the *Registro Municipal*, the journal of the council of Bogotá among others, were also useful for completing the set of views on the issues debated within the medical and hygiene societies. I found all medical journals, press and medical books in the *Biblioteca Nacional* and the *Biblioteca Luis Angel Arango* in Bogotá.

Finally, I also used some of the French medical textbooks that were commented upon or translated by Colombian doctors. When not available online, I found them in the Special Collections of the University of Edinburgh Library as well as in the Countway Medical Library at Harvard University.

CHAPTER 1

CLIMATE AND DISEASE IN THE PRE-BACTERIOLOGICAL ERA, 1830-1880

Introduction

Historians of medicine have widely recognized that before the establishment of the germ theory of human diseases, explanations for disease causation were dominated by environmentalist notions that can be traced back to the Hippocratic text *Airs, Waters* and *Places*. Scholars have shown that versions of this environmentalist framework, particularly those from the nineteenth century, took the form of medical geography which incorporated tools, data, and concepts from the natural sciences and the emerging science of geography.[51] As this chapter will show, Colombian medicine in the nineteenth century inherited these two western traditions. Hippocratism was introduced to Colombian medical schools during the late years of Spanish domination, which formally ended in 1810. From the middle of the century, physicians transformed this Hippocratism into the study of the medical geography of the country. In this chapter, I will begin by analyzing these two medical versions of

[51] For a summary of the historical relationship between Hippocratism and medical geography in western medicine see Caroline Hannaway, "Environment and Miasmata", W.F. Bynum and Roy Porter, *Companion Encyclopedia of the History of Medicine* (London and New York: Routledge, 1993), pp. 293-308. For an overview of the history of medical geography see Nicolaas A. Rupke (ed.), "Medical Geography in Historical Perspective", *Medical History*, Supplement No. 20 (London: The Welcome Trust Centre For the History of Medicine at UCL, 2000). For an example regarding the French case see Michael Osborne's paper, "'Resurrecting Hippocrates": Hygienic Sciences and the French Scientific Expeditions to Egypt, Morea and Algeria", in David Arnold (ed.), *Warm Climates and Western Medicine : The Emergence of Tropical Medicine, 1500-1900* (Amsterdam/Atlanta: Rodopi, 1996), pp. 80-95; on British colonies see, for example, Mark Harrison, "'The Tender Frame of Man': Disease, Climate, and Racial Difference in India and the West Indies, 1760-1860", *Bulletin of the History of Medicine*, 70.1, 1996, pp. 68-93. For Germany see Nicolaas A Rupke, "Humboldtian Medicine", *Medical History*, 40, 1996, pp. 293-310. On America see Conevery Bolton Valencius, *The Health of the Country: How American Settlers Understood Themselves and Their Land* (New York: Basic Books, 2002). There are few works about medical geography in Latin America. See for example Marcos Cueto, "Nationalism, Carrión's Disease and Medical Geography in the Peruvian Andes", *History and Philosophy of the Life Sciences*, 25, 2003, pp. 319-335; Enrique Beldarraín, "Evolución Histórica de la Geografía Médica en Cuba", *Ilé. Anuario de Ecología, Cultura y Sociedad*, 3, 3, 2003, pp. 93-110; and, finally, Claudia Mónica García, "Las 'fiebres del Magdalena': medicina y sociedad en la construcción de una noción médica colombiana, 1859-1886", *Historia, Ciencias, Saude-Manguinhos* (Rio de Janeiro), v. 14, n. 1, ene.-mar. 2007, pp. 63-89.

climatic determination of disease that dominated nineteenth-century Colombian medicine before the arrival of the germ theory. I will then proceed to show how these explanations incorporated physicians' vision with regards to social groups, as well as how these ideas were strongly related to their concern for the economic conditions of the country and their struggle to consolidate a professional/scientific body.

In the first section I will analyze the content of the Hippocratic causal framework of diseases in Colombian medicine that consolidated around the 1830s, and describe how physicians used these ideas to explain the 1830 epidemics in Bogotá. In the second section I will analyze the way that this neo-Hippocratism incorporated notions of the differences between the *castas* – or *races* - and their response to diseases, i.e. acclimatization. In the third section I will describe the content of the medical geography that emerged by 1850: the divide between diseases of the warm, temperate, and cold lands. In the fourth section I will examine how physicians analyzed fevers of the low warm lands following medical geographical principles, as well as the socio-professional reasons for physicians' interest in studies of medical geography. In the last section I will offer some comments on the transformation of the first Hippocratic causal framework by medical geography and its consequences for the next few decades.

As the core Hippocratic and medical geography schools were rooted in the physicians' experience of the geographical and social landscape of their country, it may be useful first to provide a very brief introduction to Colombia, or New Granada as it was known during the colonial period.[52] The Colombian landscape is structured by the Andes, which cross the country from south to north in three principal mountain ranges, the western, central and eastern *cordilleras*. Between them lie the lowland valleys through which run the Cauca and the Magdalena Rivers, connecting the mainland with the Caribbean and foreign commerce. The tropical location of Colombia sees little variation in temperature throughout the year, but the presence of the Andes adds a significant reduction of temperature with altitude to the equation,

[52] After the wars of independence against the Spanish rulers, the Colombian republic was formed by the present territories of Venezuela, Equator, Colombia and Panama. In 1830 that republic was divided and the republics of Venezuela, Equator and New Granada emerged. Despite several constitutional changes that would further modify the name of the country (Granadine Confederation in 1861, The United States of Colombia, 1863, and the Republic of Colombia, 1886), for the sake of clarity, I will use the name of Colombia in this thesis.

which has had considerable effects on both population patterns and the culture of the country. The highlands of the *cordilleras* have been home to the majority of the population throughout Colombia's varied history, from the pre-Hispanic era through Spanish conquest and settlement, and even after independence (1810). The main population centre has been the highland plateau around Bogotá on the Eastern *cordillera*. The Spanish conquerors of the sixteenth century found a vast source of indigenous workforce in this region and set up their political headquarters in the city of Santa Fe, later renamed Bogotá. This contrasted sharply with the low population of the lowlands, which tended to be inhabited by sparser, indomitable tribes. From the sixteenth century onwards, the Spaniards imported African slaves, mainly to work in the mines and haciendas of the coastal plains. Thus, Indians, Spaniards and Africans were the basic social groups that would shape the period of Spanish rule from the sixteenth century onwards. However, in the Colombian case, *mestizaje* (the mixture of Europeans with other groups), as well as the adoption of the dominant culture, language and religion by non-Europeans, was widespread. By the end of the eighteenth century, the majority of the inhabitants in all regions were thus *mestizos* (a term that was frequently used to refer specifically to the mixed-race descendants of "whites" and Indians), although people could easily see the way in which the region had been populated from their physiognomy, occupation and subtle cultural traits. As I will show in this chapter, this will be significant for an understanding of Colombian physicians' perspectives on the relationship between environment, people, and diseases during the nineteenth century.

Neo-Hippocratism in the first half of the nineteenth century

Hippocratism was part of the first medical curriculum delivered in Santa Fe during Spanish Rule at the *Colegio Mayor del Rosario*, starting in 1802, and the *Colegio de San Bartolomé*, starting in 1813.[53] Courses in the *Colegio Mayor del Rosario* were

[53] Pedro María Ibáñez, *Memorias para la historia de la medicina en Santafe de Bogotá* (Bogotá: Imprenta Nacional, 1968 [First edn. 1884]), p. 43. Even though the *Colegio de San Bartolomé* was created in 1813, lectures were not delivered there until 1819. See Emilio Quevedo and Camilo Duque, *Historia de la Cátedra de Medicina, 1653-1685* (Bogotá: Universidad El Rosario, 2002), pp. 122, 146, 149.

based on the plan by Father Miguel de Isla that included the Hippocratic aphorisms, presented with commentary by Spanish authors, and the Hippocratic treatise *De aere, aquis et locis*.[54] The wars of independence against the Spanish crown (1810-1819) disrupted this medical training until the new republic established its educational institutions in 1826. In that year the government issued an Educational Plan that included the creation of the *Universidad Central* with a Faculty of Medicine, Surgery and Pharmaceutics in Bogotá.[55] This university, reformed in 1842 as the *Universidad del Primer Distrito*, lasted until 1850 and licensed the majority of certified medical practitioners of the first half of the nineteenth century.

The 1826 Educational Plan ordered the Faculty to design a course in medical sciences which would be "adjusted to the climate, constitution and diseases of the inhabitants of Colombia."[56] The starting point of this "adjustment" was stipulated in the same plan: French treatises of pathology and hygiene such as Auguste François Chomel's *Élémens de pathologie générale*[57] and Etienne Tourtelle's *Elemens d'hygiene* among others.[58] In fact, this "adjustment" can be traced back to several noted translations of the above French treatises, which used the neo-Hippocratic scheme of causality as an etiological framework. These translations were made by the physician José Félix Merizalde (1787-1868), one of the most influential

[54] Ibáñez, *Memorias para la historia*, p. 43.

[55] After the victory of Simon Bolivar over the Spanish troops in 1819, the Colombian State was formed by three departments, Cundinamarca, Venezuela and Ecuator; the 1826 Plan of Studies created universities in each of their capitals, Santa Fe de Bogotá, Caracas, and Quito. By 1830 this State, known as the *Gran Colombia* among historians, had dissolved into three new States, the current Colombia, Venezuela and Equator.

[56] Colombia, Secretaría del Estado y del Interior. "Decreto 720 [3 de Octubre, 1826]. Reglamentación de la Educación Pública, Artículo 216" in José M. de Mier, *La Gran Colombia. 2. Decretos de la Secretaría del Estado y del Interior, 1825-1826* (Bogotá: Presidencia de la República, 1983), p. 644.

[57] Auguste Françoise Chomel, *Élémens de pathologie générale* (Paris, Crochard/Gabon, First edn. 1817). It was also suggested that the Hippocratic Aphorisms be used directly in the Colombian medical curriculum. See Colombia, Secretaría del Estado y del Interior. "Decreto 720, p. 630.

[58] Etienne Tourtelle, *Elemens d'hygiene: ou, de l'influence des choses physiques et morales sur l'homme, et des moyens de conserver la sante*. Merizalde did not specify which edition he consulted. By 1826 there were four editions: Strasbourg: F.G. Levrault, imprimeur-libraire, 1796/7 and Paris: Théophile Barrois, libraire, An V [1797]; Paris: Levrault frères, Second edn. 1802; Paris: Raemont et Fils, Third edn. 1815; Paris: Fourth edn., 1823. Tourtelle's book had been one of the textbooks in the *Colegio of San Bartolomé*, introduced by its founder, José Félix Merizalde, but there is no evidence for the exact function of this course. See Quevedo y Duque, *Historia de la Cátedra*, pp. 145-146. The other hygiene text proposed in the 1826 educational plans was "Foderé's book on public hygiene" (which must have been *Les lois éclairées par les sciences physiques, ou traité de médicine légale et d'hygiene publique* (1798) as the Spanish translation had been available in Bogotá since the early nineteenth century according to the catalogue of the Biblioteca Nacional de Colombia). See Colombia, Secretaría del Estado y del Interior. "Decreto 720, p. 630.

Colombian physicians of the first half of the nineteenth century. Merizalde graduated from the *Colegio Mayor del Rosario* in 1810 and then held several important medical positions during the wars of independence and their aftermath, serving as chief physician of the army hospital in Bogotá until 1830. He had produced the official regulations for pharmacies in the 1830s, and founded the vaccination office in 1841.[59]

Merizalde joined the new faculty of medicine at its inception in 1826, and in his capacity as a professor he decided to excerpt and translate sections of Tourtelle's book *Elemens d'hygiene*, due to the lack of original copies of medical textbooks for students.[60] Later, Merizalde published his pathological lectures based on the first edition of Auguste-François Chomel's book *Élémens de pathologie générale*.[61] In fact, Merizalde's pathological lectures were, aside from mild changes, a literal translation of Chomel's book.

Dr. Merizalde's task of adapting medical sciences to the local situation required a simple addition of some of his personal observations to the mainstream knowledge represented by the French textbooks. Indeed, it was not his aim to contest any concept or even to make an original contribution to the current medical knowledge. However, by excerpting, modifying and adding to the French books, Merizalde sought to harmonize the physical reality of Colombian geography and the pathological profile of the population with the medical theories of the European doctors. For the purpose of this chapter, I will focus on the sections concerning

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[59] Ibáñez, *Memorias para la historia*, pp. 104-7.

[60] José Félix Merizalde, *Epítome de los elementos de higiene o de la influencia de las cosas físicas i morales sobre el hombre i de los medios de conservar la salud. Extractados de Estevan Tourtelle i traducidos al castellano, i añadidos con otras observaciones nuevas por José Félix Merizalde* (Bogotá: Imprenta de Pedro Cubides, 1828). See section "Aviso", s/p.

[61] José Félix Merizalde, *Elementos de patología general* (Bogotá: J. N. Barros, 1831), p. 165. Based on the first edition of Auguste Françoise Chomel, *Élémens de pathologie générale* (Paris, Crochard/Gabon, 1817). The analysis I present here of the etiology of the almost literal translation of Chomel's book complements the analysis made by L.S. Jacyna of the diagnostic and treatment stances by the French physician during the 1810s and 1820s. Jacyna shows that by 1817, Chomel acknowledged the new bedside techniques of physical examination (percussion and auscultation), which contrasts with his Hippocratic posture on therapeutic issues that apparently conceded major claims of therapeutic nihilists (most diseases are susceptible to cure without active treatment, by the beneficence of the nature alone). As Jacyna says, while methods of investigating the disease process in the body had undergone major changes since the turn of the century, methods of treatment changed relatively little; as a conclusion of the analysis I present below, I can add that etiological notions, at least as presented by the 1817 edition of Chomel's book, had also changed little: they continued to be Hippocratic. See L.S. Jacyna, "Au Lit des Malades: A.F. Chomel's clinic at The Charité, 1828-9", *Medical History*, 33, 1989, pp. 434, 444, 447.

etiology in order to locate the role of the environment in that etiological scheme. This will allow me to show more precisely how the "accommodation" process ordered by the Faculty took place in practice, that is, in the analysis of the 1830 epidemic by Merizalde and his colleagues in the Faculty of Medicine.

The environmental framework of disease causation in Merizalde's books fell within the Hippocratic themes used to explain body imbalance. Merizalde's commitment to the Hippocratic environmental ideas is shown by the paragraph he appended to his translations of Tourtelle's chapter "On waters and localities:"

> After attentively reading this chapter, most of which is taken from Hippocrates's book Airs, Waters and Places, whenever the reader compares the places that Hippocrates talked about with those that he inhabits, he [the reader] can conjecture about the situation regarding health, climate, the nature of waters, food, costumes, diseases, etc. Guided by this knowledge the reader could apply those precepts given in this book for preserving health, prolonging life and avoiding diseases.[62]

An analysis of the place of Hippocratic environmental elements in the causal scheme of disease in nineteenth-century medicine requires consideration of the other elements that were thought to be involved in the production of body imbalance, namely the "non-naturals:" nutrition, excretions, movements and perceptions.[63] As Merizalde's lectures reveal, nineteenth-century medicine placed all the non-naturals, as well as the environmental elements, within the etiological scheme of specific, predisposing and occasional causes, that is, according to the way in which those environmental and non-natural elements acted in the development of disease.[64]

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[62] Merizalde, *Epitome de los elementos de higiene*, p. 153.

[63] Hannaway, "Environment and Miasmata", p. 293. For a historical account of the origin of the non-natural elements in disease causation see Vivian Nutton, "Humoralism", W.F. Bynum and Roy Porter, *Companion Encyclopedia of the History of Medicine* (London and New York: Routledge, 1993), pp. 288-9.

[64] Merizalde, *Elementos de patología*, p. 12. As Margaret Pelling has noted, the traditional structure of causation was based on the classical epistemology hierarchy of causes: Aristotle's formal, final, material, and efficient. Thus, the nineteenth century usage, at least in the English speaking world, included: the first or primary causes, that is, cosmological or divine; remote causes, related to the state of the atmosphere or influences broad enough in scope to bring about the rise and fall of epidemics (the epidemic constitution of Thomas Sydenham is an example); exciting, efficient, or immediate causes, related to the more local environment or the experiences of the diseased person, and more congruent with the "non-naturals" (diet, emotion, exposure to weather, injury, poison or other more specific agents of disease); predisposing causes, which could overlap with the previous category, but also involved the individual's life or heredity that might render him or her unusually liable to a given disease; and proximate causes, which came closest to defining the disease stage or process occurring in the diseased body. See Margaret Pelling, "Contagion, Germ Theory/Specificity", W.F. Bynum and Roy Porter, *Companion Encyclopedia of the History of Medicine*, Vol. 1 (London/New York:

Specific or determinant causes, both contagious and non-contagious, were supposed to act in an evident way, always producing the same effects.[65] In the non-contagious group, Merizalde gathered a wide variety of causes: causes originated in the *functions* of nutrition, excretion, motion and perception (e.g. eating pestilential meat, rupture of the bladder by urine retention and dislocations), causes *applied* to the body (such as poison from snake bites), and those *conducted by air* (like animal and plant emanations that cause typhus and intermittent fevers respectively).[66] Qualities of the *atmosphere* like temperature and electricity, and *parasites* like tapeworms, were also grouped among the specific non-contagious causes.[67]

The specific contagious causes, on the other hand, referred to invisible elements that were transferred from sick bodies to healthy people by fluids and transpiration. They had the capacity to replicate and multiply *ad infinitum* in the presence of a suitable host, modifying susceptibility to further attacks in the process.[68]

In Merizalde's account of the specific causes, the environment played an important role: in the non-contagious group, air was labeled the vehicle of the emanations or miasmas and plant matter was causally associated with their production. In the contagious group, heat would either favor or destroy contagions whereas humidity, lack of light and vegetable emanations could foster their

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Routledge, 1997), p. 312. Christopher Hamlin has in fact talked of the "predispositionist-oriented thinking" in early nineteenth-century British medicine, which in general recognized two main divisions of causes of disease: the proximate causes and the remote causes. The latter were further distinguished into predisposing causes (the general state of the individual's constitution) and exciting or occasional causes (specific events that set off the disease, be they contagion, miasmas, or something else). This division was seen as troublesome and sometimes arbitrary, and some agreed that with sufficient predisposition, an exciting cause was not necessary; likewise, it was often held that without predisposition no amount of exposure to an exciting cause would lead to the disease: "in other words a so-called predisposing cause could be more important in determining whether one got the disease than an exciting cause." See Christopher Hamlin, "Predisposing Causes and Public Health in Early Nineteenth-Century Medical Thought", *The Society for the History of Medicine*, 1992, p. 44, 51, 54-55. As I will show, these schemes differ substantially from early nineteenth-century French and Colombian medicine. While in Britain miasmas and contagia were included as occasional causes, they were considered specific causes in French and Colombian medicine.

[65] Merizalde, *Elementos de patología*, p. 12.

[66] Animal vapors from overcrowded prisons and hospitals would cause typhus; vapors resulting from the exhumation of corpses may have caused malignant fevers, while those from latrines caused asphyxiation. Vapors derived from animal putrefaction could cause asphyxia. Plant vapors like the smell of certain flowers produced headache, vomiting and syncope, whereas miasmas from plant putrefaction produced intermittent fevers in swampy places. See Merizalde, *Elementos de patología*, p. 13.

[67] Merizalde, *Elementos de patología*, pp. 12-15.

[68] Typhus appears in Chomel's and Merizalde's books as a non-contagious and contagious disease, that is, caused by animal vapors and contagious at the same time. See Chomel, *Élémens de pathologie*, pp. 45, 53 and Merizalde, *Elementos de patología*, pp. 13, 15.

transmission.[69] For Merizalde, intermittent fevers were environmentally determined whereas contagious diseases like yellow fever and smallpox were not.

In contrast to the evident action of specific causes, predisposing and occasional causes acted in a more uncertain or obscure way. Predisposing causes gradually predisposed to particular diseases, whereas occasional causes acted as triggers for several diseases.[70] Occasional causes were thought to be less relevant than predisposing ones given their lack of specificity: one occasional cause could provoke the invasion of all diseases, and the same disease could be raised by many occasional causes. Cold, for example, could be the specific cause of rheumatism, but could also be an occasional cause of many other diseases.[71]

With regards to the predisposing causes, Merizalde stated that the majority of diseases, particularly internal ones, occurred without specific causes "if predisposing causes were influencing them". The Colombian doctor divided the predisposing causes into two groups, the individual predisposing causes and the general predisposing, always following Chomel's pathology. The individual predisposing causes derived both from individual conditions such as heredity, age, temperament (bilious, lymphatic, nervous and mixed temperament), constitution (strong or weak), profession, wealth and state of health; and from individual attitudes related to the functions of nutrition, excretion, motion and perception. All of the individual predisposing causes could produce diseases by themselves and also favor or counteract with the other group, the general predisposing causes.[72]

The general predisposing group of causes were those that affected more than one individual, that is, a population. They were mainly found in the air and places. Air, said Merizalde, had a great influence on the body. In order to predispose one to a disease it had to act for a long period of time; different mixtures of dryness, coldness, heat and humidity of the air thus had the power to produce several diseases if associated with particular individual and occasional causes. For example, dry and

[69] Merizalde *Elementos de patología*, p. 16.
[70] Ibid., pp. 12, 17.
[71] Occasional causes varied in nature. They could be a sudden change of temperature, bloodletting or strong emotions. Ibid., p. 27.
[72] Ibid., pp. 17; 19; 21-23, 27-29, 32.

cold air would predispose to inflammatory diseases and bleeding, whereas cold and humid air would produce scurvy and rheumatism.[73]

Change in the atmosphere during the succession of seasons also acted as a general predisposing cause, giving a particular character and course to diseases. There were two identifiable patterns: in Europe, according to Chomel's text, existed diseases of spring and the surrounding months which had an inflammatory character, lasted for short periods, responded well to medicines yet tended to repeat; and diseases of autumn and the surrounding months which had a bilious or mucous character, lasted longer, and tended to resist therapeutic measures.[74] As for Colombia, Merizalde stated that seasonal disease patterns followed the only two seasons of the Americas, summer and winter (according to the amount of rain), mirroring their European counterparts respectively.[75]

In addition to the influence of the air and atmospheric change which followed the seasons, locale played an important role in the theory of general predisposing causes. The French author, Chomel, had differentiated the diseases of the northern cold regions, where inflammatory disease predominated, from those of the warmer southern regions where tetanus, yellow fever and "many other" illnesses practically unknown to the north were dominant.[76] Merizalde's lectures, which had closely followed the French physician's textbook, entirely omitted the above distinctions. Instead, Merizalde narrowed down his analysis to South America and introduced another division that fitted the Colombian context: the northern regions of the coastal warmer lands by the Caribbean, with tetanus and yellow fever, contrasting with the southern cooler regions where inflammatory diseases were common. Even though Merizalde inverted the European reference, he maintained the association between temperature and disease.[77]

In Merizalde's scheme, locale also referred to the division between towns and country. While certain diseases were more prevalent amongst rural populations,

[73] Ibid., pp. 17-18.
[74] Chomel, *Élémens de pathologie*, p. 62; Merizalde, *Elementos de patología*, p. 18.
[75] Merizalde, *Elementos de patología*, p. 18.
[76] Chomel, *Élémens de pathologie*, p. 64.
[77] Merizalde, *Elementos de patología*, p. 19.

diseases such as scurvy, dysentery, and typhus were clearly produced in the crowded hospitals and prisons of Colombian cities.[78]

According to Merizalde, there were also some conditions that protected against diseases. He invoked a condition called vital force that seemed to be in constant conflict with the agents that surrounded people. This force would not only take away destructive things from food but would also diminish the power of subtle poisons. Vital force, and most importantly its reinforcement by habit, would explain why some people were protected from contagious disease. Thus, for example, locals from yellow fever endemic areas such as Veracruz (Mexico), Habana (Cuba), and Cartagena (Colombia), were preserved, while foreigners fell gravely ill; this same theory would account for the fact that physicians in hospitals were not affected by typhus.[79] Individuality played an important role, however, in the level of resistance. There might have been individuals whose predisposition prevented them from ever suffering the effects of contagious diseases.[80]

The above description of the Hippocratic environmental causality of disease was put into practice by Colombian doctors of the Faculty of Medicine during an epidemic in the capital city, Santa Fe de Bogotá, in 1830. The Faculty of Medicine, for whom Merizalde adapted the French texts, held the role of advising the government and the public on health issues. The 1826 Educational Plan stated that the Faculty should propose to the health boards of the cities the most appropriate health measures "according to the climate and circumstances of the country" and also advise people on how to protect themselves from the "most common or typical Colombian diseases."[81] The 1830 fever epidemic in Bogotá provided members of the Faculty of Medicine, including Merizalde, with the opportunity to test their theory and also to accomplish their role as advisors of public health.

Several members of the Faculty participated in the redaction of the report on the epidemic and the hygienic recommendations for the public.[82] The report began with

[78] Merizalde, *Elementos de patología*, p. 19.
[79] Ibid., p. 32.
[80] Ibid., p. 33.
[81] Colombia, Secretaría del Estado y del Interior, "Decreto 720", p. 649.
[82] Facultad de Medicina de Bogotá, *Epidemia reinante en Bogotá, a fin de 1830; y preceptos de higiene publica* (Bogotá: Impr. de B. Espinosa por José Ayarza, 1831). According to Pedro María Ibañez, the authors of this report were Professors Manuel M. Quijano (1782-1838), José Félix

a brief description of the city of Bogotá including the quality of its winds and waters, following the Hippocratic imperative. But it also introduced geographical and meteorological measurements for the city such as the latitude, altitude and average temperature.[83] It is very important to note that interest in meteorological measurements in the Colombian medical tradition was not new. There is some evidence suggesting that the study of the meteorology of Bogotá (and other places where students practiced) was part of the curriculum of medical studies around the turn of the nineteenth century when Colombia was still under Spanish rule.[84]

The application of instrument-based meteorology to environmental variables in medicine, a practice known as medical meteorology, had developed in Europe during the seventeenth and eighteenth centuries. Historians have pointed out that rather than eclipsing the classical environmental medicine of the Hippocratic tradition, medical meteorology gave renewed vigor to the quest for correlations between disease outbreaks and environmental conditions. However, historians have also argued that medical meteorology did not modify the explanations of disease causation.[85]

This also seems to have been the case in Bogotá at least until the 1830s, when the Faculty's report on the epidemic was written. Although the authors included barometric data and thermometric averages, this information did not modify the fact that, as far as they were concerned, the interaction between the specific and general predisposing causes (the atmosphere and the specificities of a given locale) was responsible for the epidemic. This mirrored the similar irrelevance of the published chemical composition of the air invoked by physicians (oxygen, *azoote* (nitrogen), and carbonic acid in proportions 21, 78 and 1). Again, they interpreted those variables within the Hippocratic framework, noting that diseases would occur if any of these gases dominated over the others. Indeed, some of the gases were treated as miasmas, as we will see later. But before detailing how changes in the air had

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Merizalde, Benito Osorio, Pedro Herrera and Vicente Lombana (1809-1880). See Ibáñez, *Memorias para la historia,* p. 64.

[83] They were 4.5 degrees of boreal latitude; 1,500 Toesas (2.715 meters) of altitude and 12-14 Réaumur degrees (15-17.5 Celsius) of average temperature. Facultad de Medicina de Bogotá, *Epidemia reinante en Bogotá*, p. 3.

[84] Ibáñez, *Memorias para la historia*, p. 43.

[85] Hannaway, "Environment and Miasmata", pp. 297-299.

produced the 1830 epidemic, the authors offered an account of the normal atmospheric pattern of Bogotá and of its associated diseases.[86]

They claimed that air in Bogotá maintained its right proportions and hence its healthy character as long as the seasonal patterns were regular. These patterns were driven by the differing amount of rain during the solstices and equinoxes, which are associated with a lower and higher rainfall respectively. Within this regularity, Bogotá remained in an apparent permanent, healthy, spring-like season. This explained why, in Bogotá, seasonal and chronic diseases were benign while phthisis (tuberculosis) and *hectic fever* were rare (diseases such as plague, *vomito prieto*, and yellow fever never reached the high plateau where Bogotá is located). But if any of the seasons were prolonged, breaking the permanently benign health state of the atmosphere, diseases were likely to occur.[87]

According to the faculty members, the 1830 epidemic started in September after months of an extended severe winter caused by southeastern and northern winds, exposing people to an excessively cold and humid air which affected breath and damaged the mucous membranes, ultimately producing catarrhal fevers. During the following months, these catarrhal fevers spread by contagion and were then transformed into malignant ataxic-adynamic fevers under the influence of the city. The faculty claimed that winds that hit the mountains along the eastern side of Bogotá exposed people to an excessive column of "*azootic* miasmas" produced in the city; these gases or miasmas, when combined with hydrogen, altered the already damaged constitution of the atmosphere, thus turning the benign catarrhal fevers into true ataxic-adynamic fevers.[88]

A mixture of specific causes originating in the city and individual predisposing causes explained this second alteration in the air. Specific causes such as animal vapors or miasmas from the putrefaction in sewage, exhumation of corpses, slaughter of pigs, infection of water, and fermentation in filthy places, produced large amounts of hydrogen, carbon and *azoote* which "infected" the air, severely altering its quality.

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[86] Facultad de Medicina de Bogotá, *Epidemia reinante en Bogotá*, p. 17.
[87] Sporadic diseases such as smallpox, measles, mumps, and exanthematic, ataxic and adynamic fevers may occur along with seasonal diseases. See Facultad de Medicina de Bogotá, *Epidemia reinante en Bogotá*, pp. 4-5; 16.
[88] Facultad de Medicina de Bogotá, *Epidemia reinante en Bogotá*, pp. 17-19.

Individual predisposing causes such as abuse of alcohol and food had also contributed to the development of the epidemic.[89]

As the report on the 1830 epidemic of Bogotá shows, Colombian physicians of the first half of the nineteenth century introduced contemporary meteorological and chemical notions to their studies in order to understand epidemics. However, these data were incorporated within the dominant conceptual scheme for explaining epidemics as described above: the neo-Hippocratism of French pathological and hygiene texts, in which the environment (seasonal changes and local conditions), that is, the general predisposing causes plus some specific ones such as miasmas, accounted fully for the development of the epidemic.

Neo-Hippocratism continued to be used for several decades, from the 1830s until the 1870s, particularly when describing health conditions in the capital city. For example, by the mid-nineteenth century, Colombian physicians utilized the Hippocratic notion of individual constitutions (strong or weak) to qualify the general state of health of a particular area, through what they called medical constitution. The benign or malign constitution of an area meant that diseases tended to result in either a full recovery or death respectively.[90] For example, the medical constitution of Bogotá in 1852 was evaluated by physicians as "good", considering that only mild diseases such as eruptions of the skin, bronchitis and pneumonias were prevalent in the city.[91] The descriptive role of medical constitution was not limited to the above, however; it was also used to qualify seasonal diseases, as Dr. Antonio Vargas Vega did when he affirmed that during rainy periods and cold winds, bronchial, lung and digestive illness always occurred with a catarrhal character.[92] Physicians in Bogotá also invoked the atmospheric constitution, which referred to a combination of weather and climate that characterized a specific period in time.[93] They claimed, for example, that changes in the atmospheric constitution were the most likely

[89] Ibid., pp. 17-19; 32.
[90] *Lanceta*. "Personas notables que han muerto en esta ciudad en el intervalo de los dos primeros Nos. de la Lanceta", *La Lanceta*, Año 1, No. 2, 16 de mayo, 1852, p. 9.
[91] *Lanceta*, "Hechos diversos", *La Lanceta*, Año 1, No. 5, 20 de septiembre, 1852, p. 18.
[92] Antonio Vargas Vega, "Constitución médica. La causa de decadencia física en la niñez", *La Lanceta*, Año 1, No. 6, 26 de octubre, 1852, p. 21.
[93] For a description of this notion see Hannaway, "Environment and Miasmata", p. 294.

etiological candidate to explain the epidemic of diphtheria in 1873 that killed 16 children.[94]

Neo-Hippocratism, *castas* and diseases

One aspect closely related to this environmental determinism of diseases in the first half of the nineteenth century in Colombia was the use of the same medical notions to explain social and racial differences inherited from the colonial period. The authors of the 1830 epidemic report distinguished between the Indians of the *sabana* of Bogotá (rural areas surrounding the city) and "families who have long lived in the city", that is, Spaniards who had been arriving from Spain since the sixteenth century and their descendants, probably including *mestizos*. They used this differentiation to characterize their similar acclimatization to the diseases of the *sabana*, but also to highlight that natives were morally docile.

Merizalde and his fellows in the Faculty of Medicine affirmed that indigenous people were healthy, enjoyed a long-lasting life, and were rarely affected by seasonal and chronic diseases. The faculty implicitly assumed that these people's relative healthiness was due to a natural acclimatization to their environment.[95] This can be deduced by the way that the authors characterized the other group, "families who have long lived in the city": these families were "already very well acclimatized" and kept good habits, and so their responsiveness to seasonal and chronic disease was similar to that of the indigenous people, that is, they were rarely affected by them.[96] Natives' healthiness and relative resistance to certain diseases was, according to the authors, accompanied by another virtue: the natives' moral docility. While their acclimatization to the territory had made them resistant to seasonal and chronic

[94] J.M. Buendia, [informe "epidemia de angina diftérica"], *Revista Médica*, Serie I, No. 2, 2 de agosto, 1873, p. 10.
[95] As Harrison has pointed out, the doctrine governing the question of which climate exerted a powerful effect upon human health and physical characteristics, as well as the adaptability of physical constitutions, derived from popular and Hippocratic notions of environmental determinism that reached a high stage of sophistication in the eighteenth century in the writings of Montesquieu and Buffon among others. See Harrison, "'The Tender Frame of Man', p. 74., op. cit, footnote 1. For an overview of theories of human acclimatization in the English-speaking countries during the nineteenth century see David N. Livingston, "Human Acclimatization: Perspectives on a Contested Field of Inquiry in Science, Medicine, and Geography", *History of Science*, 25, 1987, 359-394.
[96] Ibid., p. 4.

diseases, physicians affirmed that natives' phlegmatic character accounted for their docility, generosity and hospitality, which went very well with their "conscious subjugation to law" and their tendency to maintain social pacts.[97]

This moral weakness, the authors explained, was not only due to natives' dominant phlegmatic temperament. According to Merizalde, the hygiene treatise by Tourtelle stated that inhabitants of high, open places, exposed to winds, with abundant waters, fertile lands and mild season (a description that Colombian physicians matched with the *sabana* of Bogotá), were described as without courage and more docile; the fertility of the land, according to the same text, saved them from continuous and arduous work and this condition, combined with mild seasons that exposed them to little temperature variation, meant that they would not suffer from frequent and violent shocks in the epigastrium and brain which disposed to strong and violent passions.[98] As such, these features of high, open places like the *sabana* of Bogotá would reinforce the moral weakness of the natives' phlegmatic temperament. But it could also soften the character of the rest of the population, as Merizalde seems to imply here:

> It is this special physical influence over the moral faculties that one has to blame for the mild, gentle and human character of inhabitants of the central provinces [the *altiplano*] of former Nueva Granada, in particular those of Santafe de Bogotá, who during the worst days of the 20th of July revolution and the fateful anarchic days in which its inhabitants have been involved, have verified this observation already made since the period of the divine Hippocrates. [99]

Besides the fact that Merizalde medically justified the inferiority of natives, blaming both the environment and their temperament, he did not contest Tourtelle's general vision of the inferiority of men in the tropics. Merizalde translated, without any comment, Tourtelle's idea that the closer one was to the equator and poles, the more "degraded people" one would find; that Africa and America's native people

[97] Ibid., p. 5. In Merizalde's extracts of Tourtelle's book phlegmatic individuals were described as having slow functions, weak pulse and breath, little appetite and difficult digestion, but also of weak character that tended to laziness and laxity. See Merizalde, *Epitome de los elementos de higiene*, p. 45.
[98] Merizalde, *Epitome de los elementos de higiene*, p. 151.
[99] Ibid., p. 151.

had less developed spiritual/mental capacities; and that those places were the "scourge of human intelligence." [100]

Similar arguments about the physical and moral differences between the natives and other groups due to climatic conditions are also found in the work of the botanist and astronomer Francisco José de Caldas (1771-1816). Caldas was a self-educated scientist who engaged in the Spanish botanical expedition in 1803 and became director of the Astronomical Observatory in Bogotá in 1805. Caldas founded the first scientific journal of the country, the *Semanario del Nuevo Reino de Granada* (1808-1810), which secured him recognition for the rest of the century. In his famous 1808 paper on the influence of climate on organized beings, which he based on Buffon's *Natural History*, Caldas stressed that heat and cold were responsible for the differences between all animals, not only in color and size, but also in strength and courage.[101] Man, Caldas continued, had also suffered the influence of different latitudes as differences in color revealed around the world. As for the peculiar color of natives of the New World, which he described as reddish and copper, Caldas blamed other geographical variables such as the Andes mountains, the rainforest, the "vigorous vegetation," the "huge rivers", lakes, seas, the abundant rain, winds, electricity and clouds. "It is true," noted Caldas, "that the color of natives of the Nueva Granada is copper but it goes up and down, it darkens or becomes lighter with regard to the level, heat, climate, occupation, ways of living: natives of the south coasts are darker than those of the slopes of *Cordilleras*."[102] Caldas also extended this argument to explain moral differences between people. Natives of the Pacific coasts, where temperatures typically varied between 18 and 30 degrees Reaumeur (22.5 and 37.5 degrees Celsius) were not only of tanned skin, medium height, and dark hair, but also had an intrepid character particularly suited to fishing and hunting. In contrast, natives and other *castas* (i.e. Indians and *mestizos*) who lived in the highlands or *cordilleras* were whiter, sweeter in character and had calmer emotions; like Dr. José Félix Merizalde, Caldas affirmed that natives of the *cordilleras* had

[100] Ibid., pp. 85-86.
[101] Caldas, "Del influjo del clima", pp. 88-9.
[102] Ibid., p. 92.

moderated customs and moral principles due to the benign temperature and soft air. [103]

Caldas's and Merizalde's vision of the moral weakness, docility and inferiority dependent on climate and individual temperament corresponded to the colonial social order that was based on the differentiation between the "white" Spanish and the other groups or *castas*. While the colonial system was meant to be protective of Indians, it was based on the perception of their moral weakness and inevitable corruptibility upon contact with "whites" and especially *mestizos*, the latter becoming the predominant social presence and threat to the order by the end of the colonial period. The *castas* could not participate in institutions such as education and local councils. According to the law only nobles [some Indians included] and "whites" were entitled to enter university, and there were frequent disputes with candidates who were rejected because they were "stained by the blood of the soil, not only them but also their parents or their grandparents." [104]

Although the legal base for the discrimination of Indians was abolished with the establishment of the republic, based on the principles of the Enlightenment, in practice they, as well as *mestizos*, remained lower in the social scale and were valued as inferior people, although the specific grounds for that inferiority were under discussion.

The independence movement was led by the "white" established elites or creoles, who found, in the dynastic crisis of the Bourbons (caused by the Napoleonic invasion of Spain), the opportunity to break the monopolistic control of international commerce by the Spanish crown, and also the opportunity to liberate themselves from crown control, while guaranteeing the privileges earned during *de facto* autonomy of centuries of settlement. As such, once the creoles thoroughly defeated the Spanish army in 1819 and funded the new independent republic on the basis of the French revolutionary principles of "liberty and equality" for the citizens, they did little to transform the social and economic *status quo* that kept them in the elite position in the first place.

The careers of Caldas and Merizalde illustrate this point. Francisco José de Caldas, son of a Spanish military officer, who entered the local webs of power by

[103] Ibid., pp. 96-98.
[104] Renan Silva, *Los ilustrados de la Nueva Granada, 1760-1808* (Medellin, EAFIT, 2002), p. 37.

marrying a woman of an old prestigious family in Popayan, was part of the creole elite that participated in the independence movement,[105] and his prominence throughout the nineteenth century remained considerable, not least because of his martyrdom to the patriotic cause in 1816.

Dr. Jose Felix Merizalde was the son of a Spanish medical professor who had been sent on a mission to Nueva Granada to search for ways of exporting quinine.[106] Merizalde studied medicine in the *Colegio Mayor del Rosario* during the colonial period. Like Caldas, he participated in the independence movement on the side of the rebels, but in his case it was after independence that he gained prominence. Still, Merizalde's and Caldas's ideas with regards to the health and character of Colombia's inhabitants were very similar, and clearly associated with the inherited colonial vision of the inferiority of the *castas*.

Throughout the nineteenth century, physicians and other professionals in natural sciences inherited Calda's and Merizalde's visions of the inferiority of the old *castas*, later called *races*, as well as their ideas of the climatic determinism of temperaments and diseases. It was a vision that framed inferiority in neo-Hippocratic and scientific terms.[107]

Medical geography in the mid-nineteenth century

[105] Ibidem.
[106] Ibañez, *Memorias para la historia*, p. 104.
[107] Frank Safford has described the continuities of the Colombian elites' attitudes towards Indians from the late colonial period to the nineteenth century and the rhetorical changes that came with the political needs of the republican era in "Race, Integration, and Progress: Elite Attitudes and the Indian in Colombia, 1750-1870", *Hispanic American Historical Review*, 71: 1, 1991, pp. 1-33. Safford does not differentiate, however, between the colonial understanding of *castas* and the republican idea of *races*. I will show in the next section that there were also continuities between the colonial descriptions of the physical constitution of *castas* and the republican *races* in medical literature. But, considering that the use of the notion *races* by Colombian physicians of the second half of the nineteenth century carried ideas that were not present in the colonial understanding of *castas* (such as the question of the unity or diversity of the human race) and vice versa (the *castas* refered mainly to purity of blood, not to mention the other social and political implications that were not present in the idea of *races*), we should treat them differently. Carlos López Beltrán is among the few scholars that has tried to problematize the link between the classificatory system of *castas*, based on the purity of blood, and that of the Hippocratic corpus in Spanish America (based on sources from Mexico and Peru) during the colonial period in: "Hippocratic Bodies. Temperament and Castas in Spanish America (1570-1820), *Journal of Spanish Cultural Studies*, 8, 2, 2007, pp. 253-289.

One characteristic of the neo-Hippocratic version of disease during the first half of the nineteenth century was the distinction between diseases of different latitudes, that is, between those of the cooler Europe and those of the warmer tropical areas. But for Colombia, distinctions according to altitude were more relevant, as its tropical location determined that temperature varied according primarily to altitude rather than to latitude. As I showed above, from the first decades of the nineteenth century, figures like Caldas and Merizalde had introduced comparisons of the physical and moral characteristics between people of the highlands and the lowlands. Even Caldas stressed that it was important for medical practitioners to study the pressure and air density experienced by inhabitants of the *cordilleras* and the coasts or *llanos* (lowland plains).[108] However, it was not until the mid-nineteenth century that physicians developed more precise explanations for the ways that altitude and temperature were related to disease, under the influence of the field of medical geography, which would transform the causal framework of specific, predisposing and occasional causes of disease. For Colombian physicians, medical geography - or medical topography - referred to the study of the "local" pathologies, that is, those that were determined by the climate of each locality.[109]

Physicians expressed an intention to consolidate a classification of diseases according to altitude as those of warm, temperate and cold land. Along with the study of Hippocratic themes such as nutrition, perceptions, temperaments, airs, seasons, miasmas and contagions, courses of hygiene in the Colombian medical schools from 1845 onwards included an analysis of the "different climates and temperatures of *Nueva Granada*" and the effects of the "transit from one place to another of different temperature and humidity."[110] Students had to learn the boundaries between *tierras calientes*, *templadas*, and *frias* (warm, temperate and cold lands), and the diseases to which inhabitants of each of these regions were exposed, considering variations caused by humidity, rain and the seasons.[111] This

[108] Caldas, "Del influjo del clima", pp. 96, 106-109.
[109] Scholars have shown that medical topography works in Europe focused on the medical conditions of specific localities whereas medical geography extended those preoccupations to a global level.
[110] Jorge Vargas, *Programa para la enseñanza de la hijiene en las universidades de la República, formado por el Dr. Jorge Vargas, i aprobado por la dirección jeneral de instrucción pública*. Bogotá, [1845], pp. 18-21. The date of the document is taken from the Biblioteca Nacional de Colombia catalogue.
[111] Vargas, *Programa para la enseñanza de la hijiene,* pp. 18-20.

focus must have been influenced by people's increasing acquaintance with the differences between the regions, furthered by the introduction of steamboats to the Magdalena River in 1847. Steamboats facilitated and accelerated the communications between the Andes and the Caribbean coast and were strongly associated with the increase in economic activity in the Magdalena valley, due to the production of agricultural goods for export from the mid-1840s. Despite the existing interest in studying the diseases of those three different regions, medical geographical works were scarce; those which were published concentrated mainly on the diseases of the low warm lands.

In contrast to the medical generation of the first half of the nineteenth century, who were trained in local medical schools, the mid-century generation of physicians who initiated the study of medical geography belonged to the first tide of doctors who traveled to Paris to complete their medical training and began to return around the late 1840s. The most influential person campaigning for work in medical geography was Dr. Antonio Vargas Reyes (1816-1873). According to his biographers, Vargas Reyes obtained a medical degree in Bogotá before attending the Paris medical school between 1842 and 1846. On his return, Vargas Reyes created the first medical journals ever to appear in the country, *La Lanceta* (1852) and *Gaceta Médica* (1864-1867) from which he campaigned in favor of the study of local pathologies. In contrast to the acquiescence to French neo-Hippocratism when explaining pathologies in the country, shown by physicians of the first half of the nineteenth century like José Félix Merizalde, Antonio Vargas Reyes argued that "it would be useless to be good at the art of healing the diseases of Europeans, if we do not know ours and how to combat them", hence defending the originality of medical research conducted in the localities where it originated.[112]

[112] Antonio Vargas Reyes, 'Discurso preliminar', Trabajos Científicos del Doctor Antonio Vargas Reyes recopilados en obsequio de la humanidad doliente i de la juventud estudiosa de Colombia. Tomo Segundo (Bogotá: Imprenta la Nación, 1862), p. VIII. John Harley Warner has described a similar skeptical attitude towards French medicine from American antebellum physicians, but in the medical realm of therapeutics. According to Warner, American physicians trained in the Paris medical school between 1820 and 1840 rejected the Parisian therapeutic practices. Following a "specificity" principle, says Warner, American physicians judged the French therapeutic practices irrelevant to American patients since the constitutions of Americans and of American diseases were inherently more energetic than those of Europeans, thus reclaiming a specific treatment. Even though American physicians rejected the French therapeutic practices, they embraced the epistemology in which that practice was based, that is, in the empirical experience at the bedside. This "selective transport" of French medical knowledge, explains Warner, was due to the division that American physicians held

In the early 1850s, Dr. Miguel Uribe Angel (1822-1904), from Medellín, also questioned the universal application of medical theories, arguing that humans did not have the same *organization* (that is, organic arrangement) everywhere, especially as diseases were specific to their localities. He wrote that physicians were in an analogous position to that of lawyers. Just as lawyers needed to adjust their knowledge to the social organization in which they practiced, physicians needed to know "the influence of localities, customs, races, and the specific illnesses of the people".[113] Physicians like Antonio Vargas Reyes and Domingo Esguerra Ortiz extended this argument further, seeing in the study of local pathologies the opportunity to contribute to universal medical knowledge, to challenge the European knowledge, and thus highlight the advantages of having a direct experience of the diseases *in* the places where they occur.[114] According to Vargas Reyes, "the day that physicians of Colombia communicate the result of their experience and study our diseases in the great book of nature, that day we will have our medical geography and we will not be forced to adapt our practice to the books that come from foreign countries." [115]

In order to justify that diseases were causally attached to localities, Vargas Reyes looked beyond the Hippocratic analysis of air, waters and places. He mixed the old notions of the effect of climate on living entities with French transformist theories of the distribution of species, developed since the eighteenth century's natural history. During either his years as a student in Bogotá, where Lamarck and Buffon were part of the curriculum of Natural Sciences of the *Universidad Central* of Bogotá,[116] or his Paris years, Vargas Reyes became acquainted with the transformist argument, which made the claim that living forms were gradually transmuted by the influence of the environment and then transmitted these acquired characteristics to their descendants. Vargas Reyes allied with the monogenists, who supported the idea that human races

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between the universal and specific parts of medicine, but also (and most importantly) to the needs of the medical profession of that period. See John Harley Warner, "The Selective Transport of Medical Knowledge: Antebellum American Physicians and Parisian Medical Therapeutics", *Bulletin for the History of Medicine*, 59, 1985, pp. 213-231.

[113] Manuel Uribe Angel, "Medicina-Fibre de Cauca i sus variedades", *El Pueblo* (Medellín), 42, 5 de junio, 1856, p. 170.

[114] *Gaceta Médica*, "Prospecto", *Gaceta Médica de Colombia*, No. 1, 6 Julio, 1864, p. 1; Domingo Esguerra Ortiz, *Memoria sobre la fiebre del Magdalena* (Santa Ana: Imprenta de D. Díaz, 1872), p. VI.

[115] *Gaceta Médica*, "Prospecto."

[116] Colombia, Secretaría del Estado y del Interior. "Decreto 720", p. 628.

came from one common "type" but took on physiological forms corresponding to the climates of the places where they settled.[117] Vargas Reyes argued that climate, and also civilization, were the active causes of these changes. "Man," he stated, "in his diverse varieties, has been trying to put himself in harmony with the environment he inhabits, his organs *adapt* to nature." [118] Immediately, Vargas Reyes linked this transformist mechanism to acclimatization: "but those changes only occur gradually until acclimatization makes him immune to deleterious influences of each locality." [119] He believed that present inhabitants of Africa or America were very different from those who originally settled there; many victims had succumbed to disease and many years had passed before they were able to inherit the "hereditary transmission that makes them refractory to the destructive influences of several temperatures" from their ancestors. [120]

Climate and this kind of hereditary transmission were thus a critical part of the explanation for differences in human resistance to diseases between different localities. In other words, Vargas Reyes replaced Dr. Merizalde's acclimatization theory, based on a "vital force" and "habit" that would have made locals resistant to diseases, with another acclimatization theory, based on the biological Lamarckian transformist mechanism of *amoldamiento* (adjustment) of organs by climate and heredity. But Vargas Reyes also used this tranformist mechanism to explain why climatic phenomena were important in developing diseases. Indeed, he wondered if "those causes that have changed man's aspect by changing his organization are not the same that originated his diseases".[121] The vigor and weakness of man's organs, established by years of acclimatization to heat, electricity, winds, waters and food, determined differences in the ways that he had suffered and healed.

The transformist argument for the justification of differences in diseases of different localities was reinforced by pre-existing ideas in Colombia about the influence of climate on plants, in particular the work of Francisco José de Caldas and

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[117] Polygenists believed that various races were uniquely adapted to particular climatic zones whereas monogenists claimed that races were the environmental products of climate conditions. The latter were, in general, pro-acclimatationists in European discussions about acclimatization. See Livingston, "Human Acclimatization", pp. 362, 386.
[118] Vargas Reyes, "Discurso preliminar", p. III. The italics are mine.
[119] Ibid., pp. III-IV.
[120] Ibid., p. IV. The intalics are mine.
[121] Ibid., pp. III-IV.

the German naturalist and geographer Alexandre von Humboldt (1769-1859). As I showed in the first section of this chapter, Caldas had described how animals, including man, were physically determined by temperature and by climates. Caldas extended this argument to the variation of plants, following the work of Humboldt in plant geography.

Indeed, Caldas and Humboldt had met when the German naturalist visited Nueva Granada between 1801 and 1802. Before leaving South American lands, Humboldt wrote a paper titled "Ideas for a Geography of Plants" that Caldas was aware of before it was published in French (1805) and German (1807). In fact, the first Spanish version of Humboldt's essay appeared in Caldas's journal, *Semanario del Nuevo Reyno de Granada*, in 1808. In that essay, Humboldt defined the geography of plants as the study of the functional relationship between plants and climate, thus defining botanical regions of associated plants according to altitude and related climatic elements.[122] Caldas expressed similar ideas based on his own travels,[123] stressing, as Humboldt did, that in the tropics it was altitude and not latitude that determined the properties of plants.[124]

In the late 1850s and 1860s, Antonio Vargas Reyes linked Humboldt's work with that of the Swiss botanist Augustin Pyrame de Candolle (1778-1841) on plant distribution, further attaching the transformist mechanism of acclimatization described above, in order to understand diseases:

> In the same way as the recent discoveries of Humboldt, Bompland, Prush, Brwon, gave de Candolle sufficient material for a geographic distribution of plants, which he believed originated from one common center, we find constant arguments that prove that different diseases that affect human species, far from having diverse origins, are alterations of the same type, but are modified by the influence of climate, by the [people's] way of living, etc.[125]

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[122] Alexandre de Humboldt et Aime Bonpland, *Essai sur la Gégraphie des plantes; accompagné d'un tableau physique des régions équinoxiales. Fondé sur des mesures exécutés, depuis le dixième degré de latitude australe, pendant les annés 1799, 1800, 1801, 1802 et 1803* (Paris: Levraut, Schoell et Compagnie, 1805), pp. 14-16.
[123] Appel, *Francisco José de Caldas*, p. 54.
[124] Caldas, "Del influjo del clima", pp. 89, 101, 104.
[125] Antonio Vargas Reyes, "Discusión sobre la fiebre amarilla", *Trabajos Científicos del Doctor Antonio Vargas Reyes recopilados en obsequio de la humanidad doliente i de la juventud estudiosa de Colombia*, Tomo Segundo (Bogotá, Imprenta de la Nación, 1862), p. 38.

As some scholars have shown, Humboldtian plant geography greatly influenced medical geography and hygiene in Europe.[126] In the *Essai de géographie médicale* (1843), the army physician Jean-Christian Boudin (1806-1867) stated that the laws of plant distribution could similarly be found in the distribution of diseases.[127] More influential in Colombian medical geography was the work of French hygienists, who had a less philosophical and more practical approach to the distribution of diseases. For example, the army physician Michel Levy expressed in his *Traité d'hygiène publique et privée* (1850) that the Humboldtian system of climates, based on isothermic lines that depended not only on variations of temperature by latitude but also on particular geographical accidents, was "not real." This "pure mental operation," according to Levy, gathered regions separated by enormous distances into the same climatic group without consulting their particular geography. For Levy, the question about climates, at least for medical purposes, could only be resolved in the climate of the localities.[128]

This localist emphasis in the approach to medical geography by the French hygienist was similar to the way in which Colombian physicians understood medical geography. Colombian doctors were more interested in the "local" conditions for the production of diseases than the search for general laws for their geographical distribution. In fact, almost all of the foreign references they used in their works on this subject were French treatises on hygiene, rather than pure medical geography works.[129] Besides, as I mentioned in the last section, they stressed the advantages of first-hand experience in the places where disease occurred, seeing an opportunity, via this direct empirical experience, to challenge conclusions made in the European contexts. This localist and empirical approach to medical geography could explain why Colombian physicians, unlike those in many other areas, thought of medical

[126] Rupke, "Humboldtian Medicine" and Sandra Caponi, "On acclimatization: Boudin and medical geography", *Historia, Ciências Saúde-Manguinhos*, Rio de Janeiro, 14, 1, en.-mar., 2007, pp. 13-30.
[127] J. Charles. M. Boudin, *Essai de géographie médicale* (Paris: Germer-Baillière/Ladé, 1843), pp. 5-9.
[128] Michel Levy, *Traité D'Hygiène Publique et Privée* T. I., (Paris: J.-B. Baillière, Second edn., 1850), p. 525.
[129] While Antonio Vargas Reyes rarely mentioned any medical geography work, another Colombian doctor, Domingo Esguerra Ortiz, extensively cited Levy's *Traité d'Higiène Publique et Privée* (1862) in his *Memoria sobre la fiebre del Magdalena*, op. cit., footnote 63.

topography and medical geography as synonymous.[130] Indeed, Vargas Reyes indicated that medical geography would make the use of knowledge from abroad unnecessary, and pointed out that the "topographic element" could be the missing element in medicine that would allow it to wander between systems.[131]

According to this emphasis on the locality, Vargas Reyes expressed that everyone in the world suffered from the disease "generated in their own localities": in Africa, plague and scurvy; in Asia, cholera morbo; in America, yellow fever; in Europe, *escrofulas, raquitis, plica, sudor miliar*, malignant pustule. It was striking, Vargas Reyes said, that the diseases of South America were almost invariably the same: dropsy, skin diseases, rheumatism, scurvy, dysentery, intermittent and continuous fevers, and yellow fever.[132] With regards to Colombia, Vargas Reyes listed the most common diseases of several zones, though he did not specify which were native and which imported: in the Atlantic and Pacific coasts, yellow fever, dysentery, intermittent fevers, elephantiasis, *tisis*, and tetanus; in Socorro, in the northeast of Colombia, *coto*, cretinism, elephantiasis and ulcers; in the *llanos*, *tisis*, and pernicious fevers; and finally, in the Magdalena and Zulia River valleys, pernicious fevers.[133]

Although Antonio Vargas Reyes sought to convince Colombian physicians of the importance of medical geography studies in his medical journals, and even published some works on fevers of the warm lowlands, he scarcely considered diseases of the temperate regions and the highlands. However, his comments on the relationship between tuberculosis and intermittent fevers reveal the extent to which he was open to the argument of the differentiation of diseases according to altitude in his medical geographical perspective. Attacking Boudin's idea of the antagonism of intermittent

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[130] Some scholars have indicated that medical topography, which developed in eighteenth century Europe, focused on the medical conditions of specific localities, whereas medical geography, which developed throughout the nineteenth century, extended those preoccupations to a global level. See Hannaway, "Evironment and Miasmata", pp. 300-2 and Rupke, "Humboldtian Medicine", p. 293.

[131] "Nuestros Votos" [editorial], p. 21. John Harley Warner has shown that American medical reformers who were trained in France "selected" the French empiricism argument, that is, "its allegiance to empirical fact…to knowledge attained and verified by direct observation and analysis of nature" to uplift the profession who were calling for a demystification of the previous established "rationalistic" medical knowledge. See John Harley Warner, "Remembering Paris: Memory and the American Disciples of French Medicine in the Nineteenth Century", *Bulletin for the History of Medicine*, 65, 91 1991, pp. 313-314.

[132] Vargas Reyes, "Discurso preliminar", p. V.

[133] Ibidem.

fevers and tuberculosis, i.e. that they never happen simultaneously, Vargas Reyes claimed:

> Tuberculosis and intermittent fevers are extremely rare in the highlands where we are located [*sabana* of Bogotá] and they progressively increase as we descend towards the sea level. If it is the case that we are able to trace, with a thermometer in one hand and a barometer in the other, the exact scale of the maximum and minimum development of these two diseases, there is no doubt that the antagonism between tuberculosis and yellow fever is false.[134]

In contrast to Vargas Reyes, his nephew, another physician called Antonio Vargas Vega (1828-1902), showed far more interest in the physiological and pathological effects of the highlands. In his 1865 paper, "Studies of compared climatology-elevation of land", Vargas Vega expressed the intention to publish several papers on the "dominant pathological manifestations in the *cordillera* climates." [135] Vargas Vega shared his contemporaries' concerns about the need to know the local pathologies of the country, in the sense of medical topography or geography. Indeed, he planned to write about the physiological principles associated with living at high altitudes, the dominant pathological manifestations of the high altitudes in Colombia, the rules for the treatment of these diseases, the preventive methods "in order to avoid the weakening effects of climate" and, finally, "the curative influence of climates." [136] Although Vargas Vega only managed to publish one of the papers, it is enough to show how mid-century Colombian physicians modified the previous neo-Hippocratic scheme with regards to the effects of altitude on health. It also allows us to see the persistence throughout nineteenth-century medicine of the differentiation between "whites" and the old *castas*, now called *races*, in medical terms, reinforcing the differences among the inhabitants of the country.[137]

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[134] Vargas Reyes, Antonio. "Carta cuarta. Al señor doctor Eloi Ordóñez", *Gaceta Médica*, Serie I, No. 10, 2 de marzo, 1866, p. 37.
[135] Antonio Vargas Vega. "Estudios de climatología comparada-Elevación del suelo", *Gaceta Médica*, Serie I, No. 1, 1 de junio, 1865, pp. 1-2.
[136] Vargas Vega, "Estudios de climatología", pp. 1-2.
[137] According to the 1851 national census, Indians accounted for the 13% of the population, "withes" for the 17% and of mestizos for a significant 47.6%. The rest were blacks, and its descendants. See Frank Safford and Marco Palacios, *Colombia. Fragmented Land, Divided Society* (New York/Cambridge: Cambridge University Press, 2002), p. 261.

According to Vargas Vega, physicians who had investigated the process of breathing in the highlands had noticed the "relative weakness" of this function in the population of those places. Through the auscultation of healthy "whites", *mestizos* and Indians working in the fields of the *sabana*, physicians had found that in all groups, *inspirations* were few, chests were narrow, and the respiratory sound was deep and weak. Vargas Vega affirmed that, for these reasons, "white" men and *mestizos* could not stand constant work in the *sabana*, as their forces would be exhausted and their constitution would deteriorate within a short period of time. In Indians, these effects were mitigated by their particular physiognomy and physiology. Indeed, Vargas Vega affirmed that in contrast to "whites" and *mestizos* of the highlands, natives had physiological conditions that compensated for the limited, narrower and weaker breath detected among all inhabitants of the highlands. Natives had larger and more muscular chests than "whites" and *mestizos* – the average width of their chest was greater, and their arms and legs were shorter than those of the other two groups. This physiological aptitude, however, implied great difficulties in acclimatizing to the low lands and their higher atmospheric pressure.[138]

Vargas Vega further suggested that these different physiological effects of high altitudes on the *races* were causally linked to the social distribution of labor. The physiological advantages of the natives of the *sabana* could explain why they did the hard work farming the land while the "whites" were limited to sedentary life. Vargas Vega was inclined to believe this, as he saw similar labor distribution in places of similar geographic characteristics to those of the *sabana* such as Boyacá, Pasto and Quito. In support of this distribution of labor, Vargas Vega invoked the argument developed by physicians and naturalists in the first half of the nineteenth century concerning the docility of natives: their "degraded" condition that would make them easy for "whites" to subjugate. [139]

But despite Vargas Vega's reproduction of the vision of the moral and social inferiority of the natives using old and new medical terms, he also suggested that the division of labor may have had something to do with the distribution of land [property]. Whatever the cause, Vargas Vega affirmed that there was a "true geographical distribution of races inhabiting the equatorial region in relation to the

[138] Ibid, pp. 1-2.
[139] Ibid., p. 1.

lifestyle." "Blacks" and their descendants did the hard work in the lowlands, "whites" worked in climates of mild height, and Indians in the highlands.[140]

Apart from this discussion of the *racial* differences in altitudes with regards to breathing capacity, and thus the distribution of labor, Vargas Vega also explored some of the consequences of altitude for digestion and circulation in those groups. He claimed that slow digestion was typical of high altitudes. The constitution of natives, "who were naturally sober", was not affected by this trait even if they were malnourished. They were supposed to be able to live for long periods of time only on vegetables, and, in fact, a strong and nutritious food would negatively affect and weaken them. "White" men of the highlands, on the contrary, would find that a slow digestion caused by high altitudes would lead them to laziness, which, added to the lack of muscular energy and nervous stimulus, could make them intellectually adynamic.[141]

In the last part of his paper, Vargas Vega analyzed the effects of altitude upon circulation. He complained about the lack of "blood measurements", meaning that physicians could only deduce general physiological manifestations of high altitudes on circulation through the dominant pathology in those places. Thus, for example, heart diseases in the highlands were dominated by insufficiencies and in the lowlands by hypertrophies. In this case, Vargas Vega did not mention any racial difference.[142]

Medical geography and fevers of the warm lands

As I mentioned earlier, Colombian physicians had constructed a divide between diseases following an altitudinal criterion, the diseases of *tierras frias, templadas* y *calientes*, as revealed by the medical curriculum during the 1840s. But, interestingly, the course paid much more attention to diseases of the *tierras calientes* than those of the other two regions. Particular questions such as the effects of rainforest upon the health state of a "burning" country and the reasons for the increased unhealthy situation of hot countries after the cleaning of forests were directly addressed during

[140] Ibidem.
[141] Ibid., p. 2.
[142] Ibidem, p. 2.

the course. In particular, students had to understand the causes of the "insalubrity" of the burning valleys of the Magdalena and Atrato rivers.[143] This focus clearly coincided with an increasing concern about fevers occurring in the Magdalena Valley, close to the tobacco-producing regions.

Tobacco had been cultivated in the region of the Magdalena River valley called Ambalema since the eighteenth century. Its production and commercialization within the local market was monopolized by the government, and continued to be so after the establishment of the new independent State up until 1850, as it constituted a sizable share of revenue. According to historians, Colombia experienced its first economic boom of the nineteenth century in the 1850s and 1860s based on tobacco exports, thus inverting the stagnation of foreign commerce that characterized the first half of the century. In fact, historians have related this boom to the beginning of the slow and painful transition to capitalism in Colombian society that was consolidated in the twentieth century by the coffee trade.[144] The economic boom from tobacco exports lasted almost two decades, between 1850 and 1869, and had a strong impact in Colombian society: not only did it prompt the liberal political reforms of the 1850s, but it also created an optimistic climate of opinion about the Ambalema region and the Magdalena River. For example, some contemporaries referred to it as the Colombian "California of tobacco" and, for the more optimistic, Ambalema was the place of "our industrial revolution".[145]

Physicians shared that optimistic vision of the economic significance of the Magdalena valley, and turned their attention to the region after the 1865-6 epidemics in two villages in the tobacco area, Girardot and Peñalisa. Drs. Antonio Vargas Reyes, Rafael Rocha Castilla (1838-1917) and Domingo Esguerra Ortiz (1841-1877) considered that endemic fevers had developed in the region after the first epidemic of Ambalema and Honda in 1856-7, and affirmed that they were strongly dependant on climatic variables and the production of tobacco. The action of climatic variables

[143] Vargas, *Programa para la enseñanza de la hijiene*, pp. 18-20.
[144] For a history of the tobacco production in Colombia see Luis Fernando Sierra, *El tabaco en la economía colombiana* (Bogotá: Universidad Nacional, 1971) and José Antonio Ocampo, *Colombia y la Economía Mundial. 1830-1910,* **(**Bogotá: Tercer Mundo, 1998).
[145] José María Samper (XYZ was his pseudonym), "Situación industrial", *El Neo-Granadino*, 9 abril, 1857, No. 395; José María Samper, "Impresiones de viaje. (Primer artículo)", *El Tiempo*, No. 9, 27 febrero, 1855, s/p; Ibid, "Impresiones de viaje. (Segundo artículo)", *El Tiempo*, No. 11, 13 marzo, 1855, s/p., s/p.

such as heat and humidity upon the organic matter resulting from the tobacco production caused their miasmatic putrefaction. Examples of that organic matter were the remains of the leather in which tobacco was wrapped and the residues left by the clearing of forests necessary for the cultivation of tobacco. Even tobacco leaves that dried under the sun, thus producing vapors, were among the list of the substances blamed by physicians for fevers.[146]

The economic importance of the Magdalena valley, because of tobacco production and export, explained why physicians turned their attention to fevers in the Magdalena valley. I have shown elsewhere in more detail how physicians who causally associated the economic activity of tobacco and fevers constructed a particularly Colombian medical notion, "Magdalena's fevers", and how they framed their analysis as a typical medical geography work.[147]

The *Memoria sobre las fiebres del Magdalena* (1872) by Dr. Domingo Esguerra Ortiz is the best available example of this kind of study. Esguerra graduated with a degree in medicine in Bogotá in 1862 and spent some years completing his training in the Paris medical school. His experience working as a factory physician for two Magdalena tobacco exporting companies formed the basis for his *Memoria*.[148] Esguerra reaffirmed that Magdalena's fevers occurred not only in epidemic form, but also as endemic fevers that developed in the area of tobacco production. Following the French hygienist Michel Levy in his idea that endemic diseases were the "pathological expression" of the localities,[149] Esguerra claimed that the geological constitution of the soil, altitude and temperature were the elements that determined the sanitary state of a geographical area. In the tobacco region of the Magdalena river valley, he said, crops, the exuberant vegetation and dense forests, as well as

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[146] Antonio Vargas Reyes, "Señor Eloi Ordóñez", *Gaceta Médica*, Serie I, No. 9, febrero 8, 1866, p. 33; Rafael Rocha Castilla, "De la etiolojía de las fiebres intermitentes i remitentes perniciosas del Magdalena", *Gaceta Médica*, Serie I, febrero 8, 1866, pp. 34-35; Domingo Esguerra Ortiz, *Memoria sobre las fiebres del Magdalena* (Santa Ana: Imprenta de D. Díaz, 1872).

[147] Claudia Mónica García, *Las 'Fiebres del Magdalena': medicina y sociedad en la construcción de una noción médica colombiana, 1859-1886*. Tesis de Maestría (Bogotá, Universidad Nacional de Colombia, 2006). A summary of the main arguments concerning the tobacco production and fevers can be found in "Las 'fiebres del Magdalena': medicina y sociedad en la construcción de una noción médica colombiana, 1859-1886", *Historia, Ciencias, Saude-Manguinhos* (Rio de Janeiro), 14, 1, ene.-mar., 2007, pp. 63-89.

[148] Esguerra Ortiz, *Memoria sobre las fiebres del Magdalena*.

[149] Ibid., p. 43. Esguerra cited the 1862 Levy's *Traité d'Hygiène* in this issue. The edition I consulted, the 1850, had the same idea. See Levy, *Traité D'Hygiène*, p. 533.

numerous animals and infinite insects, and reptiles "prodigious in their multiplication", became an endless source of detritus that formed a layer of organic matter, thus constituting a source of miasmas. This matter, once humidified by rain and by cyclical floods, was sent into fermentation by the heat producing the noxious miasmas. These miasmas were found suspended in the vapors of water that could not be absorbed by the impermeable layer of alluvium and clay on the forest floor.[150] The kind of miasma defined the type of fever: miasmas derived from vegetal matter produced paludic fevers, whereas those derived from a mixture of vegetal and animal matter produced yellow fever, which was also present during the epidemics of Magdalena's fevers.[151]

In his medical geographical analysis, Esguerra also included the action of the winds and the seasons as well as individual predisposing causes. Examples of these predisposing causes were the sanguineous or bilious temperaments which, he claimed, predisposed to fevers, as well as dangerous attitudes such as abuse of alcohol or excessive sexual activity.[152] But in contrast to the racial categories that were common in some of the contemporary analyses of the diseases of the highlands, Esguerra used the category of "labor" when identifying the groups that were most affected. This category was common among contemporaries who addressed the issue of the tobacco production, as they treated it as the "industry" of the country. Esguerra believed that workers who lived near the *canei,* the sheds where tobacco was left to dry, were among the most affected as they would be constantly absorbing the miasma.[153] One of the interesting aspects of Esguerra's work is that he did not believe that place of origin, i.e. whether one was local (that is, acclimatized) or originated from the highlands, determined susceptibility to the disease,[154] at a time when there was a common perception that people who had descended from the highlands to the Magdalena valley were more susceptible to fevers. But even for other doctors, racial distinctions were not so much of an issue as had been the case for diseases of the highlands. What was instead important was the status of workers.

[150] Esguerra Ortiz, *Memoria*, pp. 45-6, 51-2, 97, 190-1.
[151] Ibid., pp. 14-15, 22, 74.
[152] Ibid., pp. 43, 57-9; Ibid., *Trabajos médicos sobre las fiebres de Colombia* (Bogotá, s/e, 1877), p. 20.
[153] Esguerra Ortiz, *Memoria*, p. 60.
[154] Esguerra Ortiz, *Trabajos médicos sobre las fiebres*., pp. 19-20.

Dr. Rafael Gutiérrez (1823-1882) explained that workers descending to the Magdalena valley from the highlands suffered the most because they found an environment transformed by human labour, increasing the organic matter and thus exposing them more to miasmas, and also because workers' salaries would be consumed by vice, leaving them without the resources to tackle disease if it appeared.[155]

Another crucial point to keep in mind is that the study of the local pathologies of the temperate, cold and warm climates, that is, the medical geography of the country, was directly fostered by a professional interest. Although the *Universidad Central* had been responsible for medical training in Colombia since 1826, the liberal reforms of the 1850s eliminated the requirement of a university qualification for medical practice. Physicians who returned from their training in Paris from the 1840s onwards thus faced a difficult task in legitimating the medical profession and in drawing boundaries between themselves and traditional healers, as well as forging an institutional basis for the profession. Led by the aforementioned Antonio Vargas Reyes, physicians initiated an active process for consolidating a national medical community through the creation of medical journals, the regularization of medical training, and the establishment of a medical association.

As was mentioned earlier, Antonio Vargas Reyes founded the first medical Colombian journals in the 1850s and 1860s, and together with other physicians who were also returning from the Paris Medical School around those decades, such as Rafael Rocha Castilla, Vargas Reyes founded a private medical school in 1865 and intended to create a medical association. This process led to the establishment of the *Sociedad de Medicina y Ciencias Naturales de Bogotá* (SMCN) in 1873, which acted as the governmental advisor on hygiene until the creation of the *Junta Central de Higiene* (JCH) in 1886.[156] Physicians argued that they were creating a "national medicine" which involved not only the institutional basis for the medical profession and the setting of boundaries to differentiate them from other practitioners, but also the development of a "proper corpus of doctrine", derived from the investigation of

[155] Rafael Gutiérrez, "Aclimatación en el Alto Magdalena", *El Neo-Granadino*, Año II, 3ª época, No. 397, 23 abril, 1857, s/p.
[156] Claudia Mónica García, *Las 'Fiebres del Magdalena* [Thesis]. See particularly chapter 5, "Medicina nacional, geografía médica y fiebres", pp. 115-135.

local pathologies, that is, from works in medical geography.[157] This aim of creating a National Medicine based on medical geography reached its maximum development in the creation of the Colombian medical notion of Magdalena's fevers.

The medical geography of fevers by Domingo Esguerra Ortiz discussed above was very influential in Colombian medicine, at least until the end of the nineteenth century. This medical geography explicitly situated the causes of fevers within the neo-Hippocratic scheme of specific, predisposing and occasional causes, similar to those described by Dr. Jose Felix Merizalde in 1831. It is for this reason that in the next and last section I will follow Esguerra Ortiz' analysis in order to see how that neo-Hippocratic scheme was transformed 50 years later due to the imperative of the study of local pathologies of Colombian medical geography.

The etiological shift of the 1870s

Esguerra Ortiz called "determinant" causes those that directly produced the morbid state, that is, "the meteorological and cosmic conditions of the locality". He added the "determinant specific" causes to this group, which, in the case of Magdalena's fevers, was the paludic miasma.[158] In other words, Esguerra Ortiz moved the general predisposing causes (atmosphere and places), as they appeared in Merizalde's work of the 1830s, to the specific or determinant group of causes as a way of emphasizing the role of the locality in the causality of fevers. Predisposing causes, continued Esguerra Ortiz, were those that favored the development of the disease: individual conditions such as age, sex, temperament, and lifestyle.[159] As we see, these coincide with the individual predisposing causes (individual conditions and attitudes) present in Merizalde's scheme. Finally, with regards to occasional causes, Esguerra Ortiz agreed with Merizalde's notion, that is, that they were those causes that provoke the development of fevers such as emotional impression or a sudden change of temperature.[160]

[157] Ibidem.
[158] Esguerra Ortiz, *Memoria*, p. 94.
[159] Ibid., p. 98.
[160] Ibid., p. 107.

We can say that this shift in the role of environmental conditions (atmosphere and places) from the predisposing to determinant causes, that is, almost to the same level as specific causes, reflects the mid-century emphasis that physicians put on local conditions in the study of diseases, i.e. on medical geography. The case of yellow fever illustrated this shift. As I mentioned, the illness was considered contagious and imported during the 1830s. By the mid-nineteenth century, the emphasis on the climatic determination of diseases led doctors to consider yellow fever a miasmatic, non-contagious disease, caused either by putrefaction of plant matter, in which case it became a paludic fever according to Vargas Reyes's conception of fevers, or by the mixture of the putrefaction of animal and plant matter, according to Domingo Esguerra Ortiz's understanding.[161] These two physicians rejected the contagious nature of yellow fever and thus the possibility of it having been imported. By stressing its local origin, they inverted that which physicians of the first half of the nineteenth century had affirmed about yellow fever.

This shift in the environmental causality of diseases in Colombia, at least with regard to those of warm climates, can be accounted for by the interest associated with the professionalization of medicine. Indeed, in the study of the fevers of the most prosperous economic region, the tobacco growing area, physicians saw the opportunity to demonstrate the importance of building a national medicine based on medical geography.

A second consequence of this shift, by which climate reached almost the same level of importance as specific causes in disease causation, was the increasing condensation of the damaging power of the environment into one object, the miasma. By the mid-nineteenth century and until the 1880s, this intensification meant that the miasma became the object into which the climatic determinism of diseases was condensed and in which it was expressed in its most direct, if elusive way. This was not only the case for diseases of low warm climates, as I showed in the case of Magdalena's fevers, but also for diseases of the highlands of the *cordilleras*. In December 1880, the *sabana* of Bogotá was affected by an epidemic of jaundice, followed by another one of *cholerina* in January 1881. Drs. Nicolas Osorio (1838-1905) and Proto Gomez (1843-1918), both graduates from the Paris medical school

[161] Vargas Reyes, "Discusión sobre la fiebre amarilla", pp. 40, 47-48; Esguerra Ortiz, *Memoria*, pp. 29, 85-87.

in 1865 and 1874 respectively, invoked the neo-Hippocratic and medical geography explanations when analyzing the causes of the epidemics. They claimed that the jaundice epidemic occurred after a long dry season of high temperatures of 24 degrees Celsius on average, according to the measurements of Benito González, then director of the Astronomic Observatory of Bogotá. They also noticed that excavations made in order to build artesian wells were providing evidence that the soil of the *sabana* was formed by alluvium with abundant vegetal matter. This explained, then, that when the dry season came, high temperatures decomposed vegetal and animal matter in the upper layers of the soil. The water vapor and gases, also produced by the sun's heat, would have brought those miasmas to the surface, thus producing the jaundice epidemic.[162]

Osorio and Gómez claimed that the same causes that produced jaundice in December 1880 also produced *cholerina* one month later. They based this argument on the fact that *cholerina* appeared when the jaundice epidemic decreased and that people who suffered from jaundice were later preserved from *cholerina*. They also invoked Hippocrates, who had described a certain parallelism between intermittent fevers (which, like jaundice, were also caused by miasmas) and *cholerina*. Osorio and Gómez concluded that both epidemics were caused by miasmas formed as a consequence of the long dry season and the excessive heat.[163]

While doctors during the 1850s and 1860s used geological and climatic factors to explain the miasmatic origin of fevers of the low warm lands of the Magdalena River valley, in the 1880s Osorio and Gómez blamed the geological constitution (alluvium soil with vegetable matter), the seasonal conditions (the long dry season) and other climatic factors (the high temperature) for the production of miasmas responsible for two clinically different epidemics in the *sabana* of Bogotá, jaundice and *cholerina*. As had been the case for the Magdalena's fevers, the miasma also represented the very product of the conjugation of those environmental elements, a true incarnation, though invisible, of the environmental determinism of those epidemics. Before

[162] Nicolás Osorio y Proto Gómez, "Epidemias de ictericia y colerina en Bogotá y pueblos vecinos. – Fiebres epidémicas de la hoya del Magdalena.- Naturaleza de estas fiebres", *Revista Médica*, Serie VI, No. 61, 20 de mayo (1881), p. 41.
[163] Nicolás Osorio y Proto Gómez, "Epidemias de ictericia y colerina en Bogotá y pueblos vecinos. – Fiebres epidémicas de la hoya del Magdalena.- Naturaleza de estas fiebres. (Continuación)", *Revista Médica*, Serie VI, No. 62, 20 de junio (1881), pp. 83-94.

obvious questions emerged, for example, about how miasmas could cause different diseases in different altitudes, a new element was introduced into this complex landscape of an etiological scheme based on medical geography. Two months after Osorio and Gomez published their report on the jaundice and *cholerina* epidemics of the *sabana* of Bogotá, the naturalist Carlos Michelsen Uribe (1850-1930) described an epizootic of cholera among chickens that had occurred simultaneously with the epidemic of jaundice. Michelsen linked the epidemics with the epizootic. For him, the coincidence in timing "proves the morbid alterations of the atmosphere [that] originated this pathological situation." But this time, however, Michelsen wondered if the coincidence of the occurrence could also have been due to the same cause that M. Pasteur had found to be responsible for the animal disease: the "microscopic infusoria or microbe".[164]

The way germ theory transformed medical theories about disease in Colombia was strongly determined by the emphasis that Colombians put on the geographical determination of diseases. However, it is worth remembering that even by the mid 1880s, the geographical divide that physicians intended to adopt for analyzing the local pathologies of the country was still valid. Dr. Ignacio Gutierrez Ponce, for example, intended to present information about medical education, diseases and therapeutic plants in Colombia at the 1884 international medical congress in Copenhagen. He requested information from the SMCN about the endemic diseases of the regions of the country "according to height and temperature": those of the warm lands, whose limits he drew from sea level to 600-1000 meters above; temperate land, from 600-1000 to 2300-2600 meters; and finally, cold land from 2600 to 3500 meters above sea level. He also wanted information about the epidemics in each of these areas and their causes, as well as the "special symptoms of diseases that develop in Colombia according to climate" which "makes them different from those known in Europe". This request went unfulfilled as the letter arrived very late. [165]

[164] Carlos Michelsen Uribe, "Colera de las Gallinas", *Revista Médica*, Serie VI, No. 64, 20 de agosto (1881), pp. 177, 185.
[165] Ignacio Gutierrez Ponce, "Congreso médico de Copenhague", *Revista Médica*, Serie IX, No. 98, 20 de agosto, 1884: 49-50.

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Having exposed the complexities of neo-Hippocratism and the medical geographical arguments developed by Colombian doctors by the mid-nineteenth century, in the following chapters I will show how they incorporated germs within that frame and how the frame was simultaneously transformed. As I made clear in the introduction, I will follow this transformation by looking at the following cases: intermittent or periodic fevers (yellow fever and malaria) in chapters 2 and 3; typhoid and typhus fever in chapter 4, and leprosy in chapter 5. In the next chapter I will concentrate on the controversy surrounding preventive inoculations against yellow fever, a practice that was reinterpreted in light of Pasteurian principles. This will allow me to explain the transformation in the medical geographical approach to fevers by the germ theory in the subsequent chapter.

CHAPTER 2

YELLOW FEVER AND THE CONTROVERSY ABOUT PREVENTIVE INOCULATIONS IN 1887

In this chapter I will explore the controversy surrounding a germ-theory practice, the preventive inoculations against yellow fever in 1887. The inoculations were among the first therapeutic measures that some physicians in Colombia applied, based on the rationale of the Pasteurian methods of the early 1880s. As I will show, the controversy had practical and theoretical consequences for the management and understanding of yellow fever. Considering that the fever was changing in its epidemiological behavior at that time, I will also explore how physicians reconfigured their understanding of fevers when faced with changes in the diseases and the new theory. I will first situate the debates about malarial and yellow fever in the early 1880s before the preventive inoculations started. Then I will explore in detail the controversy over inoculations, and finish the chapter with the immediate consequences that it had for the Colombian medical thought about fevers. The way that this debate, and the changing epidemiological behavior of fevers, consolidated the emerging view that yellow fever and malaria were distinct diseases, therefore irreversibly transforming the medical geographical explanations of fevers, will be the subject of chapter four.

The emerging distinction of yellow fever and paludic fevers in the early 1880s

According to Coleman, before 1860, physicians in Europe considered paludic or marsh fevers and yellow fever to be variations of a single disease, with yellow fever probably representing its most advanced and severe form.[166] This perspective had been accepted common knowledge since the 1830s, partly due to the French

[166] William Coleman, *Yellow Fever in the North. The Methods of Early Epidemiology* (Madison: The University of Wisconsin Press, 1987), p. 45.

physician Nicolas Chervin (1783-1843), one of the most influential anticontagionists of the first half of the nineteenth century.[167] His influence throughout Europe was most powerful during the 1830s and 1840s, decades that saw the peak of non-contagionist doctrines. Chervin considered that yellow fever was neither contagious nor imported, but instead locally produced by the exhalations generated by decomposing vegetable or animal substances, encouraged by a high temperature and probably also a distinctive atmospheric situation.[168]

Historians like Ackernecht and Coleman have described how Chervin's anticontagionism and his ideas on the continuity of fevers began to be questioned on clinical and epidemiological grounds by the 1860s. This revision led Europeans and Americans to consider yellow fever a non-paludic and independent disease, caused by a specific agent, just before the germ theory of diseases began to transform the causal framework of epidemic diseases.[169]

In Colombia, on the contrary, Chervin's ideas on yellow fever and on fevers in general reached their highest recognition between 1860 and 1880. The author of the most influential essays on yellow fever, Antonio Vargas Reyes, not only publicly acknowledged his intellectual debt to Chervin's concepts, but also explicitly acknowledged that he was against the European medical community which had already discarded the French doctor's views on the matter.[170]

Indeed, I mentioned in the first chapter that Colombian physicians, following Vargas Reyes's essays, considered yellow fever a "pernicious fever in its highest level of severity."[171] Pernicious fevers were thought to be the most deadly of the remittent and intermittent fevers, characteristic of warm lands like the coastal plains and river valleys.[172] Theoretically at least, milder forms of the remittent and

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[167] Erwin H. Ackernecht, "Anticontagionism between 1821 and 1867," *Bulletin of the History of Medicine*, 22 (1948), pp. 570-4; Coleman, *Yellow Fever in the North*, p. 28.
[168] Coleman, *Yellow Fever in the North*, pp. 5-6.
[169] See Coleman, *Yellow Fever in the North* and François Delaporte, *The History of Yellow Fever. An Essay on the Birth of Tropical Medicine* (Cambridge, MA: MIT, 1991), pp. 15-16, 42-45.
[170] Antonio Vargas Reyes, "Discusión sobre la fiebre amarilla", *Trabajos Científicos del Doctor Antonio Vargas Reyes recopilados en obsequio de la humanidad doliente i de la juventud estudiosa de Colombia*, Tomo Segundo (Bogotá, Imprenta de la Nación, 1862), p. 49.
[171] Ibid. pp. 40, 49.
[172] Antonio Vargas Reyes, "Clasificación de las fiebres", *Trabajos Científicos del eminente médico granadino Dr. Antonio Vargas Reyes recopilados en obsequio de la humanidad doliente i de la juventud estudiosa de Colombia por Próspero Pereira Gamba*, Bogotá, Imprenta de la Nación, 1859, pp. 29-30.

intermittent fevers – the benign fevers - could occur in temperate and colder areas such as the highlands if the right conditions of heat, organic matter, and humidity were present.[173]

With regards to the geographical distribution of yellow fever in Colombia, Vargas Reyes affirmed that it had been historically common in the Caribbean coastal plains, but also that since the mid-nineteenth century, it had begun to extend into the Magdalena River valley in the tobacco production areas.[174] In Chapter 1, I described the professional reasons that led physicians to support the idea that epidemic and endemic Magdalena's fevers – including yellow fever and many other varieties of pernicious fevers - were caused locally by the miasmatic putrefaction of animal and vegetal matter from tobacco production. Given this local origin, physicians denied that the fever was imported from the Atlantic coasts or extended by contagion.[175] Although there is evidence that some physicians might have suggested that fevers in the Magdalena Valley could have been introduced in 1856 by the boat *El Cauca* (that came from New Orleans, where endemic fevers were present), this vision was overshadowed by the hegemony of Chervin's and Vargas Reyes's model of the disease.[176]

Thus, until the early 1880s, Colombian physicians considered yellow fever to be a variety of the pernicious remittent and intermittent fevers – called paludic fevers since 1872[177] - that were frequent in the Caribbean coastal plains and in the Magdalena river valley, non-contagious, and locally produced by miasmas of the putrefaction of organic matter generated by the action of the sun's heat and humidity. At that moment, this conceptualization began to change in Colombia, as physicians saw an unprecedented extension of the geographical boundaries of pernicious fevers. They described pernicious fevers, similar to Magdalena's fevers, beginning to "climb up the slopes" of the eastern *cordillera* into more temperate climates. Epidemics in the small towns of Guaduas and Tocaima, on the way from the Magdalena River to the *sabana* of Bogotá, occurred in 1880 and 1884 respectively. These epidemics

[173] Vargas Reyes, "Clasificación de las fiebres", pp. 29-30.
[174] Antonio Vargas Reyes, "Señor Eloy Ordóñez", *Gaceta Médica*, Serie I, No. 9, 8 de febrero, 1866, pp. 33; Vargas Reyes, "Discusión sobre la fiebre amarilla".
[175] Ibidem.
[176] Esguerra Ortiz, *sobre las fiebres del Magdalena* (Santa Ana: Imprenta de D. Díaz, 1872), pp. 85-86.
[177] Ibid., pp. 11-13.

were an anomaly in the history of fevers in Colombia. Guaduas and Tocaima were places that had been traditionally recognized for their "healthy" climates; Tocaima in particular had mineral waters that were believed to have healing powers. Since the first half of the century, laymen and physicians had recommended baths in Tocaima's waters for several diseases such as syphilis, paralysis and leprosy.[178] Physicians were thus surprised that Magdalena's fevers could occur in places "not apt" for paludic fevers, that is, beyond the boundaries of the low warm plains.[179] Since the geographical boundaries of Magdalena's fevers were thus broken, physicians abandoned the idea that fevers were associated with tobacco production and also began to question the idea that they were dependent on climate; the epidemics in Guaduas and Tocaima did not seem to follow any particular agricultural development and happened away from the warm lands of the River valley.

Physicians saved this anomaly by introducing a distinction between the Magdalena's fevers and by changing the identity of yellow fever. Nicolás Osorio and Proto Gómez, in particular, claimed that the Magdalena's fever epidemic in Guaduas (1881) and the Magdalena's fevers of previous years were cases of bilious remittent hematuric fever, an entity described in European medical treatises. This fever was also miasmatic and had a clinical resemblance to yellow fever. In fact, they were so similar, Osorio and Gómez claimed, that the practitioner would find it difficult to distinguish between them. The two physicians also wanted to separate yellow fever from the Magdalena's fevers. They seemed to agree with the idea that yellow fever was a disease that could only occur on the Caribbean coast, and therefore doubted that it could be responsible for the Magdalena's fevers.[180]

Luis Cuervo Márquez (1863-1941), a young physician who graduated in Bogotá in 1884, also tried to explain the extension of Magdalena's fevers to the *cordillera*. Before describing the kind of fever that was climbing the Andes – which, in his view, was not yellow fever - he began his analysis by differentiating Magdalena's fevers according to therapeutic and causal criteria that he had developed in the course of treating them.

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[178] García, *Las Fiebres del Magdalena* (Tesis de maestría), pp. 139; 149-50.
[179] Luis Cuervo M., "Fiebres del Magdalena", *Revista Médica*, X, 106, 20 de septiembre, 1886, p. 235.
[180] Nicolás Osorio y Proto Gómez, "Epidemias de ictericia y colerina en Bogotá y pueblos vecinos. – Fiebres epidémicas de la hoya del Magdalena.- Naturaleza de estas fiebres. (Conclusión)", *Revista Médica*, VI, 65, 20 de septiembre, 1881, pp. 206-9.

Therapeutically, Cuervo Márquez had initially prescribed quinine for all cases of fevers in warm climates, following the teaching of the medical school in Bogotá, and whenever quinine was not effective, he had blamed the intensity of the paludic miasma. However, in his practice he found that one of the epidemics in the Magdalena valley was particularly resistant to quinine. Based on this experience, and on the fact that epidemics were happening in non-paludic places such as Guaduas and Tocaima, Cuervo Márquez came up with the suggestion that this new fever could be independent of the paludic miasma.[181]

Cuervo Márquez also differentiated two kinds of Magdalena's fevers according to their cause. In the first group, there were those produced by "malaria" (that is, telluric or paludic fevers) and in the second, those in which this element did not play any role (typhoid fevers). In the typhoid group he included typhoid fever, recurrent fever, yellow fever, and the new fever that was climbing the *cordilleras*.[182] Unfortunately, Cuervo Márquez's paper was not published completely, and therefore we do not know what his diagnostic was in this case. He did say, however, that the new fever appeared similar to yellow fever. Like Osorio and Gómez, Cuervo Márquez was completely certain that yellow fever was a disease of the coasts.[183]

Thus, between 1881 and 1886, and partly as a consequence of the extension of the geographical boundaries of Magdalena's fevers to the colder climates of the *cordilleras*, Colombian physicians began to invert Vargas Reyes's – and hence Chervin's - model of yellow fever. They denied that yellow fever was a pernicious or paludic fever, and doubted that it was one of the Magdalena's fevers; furthermore, they conceptually confined yellow fever to the Caribbean coasts. This last shift in opinion could have been provoked by the fact that American health policies, which were based on the idea that yellow fever was a disease that only happened in the ports, were accepted by the Colombian government in 1881. Indeed, Colombia agreed with the system of notification of ports and ships infected by cholera and yellow fever that was accepted during the International Health Conference in

[181] Luis Cuervo Márquez., "Apuntes para el estudio clínico de las fiebres del Magdalena", *Revista Médica*, Serie X, No. 104, 20 de julio, 1886, pp. 121-122.
[182] Cuervo Márquez, "Fiebres del Magdalena", No. 106, pp. 232-237.
[183] Cuervo Márquez, "Apuntes para el estudio clínico", No. 105, pp. 185-186.

Washington in 1881.[184] The evidence available only allows me to hypothesize that in theoretically confining yellow fever to the Caribbean ports, and thus denying its place among the Magdalena's fevers, physicians did not want to alarm the government at the precise moment that it was signing international agreements on port health; neither did they want to provoke further consequences of their rejection of Vargas Reyes's ideas, having argued that the notions of Chervin (that had originally inspired Vargas Reyes's view of fevers) had already been rejected by the French Academy of Medicine.[185] Finally, as for the fever in the slopes of the eastern *cordilleras*, physicians declared that it was neither yellow fever nor a paludic fever, but probably the bilious remittent haematuric fever or another variety of the typhoid fevers.

But despite this ongoing transformation of the identity of yellow fever in the early 1880s, Colombian physicians still framed yellow fevers and all other fevers within the nosological conceptualization that had been set decades before. Cuervo Márquez, for example, expected fevers to be unified as variants of the same essence. He knew about the efforts made in the early 1880s to find the microorganism of yellow fever in Brazil, Mexico and Cuba (which I will mention in the following section).[186] But for him, the organisms proposed as the cause of this fever were in fact an obstacle to the unification of fevers, because, as he said, it "individualized" the morbid entities.[187] Considerations of this kind could account for the resistance among Colombian physicians to the idea of germs as the specific cause of disease (in the early years of the idea's diffusion) that some complained about a few years later.[188] As such, the idea of germs was not seriously considered as a valid explanation of the epidemiological transformation of fevers during those years, at least for fevers of the warm lands.

The reconfiguration of the conceptualization of fevers brought about by the "climbing up" of the Magdalena's fevers in the eastern *cordillera* was complicated

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[184] Ibáñez, Pedro María, *Memorias para la historia de la medicina en Santafe de Bogotá* (Bogotá: Imprenta Nacional, 1968 [First edn. 1884]), p. 143. Ibañez says that the Colombian government sent Pio Rengifo, one of the members of the SMCN of Bogotá, to the conference, but when he arrived, the system had already been adopted. However, he signed it off *ad referendum*.
[185] Osorio y Gómez, "Epidemias de ictericia y colerina", p. 208.
[186] Delaporte, *The History of Yellow Fever*, p. 66.
[187] Cuervo Márquez, "Apuntes para el estudio clínico", No. 104, p. 123.
[188] Daniel Gutiérrez Arango, *Los micro-organismos de la fiebre amarilla*, Tesis para el doctorado en Medicina y Cirugía (Bogotá: Imprenta El Telegrama, 1888), p. 11.

by another unexpected extension of the geographical boundaries of this pathology. In 1886 an epidemic of fever broke out in the city of Cúcuta, very near to the Colombian border with Venezuela and far from the Caribbean coast and the Magdalena River Valley.[189] Cúcuta was a prosperous city and belonged to the influential commercial area of the Zulia River and the Maracaibo Lake of Venezuela. The Cúcuta epidemic not only mobilized physicians and merchants in Cúcuta, but also those in Bogotá and the central government. As we will see, these actors became involved in a controversy that was triggered and profoundly shaped by the early notions of germs as the cause of diseases.

The preventive inoculations of the yellow fever germ and the epidemic of Cúcuta

In January 1887 the Ministry of "Fomentation"[190] received a request from Cúcuta, probably from the Cúcuta Health Board, for guidance on how to tackle the epidemic that they had already named as yellow fever. The Ministry forwarded the request to the Society of Medicine and Natural Sciences of Bogotá (SMCN), hoping that the Society would propose preventive and curative methods against the disease.[191] For that purpose, the Ministry also sent them the book *Leçons sur l'étiologie et la prophylaxie de la fièvre jaune* by Manuel Carmona y Valle (1832-1902), the Mexican professor of the Faculty of Medicine in Mexico City.[192] The book had already been reviewed in the local *Revista Médica* of Bogotá several months earlier.[193] In it, Carmona y Valle claimed not only to have isolated the yellow fever germ, the fungus *Peronosporea lútea*, but also to have developed prophylactic inoculations against the disease.

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[189] SMCN, "Actas de las sesiones ordinarias de los días 30 de noviembre de 1886, 4 y 9 de febrero de 1887", *Revista Médica*, Serie X, No. 111, 20 de febrero, 1887, p. 466.
[190] In Spanish "Ministerio de Fomento." This ministry guided hygiene policy and simultaneously sought to stimulate economic growth, develop agriculture, etc.
[191] Ibidem.
[192] Manuel Carmona y Valle y Valle, *Leçons sur l'étiologie et la prophylaxie de la fièvre jaune* (México: Impr. du Ministère des Travaux Publics, 1885).
[193] *Revista Médica*, "Fiebre amarilla. Etiología y profilaxia de la fiebre amarilla", [Extracto de libro extranjero], *Revista Médica*, Serie X, No. 103, 20 de junio, 1886, pp. 75-83.

Of course, the method of preventive inoculations was not new. Inoculations for smallpox (variolization) and cowpox were among the first preventive inoculations of the pre-bacteriological era. Variolization, which probably originated in China and appeared in Europe during the eighteenth century, consisted of applying the matter of the pustules of one with smallpox to the nostrils of those that had not yet been infected in order to prevent the disease. The risks of this controversial technique were reduced when, at the end of the eighteenth century, the English physician Edward Jenner demonstrated that cowpox would also offer protection but with less risk.[194] The Spanish crown introduced variolization to their colonies in the early nineteenth century. But less than a decade after Edward Jenner's inoculations, the Spanish crown sent the Royal Philanthropic Expedition to spread the vaccine in their colonies. The expedition, which transported the vaccine by an in vivo method using human repositories (that is, children in poorhouses), reached Cartagena in the Atlantic coast in May 1804 and traveled via the Magdalena River and the Andes mountains to Bogotá, inoculating people as they went. The expedition continued towards the south to reach the southern Spanish colonies.[195] According to Ibañez, the vaccine introduced by the Expedition in the Colombian territory was preserved and used during the 1840 epidemic in Bogotá, but was lost due to bureaucratic changes afterwards. He also wrote that the vaccine was re-imported and used during the 1857 and 1881 epidemics in the capital city.[196]

The models of variolization and vaccination inspired the search for preventive inoculations for syphilis and yellow fever by the mid-nineteenth century. Syphilization was a form of inoculation whereby the pus from a syphilitic manifestation, usually a chancre, was introduced into the human subject in order to cure, rather than prevent, the development of the disease. Auzias-Turenne (1812-1870), who coined the name by 1850, defined syphilization as "the state of the organism in which he is not able to suffer the evolution of syphilis anymore, because

[194] Anne Marie Moulin, *Le dernier langage de la médicine: histoire de l'immunologie de Pasteur au sida* (Paris, Presses universitaires de France, 1991), pp. 19-21.
[195] A description of the antecedents of the expedition and its structure can be found in Michael M. Smith, "The 'Real Expedicion Maritima de la Vacuna' in New Spain and Guatemala", *Transactions of the American Philosophical Society*, New Series, 64, 1 (1974), pp. 1-74.
[196] Ibáñez, *Memorias para la historia de la medicina*, pp. 74-5, 93-5, 152.

of a kind of syphilitic saturation."[197] Although in 1852 his experiments were declared unjustified and unreasonable, physicians in Norway, Italy and England continued to perform syphilization in the following years.[198]

A vaccine against yellow fever was developed by Guillaume Lambert de Humboldt in the mid-1850s using snake venom.[199] Lambert, who had vaccinated 1438 people, very few of whom, he claimed, had subsequently been infected by the disease, offered his vaccine to the Cuban government for the Spanish troops in 1854. The Cubans accepted Lambert's offer and invited him to Havana. Although, according to one of his supporters, Nicolas Manzini, "his medical education seems very incomplete", Lambert's inoculations were treated seriously in Cuba.[200] He performed inoculations in the military hospital of Cuba that year. The use of the snake poison was justified on the grounds of the principle of analogue substances. Two arguments supported this principle: first, that as in the case of cowpox and smallpox, it was not necessary to presume the identity of the cause of the disease and of the cause of the reaction after the inoculations; secondly, that there was an analogy between the snake venom's action and the yellow fever symptoms, as other nineteenth century physicians had observed.[201] Lambert's inoculations were debated by physicians of the university in Havana, a commission from the military hospital of the same city, and a French commission sent from Martinica.[202] According to Claire Fredj, by 1856 Lambert's inoculations had been rejected by the French medical institutions, although during the 1867 French expedition to Mexico some physicians still considered Lambert's inoculations to be one of the possible preventive measures for the disease.[203]

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[197] For a short description of Auzias-Turenne's work see Nicolas Postel-Vinay and Anne-Marie Moulin, "La syphilisation: ou les rapports utopiques du chancre et de la lancette", *La Revue du Practicien* (Paris), 44 (1994), pp. 1727-1729 and Moulin, *Le dernier langage de la médicine*, pp. 387-391.

[198] Joan Sherwood, "Syphilization: Human Experimentation in the Search for a Syphilis Vaccine in the Nineteenth Century", *Journal of the History of Medicine*, 54, July, 1999, pp. 364, 367-8.

[199] Luis Cuervo Márquez, *La fiebre amarilla en el interior de Colombia. Epidemia de Cúcuta-Fiebres del Magdalena*., Curazao, Imprenta de la librería de A. Betencourt é hijos, 1891, p. 256.

[200] Nicolas Manzini, *Histoire de l'inoculation préservative de la fièvre jaune, pratiquée par ordre du governement espagnol a l'Hôpital militaire de la Havane* (Paris : J.B.Baillière et fils, 1858), p. 4.

[201] Ibid., 9, 86-98.

[202] There is a short description of this debate in Claire Fredj, "Cerner une épidémie: le travail des médecins militaries sur la fièvre jaune au Mexique en 1862 et 1867", *Genèses 38, mars 2000, pp. 79-104*, pp. 103-104.

[203] Ibidem.

So, even before Pasteur's vaccines for chicken cholera and anthrax in the early 1880s, there had been efforts to find a preventive inoculation against yellow fever. However, physicians in Latin America did feel encouraged by the possibility of finding a vaccine against the disease after these successful trials by Pasteur.[204] There were four causal agents proposed and widely debated during the 1880s. Apart from the *Peronospora Lutea* proposed by Carmona y Valle in Mexico, Domingos Freire reported the *Cryptococcus Xantogenicus* from Brazil, while João Batista de Lacerda singled out the fungus *cogumello*. In Cuba, Carlos Finlay isolated the *Micrococcus Tetragenus Febris Flavae*. Only Carmona y Valle and Freire developed inoculations with their microorganisms.[205]

Carmona y Valle had graduated in 1854 from the National School of Medicine in Mexico City, and in 1855 he traveled to Paris where he took courses in physiology and ophthalmology. His biographer affirms that he returned to Mexico and became professor of the medical school in 1866, and after his death he was recognized more for his work on ophthalmology than for his work on yellow fever.[206] Carmona y Valle initiated his work on yellow fever in 1881, part of which was published in the *Voz de Mexico* and the *Escuela de México* journals;[207] his 1885 book is a summary of the lectures that he delivered to his students during the year 1884. From the evidence available we can only presume that Carmona y Valle spent that entire period working in his home country; although he wrote his book in French, it was printed in Mexico City.

Carmona y Valle claimed that the *Peronospora* was a fungus that produced zoospores in the yellow fever patients, which could be eliminated by the use of urine. Those zoospores would develop into *Mucedinea* when telluric and atmospheric

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[204] Delaporte, *The History of Yellow Fever*, pp. 66-7.

[205] On the debates about these hypothetical parasitic or germ causes of yellow fever and the inoculations see Margaret Warner, "Hunting the yellow fever germ: the principle and practice of etiological proof in late nineteenth-century America", *Bulletin of the History of Medicine*, 59, 1985, pp. 361-382; François Delaporte, *The History of Yellow Fever. An Essay on the Birth of Tropical Medicine* (Cambridge, MA: MIT, 1991); Jaime Larry Benchimol, *Dos micróbios aos mosquitos. Febre amarela e a revolução pasteuriana no Brasil* (Rio de Janeiro: Editora Fiocruz, 1999); and Ilana Löwy, *La fièvre jaune au Brésil entre science et politique* (Paris: Editions des archives contemporaines, 2001).

[206] Lázaro Pavía, *Reseña biográfica de los doctores más notables de la República Mexicana* (México: Imp. Eduardo Dublan, 1897), pp. 24-26.

[207] Clementina Díaz y de Ovando, *El doctor Manuel Carmona y Valle y la fiebre amarilla son noticia periodística, (1881-1886)* (México, Facultad de Medicina/UNAM, 1993), pp. XV-XVI.

conditions were appropriated. The *Mucedinea's* spores might then enter the body via air or food. In the body, the spores would transform into *Peronosporas*, producing the uremic state typical of the yellow fever patient. Based on this description, Carmona y Valle y Valle developed a method to prevent the disease by inoculation with the *Peronospora*'s zoospore, obtained from the dried urine of patients. He first sterilized the urine by leaving it in contact with the atmosphere, following Pasteur's idea that microorganisms causing putrefaction were anaerobic.[208] He then mixed one centigram of the residue of urine and one gram of distilled water for the inoculations. By 1885 he had inoculated more than a thousand people in Mexico.

When the SMCN of Bogotá received the Ministry of "Fomentation's" request for help with the epidemic in Cúcuta in January 1887, it simultaneously received a report from Julio Uricoechea, a physician appointed to the military hospital in Cúcuta, describing how he had started to perform prophylactic inoculations following Carmona y Valle's method. Uricoechea declared that he first read about the method in the review of Carmona y Valle's book published in *Revista Medica* of Bogotá in June 1886.[209] The reviewer's intention was, ironically, to help people around the Caribbean coast, because (as I mentioned earlier) by mid-1886, physicians in Bogotá were convinced that yellow fever was a disease that affected the coastlines exclusively.[210]

Following Carmona y Valle's method, Julio Uricoechea began inoculating in Cúcuta in November 1886. He first tried the method with dogs and other animals, reporting positive results.[211] After a failure with his first attempted human inoculation, Uricoechea tried for a second time in January 1887, attempting to innoculate five members of a military base close to the city. Army soldiers were ideal for those experiments, he claimed, as they were mainly peasants from the highlands who had not been exposed to diseases of the warm lands. These five in particular, for

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[208] Carmona y Valle y Valle, *Leçons sur l'étiologie*, p. 78.

[209] Julio Uricoechea, "Observaciones sobre las inoculaciones profilácticas de la fiebre que se ha desarrollado en Cúcuta", *Revista Médica*, Serie X, No. 112, 20 de marzo de 1887, pp. 543-548. It seems that the letter by Uricoechea which was read during the SMCN session of February the 4th, 1887 was the same as that published in March of that year. See SMCN, "Actas de las sesiones ordinarias de los días 30 de noviembre de 1886, 4 y 9 de febrero de 1887", p. 466.

[210] *Revista Médica*, "Fiebre amarilla. Etiología y profilaxia", pp. 75-83.

[211] SMCN, "Acta de la sesión del día 24 de febrero de 1887", *Revista Médica*, Serie X, No. 112, 20 de marzo de 1887, pp. 514-517.

instance, had not yet entered the city because they were afraid of catching "yellow fever." Uricoechea inoculated them in their left arms with 0.02 centigrams of dry urine in one gram of distilled water, which produced "a benign form of yellow fever." On day twelve, Uricoechea sent the soldiers to Cúcuta in order to prove the efficacy of his inoculations. The results were conclusive: none of them contracted yellow fever, "even after staying in a room where a yellow fever patient had lived."[212]

Encouraged by this outcome, Uricoechea performed more inoculations and searched for the microbe with which he was inoculating. He first examined, with a microscope, the urine of seven healthy people who he intended to inoculate. He did not find any microbe, only crystals of uric acid. Then he inoculated them and took samples of urine during the subsequent fever reaction. Uricoechea claimed to have found microbes with the same shape, size, and movements that he had previously observed in the urine of yellow fever patients. After the successful experiment with the five soldiers, confident of the microscopic verification of the germ in several other cases, Uricoechea extended the inoculations. Together with the Venezuelan physician Francisco E. Bustamante, Uricoechea declared that he had successfully inoculated 279 people in Cúcuta between November 1886 and January 1887.[213]

Uricoechea sent the urine of a German man who had died of yellow fever to the SMCN of Bogotá, in the hope that Nicolás Osorio, one of the members of the SMCN, would examine the sample and hence perform inoculations on animals.[214] Uricoechea was unduly optimistic. As we will see, the SMCN treated his experiments with scepticism and even hostility. The Society appointed Gabriel J. Castañeda to prepare an official report on Uricoechea's inoculations, as well as the response to the request of the Ministry of "Fomentation."[215]

Castañeda presented his report in March. He first questioned Carmona y Valle y Valle's descriptions of the *Peronosporas*, his preventive method, and the inoculations performed in Cúcuta by Uricoechea. He then debated the specific

[212] Uricoechea, "Observaciones sobre las inoculaciones", pp. 543-546.
[213] Ibid., pp. 547-548.
[214] SMCN, "Acta de la sesión del día 24 de febrero de 1887", p. 516.
[215] Gabriel J. Castañeda, "Informe relativo a la obra del doctor Carmona y Valle de Méjico sobre etiología y profilaxia de la fiebre amarilla, y á las observaciones de inoculaciones profilácticas de la misma fiebre, practicadas por el doctor Julio Uricoechea, en Cúcuta", *Revista Médica*, Serie X, No. 112, 20 de marzo de 1887, pp. 530-453.

diagnosis of the Cúcuta epidemic, and finished by describing alternative measures that could be taken against it. Castañeda rejected the idea of alternating generations in plants, on which Carmona y Valle had based his description of the cycle of the *Peronosopora*, claiming that it was obsolete according to contemporary mycological advances. Then Castañeda disqualified Carmona y Valle on the grounds that he was not able to cultivate the *Peronospora* following Pasteurian methods. Castañeda contrasted this failure with the work of Domingos Freire, who claimed to have successfully cultivated the *Cryptococcus xantogenicus* "with good results." Castañeda also suggested that the symptoms produced by the inoculations, the "benign yellow fever" described by Uricoechea, could have instead been caused by putrid fermentation of the dry urine, by ptomains, by bacterial alkaloids, or even by the toxic action of the urine.[216] In any case, Castañeda argued, bacteriologists had not found the germ specific to the disease, and that should be enough to condemn Carmona y Valle's method, and as a consequence, Uricoechea's inoculations in Cúcuta too.[217]

In addition to condemning the prophylactic inoculations, Castañeda and others such as Proto Gomez, another member of the SMCN, doubted that the epidemic in Cúcuta was one of yellow fever. This epidemic was another unexpected anomaly in the history of fevers in Colombia. Castañeda did not explicitly reject the possibility that yellow fever could have occurred in Cúcuta, and he seemed unsurprised by the possibility. He simply claimed that "given the fact that yellow fever was unknown in those localities [Cúcuta]" – at least for physicians in Bogotá - "it was first necessary to prove its identity with the yellow fever of the Antilles to justify the inoculations."[218]

Castañeda ended his report by offering some advice for the present epidemic crisis. He recommended quinine based on its tonic, antipyretic and "microbicidal" qualities. The "microbicidal" qualities, he explained, were due to the fact that quinine competed with the parasite for the oxygen in haemoglobin and tissues, thus

[216] Ibid., pp. 536-7.
[217] Castañeda based this conclusion on Cornil's and Babes's book on medical bacteriology. See A-V. Cornil et V. Babes, *Les bactéries et leur role dans l'anatomie et l'histologie pathologiques des maladies infectieuses* (Paris: Félix Alcan Editeur, 1886), first edition 1885. See Castañeda, "Informe relativo a la obre del doctor Carmona y Valle", pp. 534-5.
[218] Castañeda, "Informe relativo a la obre del doctor Carmona y Valle", p. 543.

protecting against the disease.[219] For preventive measures, he suggested old hygienic measures such as fumigation with sulphuric vapors, isolation of ill people, and the creation of a separate cemetery for people who died of yellow fever to "halt the diffusion of the miasma into the atmosphere."[220]

The epidemic of Cúcuta proved to Castañeda that "we are in chaos" with regards to the fevers that were occurring in Colombia. He complained about the lack of consensus and exact symptomatic descriptions of fevers. He thus recommended that the recently created Central Hygiene Board (JCH) should appoint two physicians to study the fever in Cúcuta and those of the Magdalena villages.[221]

It appears likely that the JCH followed Castañeda's advice and appointed Luis Cuervo Márquez to study the epidemic of Cúcuta. Cuervo Márquez began to send reports from the city to the JCH in March 1887. He mentioned that his task was to gather information about the symptoms of the disease and to evaluate the inoculations with a microscope. In March, Cuervo Márquez reported that, contrary to the belief of physicians in Bogotá, people in Cúcuta were sure that the epidemic was yellow fever and considered inoculations to be a safe prophylactic measure.[222] In fact, the Cucuteños had already suffered a fever epidemic not too long before. Between September 1883 and December 1884, 800 people contracted fever and 213 people died. The Cucuteños initially thought that this fever was an unknown paludic fever. But the Venezuelan physician Martinez Saenz claimed that it was the contagious yellow fever. It may be possible that merchants had accepted the fact that it was yellow fever, but that they took an anticontagionist point of view. In any case, when the second epidemic occurred in 1886, the Cucuteños were convinced that it was a second attack of the same yellow fever; this time, the disease had killed 290 people between May and September, a couple of months before the prophylactic inoculations began.[223]

Thus, two divergent opinions emerged in Colombia about the inoculations and the identity of the epidemic of Cúcuta: that of the medical authorities in Bogotá, and

[219] Ibid., pp. 540-1.
[220] Ibid., p. 539.
[221] Ibid., pp. 542-3.
[222] Luis Cuervo Márquez to Nicolás Osorio, Cúcuta, Marzo 29 de 1887 in Luis Cuervo Márquez, "Fiebre de Cúcuta", *Revista Médica*, Serie XI, No. 113, 20 de abril, 1887, pp. 575-577.
[223] Cuervo Márquez, *La fiebre amarilla en el interior de Colombia*, pp. 48, 51-52, 56-58.

that of the Cucuteños. Cuervo Márquez agreed with the SMCN in Bogotá about the inefficacy of inoculations; but interestingly, he agreed with the Cucuteños, "though with high reserve", that the epidemic was of yellow fever. Judging from his reports and his papers published in *Revista Médica*, it seems that Cuervo Márquez had never been in contact with yellow fever cases apart from the controversial epidemics in the Magdalena Valley.[224]

Cuervo Márquez then focused his efforts on evaluating and testing the inoculations, and even sought microscopic evidence of the alleged cause. In April, he explained that practitioners in Cúcuta could have felt encouraged, after the success of the first inoculations, to continue with them in Cúcuta and in nearby villages; people attributed the diminution of cases during February and March to the inoculations. However, Cuervo Márquez doubted that this was the case, as he saw ten people with yellow fever, five of whom had been inoculated.[225] Thus, as he could not find factual demonstrations of the success of the procedure, he performed "experimental" inoculations on eighty individuals, presumably with the dry urine of people affected by yellow fever. They had a febrile reaction within the first thirty-six hours, which Cuervo Márquez interpreted as either a limited form of yellow fever or as a septic fever caused by the introduction of putrefactive matter. Cuervo Márquez's intention was to compare these results with those of inoculations of urine from healthy individuals, but we do not know if he actually did so. Cuervo Márquez also examined the liquid he was inoculating with a microscope and found only vibrions, bacteria, and animated corpuscles, "all agents of putrid fermentation." Most strikingly, he found the same objects in samples of sick and healthy individuals. Cuervo Márquez concluded that he could not find anything that looked like the *Peronospora lútea* of Carmona y Valle, or like the *Cryptococcus xantogenicus* of Domingos Freire.[226]

In Cúcuta, Cuervo Márquez also found that the army physician Julio Uricoechea was not the only one who had been inoculating people; in fact, he found a fully-fledged market for *Peronosporas* and for the procedure. In February 1887, Carmona

[224] Luis Cuervo Márquez to Nicolás Osorio, Cúcuta, Marzo 29 de 1887.
[225] Luis Cuervo Marquez to Nicolás Osorio, April the 14th, 1887 in Cuervo Márquez, *La fiebre amarilla en el interior de Colombia*, pp. 262, 264-5.
[226] Luis Cuervo Márquez to Nicolás Osorio, Cúcuta, Marzo 29 de 1887.

y Valle had sent cultures of his microorganisms in agar-agar from Mexico to Cúcuta.[227] By that time, the Mexican doctor had changed his method and was no longer using dry urine, but instead cultures of the microbe, for the inoculations. He was charging 25 *pesos* per 100 grams of the liquid, and physicians in Cúcuta charged 10 *fuertes* for each inoculation.[228] This practice would also be criticized by the medical authorities in Bogotá.

Cuervo Márquez had the opportunity to test Carmona y Valle's liquid on three women and to examine it with a microscope. He did not find any bacteria or vibrions in the liquid. He could only find corpuscles with Brownian motion, which were also present in other bodily fluids. Thus, he declared, "there is nothing similar to *Peronospora lútea* in this liquid [Carmona y Valle's]" and "I very much doubt Carmona y Valle's discovery."[229]

Carmona y Valle's preparations were also sent for analysis in Bogotá. Uricoechea and Cuervo Márquez sent vials from Cúcuta with the Mexican doctor's preparations.[230] Two of them, containing the *Peronospora* culture and the liquid for inoculations, were analyzed at the laboratory of the medical school in Bogotá by Gabriel Duran Borda, professor of histology, and Proto Gómez.[231] It seems that by 1887, Gabriel Duran Borda was running a laboratory on histology and micrography that the Ministry of Instruction would only recognize as the official laboratory of the Faculty of Medicine one year later, in April 1888. The laboratory's aim was to support lectures on pathological anatomy, with assistance from two of the most diligent and skilled students of clinical classes. Gabriel Duran Borda offered his unpaid services as director.[232] In the analysis of Carmona's samples, Borda,

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[227] Cuervo Máquez transcribed Manuel Carmona y Valle's letter from México, February the 9th, 1887 to Vicente Ibarra, who received the liquids in Luis Cuervo Márquez, "Fiebre de Cúcuta", pp. 575-580.
[228] Luis Cuervo Márquez to Nicolás Osorio, Cúcuta, April the 14th, 1887 in Luis Cuervo Márquez, "Fiebre de Cúcuta", p. 579. This may be a simplified version of the letter published several years later in Cuervo Márquez, *La fiebre amarilla en el interior de Colombia*.
[229] Ibid, pp. 577-9.
[230] Ibid., p. 579; SMCN, "Acta de la sesión ordinaria del día 12 de mayo de 1887", *Revista Médica*, Serie XI, No. 114, 20 de mayo, 1887, p. 593.
[231] Proto Gómez, "La fiebre amarilla y las fiebres paludosas graves", *Revista Médica*, Serie XI, No. 114, 20 de mayo, 1887, pp. 628-9; Gutiérrez Arango, *Los micro-organismos de la fiebre amarilla*, p. 70.
[232] Ministerio de Instrucción Pública (Colombia), "Decreto 326 de 1888 (9 de abril) por el cual se establece un laboratorio de histología y micrografía," *Diario Oficial*, Año XXIV, No. 7.347, 14 de abril de 1888, p. 335; Proto Gómez, "Importancia de los estudios bacteriológicos", *Revista Médica*, XII, 128, Agosto 28, 1888, p. 132.

seconded by Gómez, was assisted by the student Daniel Gutierrez Arango, who would write his medical thesis on the subject. In the first sample, they could not find anything similar to the fungus that Carmona y Valle described in his book, and in the second they could only find microbes related to putrefaction.[233] They also showed that if they left normal urine exposed to the air in the laboratory for several hours, microscopic observations would reveal mycelia identical to the *Peronospora* described by the Mexican doctor. In other words, they found the *Peronospora* in the air of the laboratory in Bogotá. Durán Borda, Gómez and Gutiérrez thus concluded that "the *Peronospora* is not the specific agent of yellow fever", and that the inoculations were, for that reason, useless.[234]

The SMCN waited until May to announce its final verdict on the inoculations in Cúcuta in the *Revista Médica*. They based their official position on Castañeda's report, the observations and experiments performed in Cúcuta by Cuervo Márquez, and the conclusions of the laboratory analysis in Bogotá. The SMCN thus unanimously accepted the conclusions reached by Castañeda that I described above, and asked the government to ban the inoculations. The government supported this request for many reasons: there was no certainty that the epidemic was of yellow fever (although in Cúcuta, nobody doubted it); there was disagreement about the "micrographic" studies of the germ that produced the disease; and, most importantly (they argued), inoculations were dangerous according to reports from Cúcuta of people who died after the procedure.[235] Some people even considered it possible that the inoculations with Carmona's liquid might infect people with true yellow fever; thus, instead of protecting Cúcuta's population, they would be risking the spread of the disease.[236] The SMCN was additionally concerned about the involvement of physicians in the economic speculation of the inoculations, since it would have a negative impact on the reputation of the profession.[237]

The JCH, whose members were also members of the SMCN (Michelsen, Posada, Osorio and Durán Borda), agreed with the proposal to ban the inoculations, and even

[233] Gutiérrez Arango, *Los micro-organismos*, p. 72.
[234] Ibid., pp. 41-44; 80.
[235] SMCN, "Acta de la sesión ordinaria del día 12 de mayo de 1887", p. 595.
[236] Proto Gomez, "La fiebre amarilla y las fiebres paludosas graves", *Revista Médica*, Serie XI, No. 114, 20 de mayo, 1887: 626.
[237] SMCN, "Acta de la sesión ordinaria del día 12 de mayo de 1887", p. 595.

wanted to prohibit "human experimentation" related to their use.[238] The request was immediately accepted by the central government. In late May, the Ministry of Government banned the preventive inoculations and left the local authorities in Cúcuta and the Governor of the Santander province with the task of providing an effective resolution.[239]

Before analyzing the reaction to this decision in Cúcuta, it is worth noting that in Bogotá, physicians explicitly contrasted the inoculations against yellow fever with the Pasteurian vaccines against anthrax and rabies. Indeed, one of the criticisms Castañeda made of the inoculations in his report was that in contrast with Pasteur's vaccine against anthrax, where "the inoculator has, in this case, a precise notion of the kind of virus that is being injected and the results that can be obtained", [240] there was no certainty about the microorganism inoculated in Cúcuta to prevent yellow fever. In a similar move, and at the same time as the publication of the SMCN's final verdict on the inoculations affair, Nicolás Osorio published an update of Pasteur's work on rabies in the *Revista Médica*. Colombian physicians had followed Pasteur's work on the subject between 1882 and 1884, when cases in Colombia and new treatments had been under discussion.[241] In the midst of the debate about yellow fever inoculations in Cúcuta, Osorio translated excerpts from the discussion of the rabies vaccine at the Academy of Medicine in Paris. He selected the section in which the rabies vaccine was questioned because one individual called Reveillac had died as a consequence of rabies inoculated in Pasteur's laboratory. Pasteurians defended the vaccine using statistical evidence. Osorio, taking the Pasteurians' point of view, claimed that there were "incontestable facts" about the action of the rabies virus, and

[238] JCH, "Sesión del día 23 de mayo de 1887", *Junta Central de Higiene* (Bogotá: Casa Editorial de M. Rivas y Cía, Director, 1887), pp. 32-33.
[239] Felipe Paul, Ministry of Government, Resolution of May the 15th, 1887 in *Diario Oficial*, Año XXIII, No. 7050, 21 Mayo, 1887, p. 562; JCH, "Sesión del día 30 de mayo de 1887", *Junta Central de Higiene* (Bogotá, Casa Editorial de M. Rivas y Cía, Director, 1887), p. 43.
[240] Ibid., p. 536.
[241] SMCN, "Actas de la Sociedad de Medicina y Ciencias Naturales. Sesión del día 1º de junio de 1881", *Revista Médica*, Serie VII, No. 74, 20 de julio, 1882, pp. 49-50; Louis Pasteur, "Rabia. Nuevos hechos que sirven para el conocimiento de esta enfermedad", *Revista Médica*, [Extracto de Revista Extranjera], Serie VII, No. 83, 20 de abril, 1883, pp. 481-486; Gabriel J. Castañeda, "Vacuna de la rabia. Un bienhechor de la humanidad", *Revista Médica*, Serie IX, No. 98, 20 de agosto, 1884, pp. 88-91; *Revista Médica*, "La rabia", [Extracto Revista Extranjera], *Revista Médica*, Serie IX, No. 98, 20 de agosto, 1884, pp. 86-87.

that the rabies vaccine was effective in the majority of cases.[242] Given the fact that Osorio, then editor of the *Revista Médica*, selected these sections from the Academy of Medicine in Paris and made this claim immediately following the SMCN paper that condemned the inoculations in Cúcuta, it is possible to surmise that Osorio wanted to contrast the statistically-demonstrated success of Pasteur's vaccine with the information from Cúcuta that demonstrated the failure of the inoculations against yellow fever.

The reaction in Cúcuta

As I mentioned above, the Ministry of Government, on the advice of the SMCN and the JCH, ordered that the inoculations be canceled. However, soon after that, "respectable" people from Cúcuta appealed against the decision, and the Ministry suspended the resolution very quickly.[243] As a consequence, inoculations continued in Cúcuta for several months.

In Cúcuta, the interest in Carmona y Valle's inoculations and the strong opposition to the Ministry of Government resolution that banned them were motivated by economic reasons: the flow of people traveling to Cúcuta for commercial purposes had substantially diminished due to fear of the disease, not only during the 1886 epidemic, but also during the first attack of the fever in 1883. Rode Heinrich, one of the German merchants who had settled in Cúcuta in the 1840s, said that during the first epidemic of yellow fever (1883), people would not go there to conduct business as "death was almost certain." His import/export company, therefore, appointed him to deliver products to clients in other parts of the country between the end of 1882 (when, he said, the first epidemic began) and 1886.[244] Other people, however, continued to find their way to the city during the epidemics in order to carry on with their business: Pablo Garcia Medina, a young physician who worked in the city of Sogamoso in the nearby Province of Boyacá to the south, remembered

[242] *Revista Médica*, "Inoculación de la rabia", *Revista Médica*, Serie XI, No. 114, 20 de mayo, 1887, pp. 599-601.
[243] JCH, "Sesión del día 30 de mayo de 1887", p. 43.
[244] Jaime Pérez López, *Colombia-Venezuela. Economía-Política-Sociedad. Siglos XIX-XX* (Cúcuta: Offset La Opinión, 2003), p. 156.

how people from Sogamoso who went to Cúcuta fortnightly for business reasons continued to do so during the epidemic, based on the popular observation that the fever "was frightened away by the sun." Thus, he said, they would only enter the city after nine in the morning and leave before four in the afternoon, and by doing so avoid contracting the disease.[245]

The fact that people in Cúcuta managed to reverse the decision of the central government to ban the inoculations – and by doing so, ignore the recommendations of the medical authorities in Bogotá - reflects the relative economic strength and political autonomy of the province of Santander. The province of Santander was the first and main producer of coffee in Colombia during the nineteenth century. While tobacco production had declined by the end of the 1870s, coffee production increased fivefold from 1870 to 1897, 70% of which occurred in Santander;[246] the coffee was stocked in Cúcuta and exported via Maracaibo in Venezuela.[247] At that time, there was a general perception that Santander was a prosperous region where poverty was rare.[248] Besides, Cúcuta's geographic location, its proximity to a Caribbean port, and its active commerce with Maracaibo (where yellow fever developed in endemic form) explains not only the locals' belief in the yellow nature of the fever, but also the fact that they were more receptive than the physicians in Bogotá to a germ hypothesis of yellow fever and hence to the preventive inoculations.

As I mentioned at the beginning of the above section, in early 1887, people from Cúcuta requested help from the Ministry of "Fomentation" who in turn forwarded the request to the SMCN of Bogotá, hoping that that body would recommend preventive measures to tackle the fever. This unleashed the controversy that was just described. But in addition to all this, the Cucuteños had not merely waited for the SMCN's advice. In addition, they attempted to analyze Carmona y Valle's prophylactic method for themselves, with the dry urine of yellow fever patients. The Health Board of Cúcuta appointed an envoy to Mexico in February 1887; the trip was paid for by

[245] García Medina said that people who managed to escape the fever by following this pattern, but who were also inoculated, died of the fever. Proto Gómez, "Algunas consideraciones sobre las inoculaciones profilácticas de Cúcuta", *Revista Médica*, Serie XI, No. 113, 20 de abril, 1887, p. 583.
[246] Marco Palacios, *Coffee in Colombia, 1850-1970* (Cambridge: Cambridge University Press, 1980), pp. 15, 18, 23.
[247] Pérez López, *Colombia-Venezuela*, pp. 18-9.
[248] Palacios, *Coffee in Colombia,* pp. 15, 18-19.

the merchants of the city, who were probably also the constituents of the board.[249] The envoy was Félix María Hernández, who probably reached Mexico by March and returned to Colombia at the end of May, when the SMCN and the JCH in Bogotá had already given their verdict on the inoculations. We know that Hernández also visited the United States, Cuba and Maracaibo on the same trip.[250] In Mexico, Hernández met Carmona y Valle and the medical body of the city. He found that by then, Carmona y Valle had modified his method from the dry urine of yellow fever patients to a microorganism that he had isolated from the blood and attenuated following the attenuation methods of the Brazilian, Domingos Freire. Hernández also found that Carmona y Valle's work had been rejected by the Medical Academy of Mexico, and received news claiming that Domingos Freire's inoculations in Brazil had failed.[251] Thus, when Hernández returned to Cúcuta, he confirmed what he had heard and seen abroad.[252] As such, the commissioner of the Cúcuta Hygiene Board reached the same conclusions as the SMCN and JCH in Bogotá.

The JCH and the SMCN of Bogotá contacted Hernández, and he began to report to them from Cúcuta on the development of the inoculations in the following weeks. In June, he declared that although in Cúcuta and in the surrounding villages, "people are intimately convinced of the inefficacy of the prophylactic method", Julio Uricoechea and Francisco E. Bustamante, the two physicians that initiated the inoculations in Cúcuta, were continuing to inoculate soldiers, probably with Carmona y Valle's liquid (that was available on the market) instead of dry urine. Uricoechea had been producing statistics from the military hospital where he was performing the inoculations. Hernández suggested that Uricoechea was manipulating the diagnoses in order to support the procedure.[253] Statistics from the hospital for the month of April showed that the sixty patients who stayed in the hospital during that

[249] José de Anzoátegui, Consulate of the EE.UU. of Colombia, to the President of the Health Board of San Jose de Cúcuta, México, March 22nd, 1887 in *Revista Médica*, "Fiebre amarilla. Inoculaciones preventivas", *Revista Médica*, Serie XI, No. 116, 20 de julio, 1887, pp. 705-6; Félix María Hernández to Nicolás Osorio, Pamplona, June the 7th, 1887 in *Revista Médica*, "Fiebre amarilla", p. 708.
[250] JCH, "Sesión del día 30 de mayo de 1887", p. 43.
[251] Félix María Hernández to Clímaco Calderón, Cónsulate of Colombia, Nueva York, April 25th, 1887 in *Revista Médica*, "Fiebre amarilla. Inoculaciones preventivas", *Revista Médica*, Serie XI, No. 116, 20 de julio, 1887, p. 703.
[252] Félix María Hernández to Nicolás Osorio, Pamplona, junio 7 de 1887, pp. 708-9.
[253] Ibid., p. 708.

month were inoculated against yellow fever and none of them fell ill with it; cases of fever were diagnosed as paludic, intermittent or remittent fever.[254]

Although reports of more inoculations continued to reach Bogotá until July, the SMCN and the JCH decided not to comment on these new inoculations, or even on the microbiological cause of the yellow fever, for at least one year. A provisional closure of the controversy came from the JCH. In September 1888 they declared that George Sternberg, who had been commissioned by the United States government to study the prophylactic inoculations of yellow fever in Mexico and Brazil, had found neither Carmona y Valle's *Peronosporea Lútea* nor Freire's *Cryptococcus Xantogenicus* in the blood or tissues of ill people. Sternberg also found errors in Carmona y Valle's and Freire's statistics, and thus considered that the inoculations had no scientific value.[255] This short statement during a meeting of the JCH closed the debate over the Cúcuta inoculations in Bogotá.

But the problem of the identity of the fever in Cúcuta was not resolved. During the debate over the inoculations, physicians in Bogotá always doubted that the epidemic of Cúcuta was yellow fever, in contrast to locals who were convinced that it was the case. As I mentioned in the first part of this chapter, by the early 1880s, physicians in Bogotá were convinced that yellow fever was a disease of the coasts; since Cúcuta was located far away from the coast, they believed it was impossible that yellow fever could have happened in Cúcuta. In contrast, physicians in Cúcuta, and those who visited the city like Cuervo Márquez, were convinced yellow fever was the fever of the epidemic. Hernández had also seen the disease in Maracaibo, Veracruz, Tampico, Yucatán and Cuba during the trip that he made on behalf of the Cúcuta Hygiene Board. He thus also believed the epidemic was of yellow fever, though he did not deny the presence of "all varieties of paludic fevers", including the remittent bilious fever of warm climates. [256]

[254] Julio Uricoechea, "Cuadro que manifiesta el movimiento del Hospital militar de Cucuta en el curso del mes de abril del corriente año [1887]" in a letter from Julio Uricoechea to JCH, San José de Cucúta, May 11th, 1887, in JCH, "Sesión del día 30 de mayo de 1887", pp. 40-42.
[255] JCH, "Sesión del día 3 de septiembre de 1888", *Revista de Higiene*, Año 1, No. 8, 25 de octubre, 1888, p. 192.
[256] Félix María Hernández to Nicolás Osorio, Pamplona, June the 7th, 1887, p. 710.

The chemical vaccine

One of the lasting consequences of the inoculations affair, however, was that the SMCN and the JCH embraced the idea that yellow fever was caused by a "parasite" or microorganism. Castañeda affirmed in his report to the SMCN on the inoculations that "despite the present uncertainty about the nature of the parasite of yellow fever, one can admit *a priori* its existence", as its general symptoms were similar to what were called "microbial diseases."[257] For the Colombian physicians of Bogotá, who were the medical and scientific authority in Colombia, yellow fever seemed to have attained a germ identity through the controversial application of a germ-theory related practice, the preventive inoculations.

But physicians in Bogotá not only embraced the new germ identity for the disease; they also sought to create a vaccine. Although the vaccine that they created was a one-time effort that seemed to have only been tested on one person, its analysis shows how, with very limited laboratory resources, Colombian physicians accommodated and applied Pasteurian laboratory methods to deal with a local problem for the first time.

In 1888 two of the hardest critics of the Cúcuta inoculations, the physicians Osorio and Castañeda, published a review of recent French debates over the immunity produced by the products of the microbes and not by the microbe itself, that is, chemical vaccines. In particular, they summarized Roux's and Chamberlain's work on the immunity against septicemia by soluble substances (1887), Chantemesse's and Vidal's work on the "tifotoxina" that immunized mice against typhoid fever, and finally, Bouchard's communication of 1884 about the presence of vaccine-like substances in the urine of "pyocyanic" animals, that could protect healthy animals against "pyocyanic" disease.[258] Castañeda and Osorio believed that these new findings "shed light on the prophylactic inoculations of dry urine from yellow fever patients that were used in Mexico and Cúcuta some time ago."[259]

[257] Castañenda, "Informe relativo a la obra del doctor Carmona y Valle", pp. 538-9.
[258] Nicolás Osorio and Gabriel J. Castañeda, "Vacunas químicas" in *JCH*, "Sesión del día 15 de octubre de 1888", *Revista de Higiene*, Año 1, No. 9, 25 de noviembre, 1888, pp. 214-19.
[259] Ibid, p. 219.

Indeed, according to Castañeda and Osorio, Paul Gibier, the envoy sent by the French to study the fever in Havana in 1888, had found a microbe in the intestines of yellow fever patients that could be the true yellow fever microorganism. They linked this finding with the fact that (invisible) "ptomains", elaborated by microorganisms in the body, were eliminated in urine and had the power to give immunity against the disease they come from. Thus, they suggested, the value of the preventive inoculations performed earlier could be analyzed from this new point of view.[260]

Indeed, one year later, in 1889, Castañeda and Borda presented the results of Colombia's first and probably last experiment ever on the creation of a vaccine against yellow fever. In addition to the suggestions to work in the direction mentioned above, they invoked more arguments regarding the action of ptomains, immunity and urine. Bouchard had demonstrated, they said, that soluble matter enhanced by the "pyocyanic" bacillus had the same vaccine properties as the soluble matter extracted from cultures of the same microbes; and, most importantly, that these substances were eliminated by urine. Based on this fact and on their experience in analyzing the inoculations of Cúcuta, and having at hand the histological laboratory recently created within the *Universidad Nacional*, Castañeda and Borda experimentally assayed the efficacy of a vaccine for yellow fever, composed of the ptomaine found in the urine of individuals affected by the disease.

Ironically, Castañeda and Borda used in their experiments one of the samples of urine sent by Julio Uricoechea, the army physician who had initiated the inoculations in Cúcuta during the 1887 epidemic, who they had strongly criticized. Their experiment consisted of injecting one guinea pig with 0.02 cg of non-sterilized urine and another one with 0.02 cg of sterilized and filtered urine. Both guinea pigs suffered weakness and rise in temperature for four days, among other symptoms. On the fourth day, the guinea pigs were reinoculated with 0.10 cg of sterilized and filtered urine. The symptoms after this second inoculation were milder, and the variations in temperature less intense.[261] Castañeda and Borda compared these findings with the inoculation of sterilized urine from healthy people in another guinea pig. This time they found that the temperature went down, and that after a

[260] Ibid, pp. 219-220.

[261] Gabriel J. Castañeda y Durán Borda, "Aplicación de la vacuna química al hombre", *Diario Oficial*, Año XXV, No. 7880, 19 de septiembre, 1889, p. 911.

couple of hours the guinea pig recovered its normal temperature without having suffered any alteration of its health.

Castañeda and Borda concluded that the symptoms of the first two guinea pigs after the two inoculations with urine from a yellow fever patient indicated the action of a toxic substance, the ptomaine of the yellow fever.[262] On August 3, they informed ,the SMCN of their experiments and promised to create cultures of the microbe in order to make "experimental comparisons".[263] Although I have not found evidence as to whether or not this promise was kept, it is very likely that they did not make any cultures, as there is no mention of that in the report published in September. One day before the publication, the SMCN credited them as the first to have found a method for a chemical vaccine in Colombia.[264]

Following the inoculation of guinea pigs, Castañeda and Borda "felt authorized to apply the vaccine to humans." They found a volunteer, the young physician Aristides Salgado, who intended to practice medicine in Cúcuta and wanted to be immunized against the fever. He received Castañeda and Borda's vaccine on August 21, 1889 in the deltoids of his right arm. He suffered local inflammation and some general symptoms such as insomnia, headache, and thirst, all of which lasted two days. He was reinoculated, and presented with symptoms in his joints, thirst, delirium and jaundice that lasted a further two days.[265] Castañeda and Borda expected that if the inoculation protected Salgado from the fever that was currently rife in Cúcuta again, this would mean that the JCH could apply the inoculation on a bigger scale. Although the vaccine granted Salgado his membership of the SMCN, he unfortunately contracted the fever soon after his arrival in Cúcuta.[266]

It is obvious that Castañeda and Borda must have accepted, by then, that yellow fever had indeed reached Cúcuta, otherwise they would not have used Uricoechea's samples in their vaccine. Physicians in Bogotá, therefore, had changed their minds about yellow fever. At the beginning of this chapter, I showed that in the early 1880s,

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[262] Ibidem.
[263] SMCN, "Acta de la session del 3 de Agosto de 1889", *Revista Médica*, Serie XIII, No. 143, 1 de noviembre, 1889: 698-9.
[264] SMCN, "Acta de la sesión del día 28 de Septiembre de 1889", *Revista Médica*, Serie XIII, No. 144, 1 de diciembre, 1889: 730.
[265] Castañeda y Borda, "Aplicación de la vacuna química".
[266] Zenón Solano, "Fiebres del Alto Magdalena", *Anales de la Academia Nacional de Medicina*, Tomo I.-Entrega Segunda, 1894 (Bogotá: Imprenta de "La Luz"), p. 256.

physicians in Bogotá were increasingly convinced that yellow fever was a disease of the coasts. I also showed that they struggled to come up with identities for the Magdalena's fevers that were climbing up the eastern *cordillera* in Guaduas and Tocaima, and the epidemic of Cúcuta. It may be the case that the reports from Cúcuta insisting that the city had been affected by yellow fever, and the reappearance of the disease in 1889, helped to change their minds.

Thus, by 1889, physicians in Bogotá had no problems accepting that yellow fever could happen in Cúcuta. But, more strikingly, they also accepted that it could happen on the slopes of the *cordillera*. That is, they accepted that it could be among the non-paludic fevers that had been climbing the slopes of the eastern *cordillera* on their way to Bogotá since early 1881. Castañeda and Borda claimed that their research on the vaccine against yellow fever was an urgent matter, because the *sabana* of Bogotá was being threatened by the fever. They had been receiving reports of cases of the fever from Juntas de Apulo and Anapoima, two villages located on the way to Bogotá from the Magdalena River. They even proposed that, along with the vaccine, they would probably need to destroy germs using *estufas* filled with hot air, in which they could disinfect luggage, clean trains, or even quarantine the *sabana*.[267] The time of Chervin and the pernicious fevers had already passed, and new explanations needed to be found for the spread of yellow fever now that the disease was not considered miasmatic but instead "parasitic."

Explanations for Cúcuta's epidemic in 1889

The Cúcuta epidemic of 1886/7 was not only the backdrop for the inoculations against yellow fever and the event that sent physicians in Bogotá on their way to finding a chemical vaccine. The very occurrence of epidemics in a city located away from the seashore, and even away from the riverside, presented a riddle to physicians in Bogotá, which they confronted when the fever reappeared in Cúcuta in 1889. They hypothesised that the microbe of yellow fever was transmitted by drinking water, or

[267] Gabriel J. Castañeda y Durán Borda, "Aplicación de la vacuna química al hombre", *Diario Oficial*, Año XXV, No. 7880, 19 de septiembre, 1889: 912.

that its persistence could have been due to climatic conditions. Thus, the JCH appointed Aristides Salgado, the young doctor who had been vaccinated with the chemical vaccine by Borda and Castañeda, to search for the causes of the disease. First, Salgado had to analyze the waters that people used for consumption, and send samples to Bogotá for examination. Why did the JCH think water was the vehicle of the disease? One possible explanation is that they believed Paul Gibier's hypothesis of a microbe located in the intestine, and so could have assumed thus that drinking water had been somehow contaminated by the feces of affected people.[268] But, as I mentioned, the JCH also believed that the cause of the disease was in the environment, as they ordered Salgado to report to them "in great detail on all the climatic conditions that caused the epidemic or the endemic form to persist."[269] What, then, did Salgado find?

Salgado reported from Cúcuta in February 1890. As ordered, he reported (though not in much detail and with no instrument) on the kind of water that people drank, and on the climatic and geographical conditions of the city (temperature, winds, waters). He also included a quick description of the hygiene of cemeteries, slaughter houses, market places, and the cleaning system. Surprisingly, Salgado did not draw any conclusion from these elements as to the causes of yellow fever or its persistence. The exception was paludism, which he found to be the "predominant" element of the region, whose "miasmas" were mainly produced by the combination of heat and water deposits left from the rain in the yards of houses.[270]

In a probable repetition of the opinion of locals in Cúcuta, Salgado claimed that the disease was imported from Maracaibo on the Venezuelan Caribbean coast, and pointed to the development of communication systems such as the steamboats and railway as responsible for the re-importation of the disease. Salgado summarized the history of the railways built between 1879 and 1888 that connected Cúcuta and the Zulia River, and of the steamboats developed since 1881, that traveled up to

[268] JCH, "Instrucciones que la Junta Central de Higiene da al doctor Aristides Salgado, como médico nombrado por esta junta para trasladarse á Cúcuta á estudiar la fiebre amarilla", *Revista de Higiene*, Año II, No. 15, 31 de octubre de 1889, p. 422.
[269] JCH, "Instrucciones que la Junta Central de Higiene da al doctor Aristides Salgado, como médico nombrado por esta junta para trasladarse á Cúcuta á estudiar la fiebre amarilla", *Revista de Higiene*, Año II, No. 15, 31 de octubre de 1889, p. 421.
[270] Aristides Salgado, [Informe de la Comisión que estudia la fiebre de Cúcuta], *Revista de Higiene*, Año II, No. 19, 21 de abril, 1890, pp. 550-551.

Maracaibo to export goods to the Atlantic world. According to Salgado, the development of these systems of transport reduced the distance between Maracaibo and Cúcuta by an eighth: 36 hours by mule between Cúcuta and the Zulia River was reduced to two and a half hours by railway; and 25 days by bongo from the Zulia River to Maracaibo was reduced to three days using steamboats; New York was then only 13 days away from Cúcuta.[271] Salgado linked the reduction of the time needed to travel between Cúcuta and Maracaibo (on the Caribbean coast, where endemic yellow fever occurred) with the persistence of yellow fever. The underdevelopment of the Colombian transport, in his view, had acted as a kind of natural quarantine system, which had now been broken by the railways (not only in Cúcuta, but also in the eastern *cordillera*, in Girardot and Juntas de Apulo).

Although the JCH expected to find the vehicle of the yellow fever germ in the water supply, and the reason for the repetition of fevers in Cúcuta in the environmental variables, its envoy, Aristides Salgado, pointed to another source of fever: importation from a Caribbean port, Maracaibo, and the new transport mechanisms of steamboats and railways that imported and reimported the disease. In any case, he and the JCH members stuck with the germ identity that the disease had gained during the inoculations affair. But with regard to paludism, Salgado retained the miasmatic and environmental definition that was linked to water deposits and heat. This divide between the environmental definition of paludism and the germ definition of yellow fever would persist for several more years, as I will show in the following chapter.

In conclusion, between 1860 and 1880, physicians and hygienists in Colombia considered yellow fever to be a variety of intermittent or paludic fevers, grouped with the Magdalena's fevers. Its climatic determinism and non-contagionist nature implied that it could occur on the Caribbean coasts and in the river valleys. The unexpected climbing of the Magdalena's fevers up the slopes of the eastern *cordillera* in the early 1880s caused physicians to differentiate between paludic and typhoid fevers. Yellow fever was made part of this latter group. As a reaction to the work of previous decades, physicians also assured people that yellow fever could

[271] Salgado, [Informe de la Comisión], p. 552.

only occur on the Caribbean coasts. But, paradoxically, the epidemic of Cúcuta, that was claimed by locals to be yellow fever in conflict with the opinion of physicians in the capital, and the controversy around the preventive inoculations, led physicians and hygienists in Bogotá to accept a microbiological, yet hypothetical, identity for yellow fever. The fact that physicians in Bogotá tried to create a chemical vaccine by 1889, using samples from Cúcuta, as well as the new challenges posed by the recurrence of the fever in that same year, illustrates the instability and rapid change of the knowledge of fevers towards the end of the 1880s. Indeed, the germ hypothesis of yellow fever consolidated its emerging differentiation as an independent disease. While in Europe and America, physicians had discredited the paludic character of yellow fever and had characterized it since the 1860s as an independent disease on clinical and epidemiological grounds, in Colombia it was not until 1887, and as a consequence of a germ-theory-based practice, inoculations, that this transformation was finally consolidated. In the next chapter I will analyze the implications of the acceptance of the germ theory hypothesis of yellow fever for the nosological and medical geographical explanations of fevers in Colombia.

CHAPTER 3

RECONCILING MEDICAL GEOGRAPHY AND BACTERIOLOGY: THE CASE OF YELLOW FEVER AND MALARIA, 1888-1891.

Introduction

I showed in the last chapter that by 1887 the Colombian medical community had accepted the bacteriological hypothesis for yellow fever. Although it is true that this hypothesis changed people's ideas about the causes of yellow fever and even malaria, it is also the case that there developed several different accounts of the causes of those diseases that not only considered the bacteriological hypothesis but also incorporated elements of the pre-existing climatic perspective. In other words, Colombian physicians reconciled the germ hypothesis within the medical geographical framework of disease causation.

In the adaptation of the germ hypothesis to the medical geographical understanding of yellow fever in Colombia, several opinions were held that fell between the two extremes of the spectrum. At one end, there were physicians who held a rather static view of microorganisms and tended to believe that they, above any other environmental or individual condition, should be considered the main causal element of yellow fever; at the other end, there were physicians who held a more complex view of germs, whose characteristics they managed to reconcile with the established climatic determinism of that fever. In this chapter I will explain how and why these different versions developed by following three books: the medical thesis *Los microorganismos de la fiebre amarilla*, written by Daniel Gutierrez for the medical school in Bogotá (1888); the medical thesis *Contribution a l'étude de la fièvre du Magdalena* by Carlos Esguerra for the Paris faculty of medicine (1889); and, finally, the book on yellow fever, *La fiebre amarilla al interior de Colombia*, by Luis Cuervo (1891). Cuervo's book was not a student thesis. It was the result of several years of the author's medical practice in the warm lands. In fact, Cuervo's book is not only more complex than Gutierrez's and Esguerra's theses; unlike these

theses, it remained a relevant reference work on yellow fever until the twentieth century. I will analyze the three documents as a whole in order to show the complexities faced by Colombian physicians when reconciling the medical geographical and bacteriological views of fevers in Colombia in the late 1880s.

My analysis of these books also reveals that the adjustment of the germ hypothesis of yellow fever to the medical geographical framework affected people's ideas about malaria. The ongoing debate among the Colombian medical community over whether the nature of yellow fever was paludic or non-paludic explains why physicians also felt compelled to explain the implications of the germ hypothesis for the other Magdalena's fevers, which would increasingly be categorized as paludic fevers or malaria. Furthermore, it was precisely during these debates that yellow fever and malaria came to be seen for definite as two distinct diseases by the Colombian medical community. However, I am not saying that the germ hypothesis was the main reason why Colombian physicians made this distinction. For, as I mentioned in the last chapter, before any germ hypothesis was seriously considered for yellow fever, physicians had already distinguished between the paludic and the non-paludic fevers on epidemiological, clinical and therapeutic grounds; the microorganism hypothesis was just one among other phenomena in the consolidation of the distinction between those two fevers.

In summary, this chapter is about the way that Colombian doctors reconciled the medical geographical and bacteriological perspectives in the case of yellow fever and malaria; and also about the way that they consolidated the emerging distinction between these two diseases in the last quarter of the nineteenth century.

The radical bacteriological perspective of fevers

In 1888, Gabriel Gutierrez's thesis *Los microorganismos de la fiebre amarilla* was published at the medical school of Bogotá. The author's intention was to defend the hypothesis of the microbiological cause of yellow fever, following current developments on the subject in Latin America and France. Gutierrez partly based his thesis on his practice as an assistant at the histological and pathological laboratory of

the Faculty of Medicine at Bogotá, run by Gabriel Duran Borda and Proto Gómez.[272] Gutierrez's thesis is the best available example of the work of those who rejected a strict climatic perspective of disease (though not always successfully) and who sought to defend the view of germs as the only cause worthy of consideration.

Gutierrez organized his thesis around microorganisms rather than the conventional medical geographical topics. He began with a general description of microorganisms, followed by a large and detailed account of those microorganisms which had been proposed as the cause of yellow fever. A large part of his work was also devoted to the international and national controversy over the preventive inoculations against yellow fever, including the bacteriological experiments that he and his advisors performed in the laboratory in Bogotá, as I described in Chapter 2. Gutierrez concluded his thesis by drawing a parallel between yellow fever and the fevers of the Magdalena basin, using the bacteriological perspective.

Gutierrez was aware that the Colombian medical community had long been concerned with yellow fever as part of the debates on the nature of Magdalena's fevers and Cúcuta fevers, so he justified his work by claiming that not only yellow fever but also Magdalena and Cúcuta fevers required intensive study from the bacteriological standpoint. Indeed, at the beginning of his book, Gutierrez affirmed of those fevers that

> It is true that there are many studies by some of our distinguished doctors; however, they do not include descriptions of the intimate nature of those fevers, that is, the study of the micro-organisms that produced them; [for] as with other infectious and contagious diseases, those fevers have a parasite or virus as their cause.[273]

Thus, Gutierrez explicitly established a watershed between previous works on fevers - which had invariably been medical geographical works - and the new microbiological perspective. That is why he affirmed, for example, that "none of the causes that had been assigned to yellow fever" — he was implicitly referring to the

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[272] Daniel Gutiérrez Arango, *Los micro-organismos de la fiebre amarilla*, Tesis para el doctorado en Medicina y Cirugía (Bogotá, Imprenta El Telegrama, 1888). Gutiérrez identified Borda as "head of the microbiological laboratory of the *Universidad Nacional*." But as I showed in chapter 2, this was officially recognized as a laboratory of histology and micrography that supported lectures on pathological anatomy. In other words, official bacteriological analysis in Colombia seems to have been first performed in the anatomopathological laboratory of Bogota.

[273] Gutiérrez Arango, *Los micro-organismos de la fiebre amarilla*, p. 10.

miasmatic cause and the climatic determinism — "explained yellow fever distribution; because those other causes are common to diseases which developed in places where yellow fever never occurs."[274] Here, Gutierrez is criticizing the medical geographical idea of the local origin of yellow fever developed by Chevrin and Vargas Reyes, with the argument that the disease did not always develop under those local conditions. Since yellow fever was a disease that spread quickly by "contagion" and "infection", he concluded, it was obvious to look for the "ferment" or microscopic causal germ.[275] Thus, after a brief description of germs in general, Gutierrez focused on the microorganisms that had recently been claimed to cause yellow fever, and on their preventive inoculations.

According to Gutierrez, microorganisms were terrible enemies living in the atmosphere, water, and food. Once in the body, they would take their sustenance from blood, tissues, and even from food. Their voracity was so high, Gutierrez claimed, that they competed with cells for critical substances, and hence affected organic functions. Microorganisms, he added, sprouted like plant seeds, and found human bodies to be the perfect place to feed and reproduce. These "little beings" could suffer high temperatures or intense cold without effect on their destructive power or their fecundity, and their tiny size made it easy to travel from the places where they were produced. By appealing to the metaphor of plants and seeds, he explained that diseases were like plants that found the conditions for their development and reproduction in the human body, through seeds – the microorganisms - which spread among the population.[276]

With regards to the microorganisms causing yellow fever, Gutierrez described and criticized those reported by Manuel Carmona y Valle, Domingos Freire and other Latin American researchers as the specific cause of the disease. Following the criteria that Koch laid down for proving that a particular germ was the cause of a specific disease, Gutierrez claimed that Latin American physicians had not only failed to prove that those microorganisms could always be found in yellow fever patients, but had also failed to isolate them, and to reproduce the same disease by

[274] Ibid., p. 20.
[275] Ibid., pp. 19-20.
[276] Ibid., pp. 7-8; 19-20.

inoculating the isolated microorganisms.[277] One of the arguments Gutierrez used to dismiss the pathogen proposed by the Mexican doctor Carmona y Valle, for example, is that he, along with Duran Borda and Proto Gómez, had found the pathogen proposed by Carmona, the *Peronospora Lútea*, in the air of the Bogotá laboratory, a place where yellow fever was not expected to occur (see Chapter 2).

Having discarded these hypotheses, Gutierrez turned to French sources such as André-Victor Cornil and Victor Babes's textbook on medical bacteriology (1886) and Paul Gibier's analysis of the preventive inoculations – the latter ordered by the French government in 1888.[278] Cornil (1837-1908) was one of Pasteur's followers in the French medical school where he taught anatomopathology, and he was also the first to teach microbiology in his laboratory. He imparted the most recent data on bacteria from both the French and the German schools to his medical students; but as the historian Anne Marie Moulin notes, bench work was not available to them.[279] In their textbook, Cornil and Babes suggested that the microbe of yellow fever might be found in the intestines and that it should be similar to the one that produced typhoid fever.[280] Paul Gibier, on the other hand, reported to the French government that he had found bacteria similar to cholera microorganisms in the intestines of yellow fever patients, and hypothesized that these bacteria would produce a toxin that caused the general symptoms of yellow fever. Gibier claimed to have cultivated and inoculated the yellow fever microorganisms in guinea pigs, reproducing the symptoms of the disease. The young Colombian doctor Gutierrez saw, in Gibier's results, a confirmation of Cornil and Babes's suggestion that the yellow fever germ might be found in the intestines.[281]

The novelty of Gutierrez's view lay not only in this radical microbiological approach to yellow fever, or in his apparent rejection of any climatic or geographic factor as the first line of causal explanation, but also in the way he treated the old

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[277] Ibid., pp. 24-26, 46-47.
[278] I could access Cornil and Babes's book but not Gibier's report. A-V. Cornil et V. Babes, *Les bactéries et leur role dans l'anatomie et l'histologie pathologiques des maladies infectieuses* (Paris, Félix Alcan Editeur, 1886).
[279] Anne Marie Moulin, "Bacteriological Research and Medical Practice in and out of the Pastorian School" in Ann La Berge and Mordechai Feingold, *French Medical Culture in the Nineteenth Century* (Amsterdam/Atlanta GA, 1994), pp. 329-331.
[280] Cornil et Babes, *Les bactéries et leur role dans l'anatomie*, p. 529.
[281] Gutiérrez Arango, *Los micro-organismos de la fiebre amarilla*, pp. 51-5, 85.

problem of the Magdalena's fevers. In an apparent contradiction of his microbiological argument, he considered that yellow fever was a disease that only happened on the Caribbean coast; hence, he continued, fevers of the Magdalena river basin (generally known as Magdalena's fevers) should be of a different nature. In fact, in Gutierrez's view, they were two different diseases, though both caused by germs. In his view, and maybe in the view of those physicians who took the same approach to the bacteriological perspective as Borda and Gómez, previous studies on the Magdalena's fevers had failed to explain the "intimate nature" of those fevers, that is (as I mentioned above), "the micro-organism".[282] Gutierrez thus justified the extension of Gibier's hypothesis of yellow fever to the Magdalena's fevers. In yellow fever and Magdalena's fevers, he found clinical similarities, which he based not on clinical experience but instead on the comparison of descriptions of Magdalena's fevers by Colombian authors with descriptions of yellow fever by Berenger-Feraud (a French author who wrote in 1878 on the yellow fever of Martinique, one of the Antilles islands). Gutierrez found "significant similarities" in the descriptions, and, in his view, the French description reinforced the idea that there was alteration of the blood by a toxin in yellow fever. This fact fit perfectly with Gibier's hypothesis of toxins produced by intestinal microbes. Gutierrez reasoned that if intestinal microbes produced toxins that caused the typical symptoms of yellow fever, and Magdalena's fevers had similar symptoms to yellow fever, then different microbes in the intestine could produce Magdalena's fevers.[283] It was this theory, I believe, that made Gutierrez's thesis original.

We can reasonably wonder why Gutierrez did not postulate that Magdalena's fevers and yellow fever were variations of the same disease if they were so similar clinically and causally (they were both produced by intestinal microorganisms, whose differences he never postulated). Gutierrez argued that the two diseases were different, since Magdalena's fevers occurred in epidemics whereas yellow fever was usually endemic. But, most importantly, in Gutierrez's view yellow fever was a disease that could only develop near the sea, in areas of salty water with temperatures above 20 degrees Celsius.[284] In other words, the aspect that would differentiate

[282] Ibid., p. 10.
[283] Ibid., pp. 53, 83-86.
[284] Ibid., pp. 81-2.

yellow fever and Magdalena's fever was, paradoxically, a climatic and geographical element.

In conclusion, Daniel Gutierrez's thesis *Los microorganismos de la fiebre amarilla* was animated by one of the first bacteriological studies, performed within the histological and pathological laboratory of the faculty of Medicine of Bogotá in the late 1880s. Analysis that centered on the microbiological cause of a disease, as Gutierrez's thesis did, had no precedent in Colombian medical literature. It came out in a milieu where yellow fever had long been understood as part of the medical geographical tradition, against which Gutierrez was aware that he had to delimit the microbiological nature of yellow fever. So strong was this tradition that Juan de Dios Carrasquilla, the professor of the medical faculty who reviewed Gutierrez's thesis, rated it as an important contribution because it was devoted to the study of a disease that was "deeply modified by the influence of our climates," an idea that was actually in opposition to Gutierrez's main argument.[285]

Gutierrez's thesis, *Los Microorganismos de la fiebre amarilla*, can be considered one of the best examples of the way that bacteriology would radically transform the medical geographical understanding of yellow fever. In his view, the microbiological perspective invalidated the climatic arguments on two grounds: first, that yellow fever did not always occur in areas where the alleged geographical and climatic causes were present; and secondly, that research into the nature of any contagious and infectious disease should be based on bacteriological studies. Gutierrez's effort to extend the microbial hypothesis of yellow fever to the Magdalena's fevers led him to emphasize their clinical similarities and thus to postulate that if yellow fever were caused by an intestinal microbe, so must be Magdalena's fevers. But it also resulted in a contradiction: he differentiated these two diseases, paradoxically, on environmental – i.e. medical geographical - grounds.

Between the bacteriological and medical geographical perspectives

[285] Juan de Dios Carrasquilla, "La tesis para doctorado", *Revista Médica*, serie XII, No. 132, 20 diciembre, 1888, pp. 273-4.

It seems that Daniel Gutierrez's belief that microorganisms were the only determinant cause of yellow fever and Magdalena's fevers was exceptional among the Colombian medical community of the 1880s. The other two books that I will examine in this chapter were framed, either explicitly or implicitly, as medical geographical works, while at the same time conveying less radical views of microorganisms. In this section I will analyze the medical thesis *Contribution a l'étude de la fièvre du Magdalena* (1889),[286] by Carlos Esguerra (1863-1841), for the Paris medical school, published one year after Gutierrez's thesis. Carlos Esguerra had obtained his medical degree in Bogotá in 1881.[287] He had practiced medicine in Colombia, probably for several years, before studying in Paris. Most of the clinical observations that he used as evidence for his thesis for the Paris medical school were obtained in 1884, during the few months he spent working at the Hospital of Honda, a town located on the Magdalena River.

Esguerra's aim was to establish the nature of the Magdalena's fevers. Since this topic might have seemed strange to his Paris professors, Esguerra justified the use of this umbrella notion by claiming that it referred to the pathology of the Magdalena river region, whose nature was still contested. But instead of taking the bacteriological perspective of infectious diseases as the starting point, he built on the Colombian medical geographical tradition to which his uncle, Domingo Esguerra, had made an important contribution in the early 1870s (see chapter 1). Thus, Carlos Esguerra structured his thesis around medical geography, with a description of the geographical and geological characteristics of Honda, including clinical and historical accounts of the endemic and epidemics of the Magdalena's fevers. Esguerra was knowledgeable about microorganisms and, as I will show, managed to reconcile some of their characteristics with more typically medical geographical causal elements, including hygienic and individual conditions.

Thus, Esguerra first analyzed the endemic manifestations of Magdalena's fevers from the medical geographical perspective. He defined the endemic fevers as paludic fevers or malaria. Esguerra used "paludic" to mean not only a clinical cluster of

[286] Carlos Esguerra, *Contribution a l'étude de la fièvre du Magdalena* (Paris: Imprimerie des Écoles, 1889).
[287] Humberto Caceres y Zoilo Cuéllar-Montoya, *Academia Nacional de Medicina de Colombia. Sus Miembros* (Bogotá, Academia Nacional de Medicina, 1998), pp. 64-5.

symptoms but also the fact that these fevers were associated with certain geological, geographical and climatic characteristics of the warm lands. This association appears in Esguerra's work as a kind of functional relationship between altitude and paludic fevers, typical of medical geographical studies. The Magdalena valley was formed, according to Esguerra, by low and high regions, which therefore had different kinds of organic life as well as particular diseases. According to Esguerra's description, the lower lands were favorable for the production of endemic malaria: they were humid and fertile; their geology consisted of alluvial deposits and chalky, clayey soil which was impermeable to water; and an overlying layer of humus fostered the spontaneous growth of trees and plants. Temperatures of around 28 to 32 degrees Celsius also contributed to malaria's development. This endemic malaria would decrease, according to Esguerra, as one moved up towards higher zones. Unlike the lower areas, the highlands had sandy and volcanic soils and a dry and cold climate which not only made them less fertile but also gave them a "different medical constitution" that was not favorable for malaria.[288]

In addition to this functional relationship between altitude, vegetal life and diseases, Esguerra also associated rain and heat with clinical types of paludic fevers. During the last months of 1884 in Honda, he observed that when it rained, all kinds of intermittent, remittent and continuous fevers occurred; when the rain stopped and was followed by a wave of heat, they tended to disappear, only to give way to the intermittent bilious fever.[289]

The heir to the medical geographical tradition, Esguerra also considered "physiological" variables that could contribute to the production of the disease. Two kinds of people seemed to be more susceptible to malaria: those who had recently arrived in warm climates, whose gastric activity and bilious production increased as a consequence of moving to the new area; and the chronic sufferers of malaria, whose constitution predisposed them to similar physiological and anatomical alterations of the digestive organs and liver, sometimes defined as "bilious temperament". [290]

To sum up, Esguerra argued on medical geographical grounds that the endemic Magdalena's fevers were paludic fevers or malaria. But what about the other

[288] Ibid., pp. 9-10.
[289] Ibid., pp. 10-12.
[290] Ibid. p. 21.

Magdalena's fevers, the epidemics? Were they *recrudesencias* of malaria, or were they a different fever? Esguerra began to answer this question by making a distinction between the rapid increases of malaria cases (*recrudesencias*) and "the fever of the epidemics", that is, the non-malaric fevers. However, he said, although they were two different infectious diseases, they had so many symptoms in common that it was sometimes impossible to distinguish between them clinically. They overlapped:

> From the description I have made of the short epidemic of Honda, one can be convinced that there is a continuous gradation from the endemic fevers to the epidemic forms, from the intermittent and remittent simple types of fevers to the grave remittent forms, the bilious remittent, and from this point to the epidemic fever without being able to acknowledge when the former ends and the latter begins.[291]

Despite this overlap between the epidemic and endemic fevers, Esguerra tried to gather groups of symptoms that might help to differentiate them. Thus, malaria *recrudescencia* would start slowly, with pains, vomiting, occasional hemorrhages through the nose and intestine, and no remissions of the fever. The "fever of the epidemics" – or yellow fever - had, on the contrary, two phases: one characterized by a quick rise in fever, strong pulse, pain, and vomiting which would last three days, followed by a short remission, and the second a period of jaundice and hemorrhages. But even though they were clinically distinguishable, Esguerra argued, malaria and yellow fever influenced each other and produced intermediary or transitional forms, which contributed to the difficulties in their diagnosis. [292]

Microorganisms played a very marginal role in Esguerra's distinction between yellow fever and paludic fevers. Esguerra placed malaria and yellow fever among the infectious diseases, that is, those caused by "infectious agents". For malaria, he recognized, there was an "*élément animé, microbe, bactérie, peut importe, cause de la malaria*"; but he immediately added that "no one could deny the telluric origin of this infection."[293] For yellow fever, he said that "even though one can recognize the

[291] Ibid., pp. 43-5.
[292] Ibid., pp. 18-22, 48-52, 124-5.
[293] Ibid., pp. 76, 81.

telluric influences in the origin of yellow fever … I place it better among the *typhique* diseases," in which, in his view, hygienic conditions were crucial.[294]

Before I analyze how Esguerra understood the role of germs and hygiene in the cause of the *typhique* disease yellow fever, it is important to remember that at the time that he published his thesis, there was no consensus on the location of yellow fever among the Colombian medical community. There was disagreement over whether the disease was typical of the coastlines or could also occur along the Magdalena River and the slopes of the *cordilleras*. I showed in the first section of this chapter that Daniel Gutierrez, for example, was convinced that yellow fever could only occur near the sea, along the coastlines. This contrasts with Esguerra's opinion, according to which yellow fever could be imported to the Magdalena riverbanks and even to the *cordilleras* from the Caribbean coast by boats and railways. Relying on medical geographical arguments, Esguerra claimed that the "infectious agent of malaria", contrary to the yellow fever agent, would not be able to climb up from the Magdalena valley to the slopes of the eastern *cordillera*, since they were places of non-paludic nature.[295]

The truly relevant point here is Esguerra's argument that the presence of the yellow fever agent was not enough, by itself, to cause the disease. Indeed, the people and hygienic conditions had to be ready before the agent could develop.[296] Esguerra found some truth in the popular opinion of the inhabitants of Honda, who blamed the Guali River (whose mouth is in the Magdalena River) for the yellow fever epidemics. The Guali River flows through Honda and people used to throw the waste and filth of the city into it, thus forming "sources of infection" where the imported "germs" of typhoid fevers were preserved. Other breeding grounds of the "typhoid" yellow fever germ could be found, according to Esguerra, in stores of leather and other putrefactive matter, public buildings like hospitals and barracks, and even the interiors of houses.[297]

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[294] Esguerra believed that yellow fever was the representative, in certain tropical climates, of the *typhus exantematique* of cold climates; therefore, he found it justifiable to use the name "typhus of America" or "typhus Amaril". Esguerra, *Contribution a L'étude*, p. 131.
[295] Ibid., pp. 128-9.
[296] This fits with Esguerra's idea that Cúcuta's epidemics were of yellow fever, imported from the Caribbean port of Maracaibo, in Venezuela, via the Zulia River. See Esguerra, *Contribution a L'étude*, pp. 134, 137.
[297] Ibid., pp. 54, 130.

Although Esguerra recognized that the "infectious agent" of yellow fever had the property of reproduction and transmission, he insisted that the agent had to be imported. Most importantly, he argued that, as in other "typhoid" diseases, if the agent did not find the right hygienic conditions to provide "soil for its culture", yellow fever would not develop. Boats or railways did not merely act as vehicles that brought the infectious agent to the Magdalena River or the villages in the *cordilleras*; Esguerra declared that those means of transport actively produced yellow fever by causing the hygienic conditions of towns to deteriorate, and hence "adapting their milieu to the needs of the infectious agents".[298] The variation of those hygienic conditions would explain, for example, why some ports that were visited by boats with yellow fever avoided infection.[299]

In conclusion, Carlos Esguerra took the medical geographical point of view in his analysis of the Magdalena's fevers. He differentiated between malaria and yellow fever based on not only clinical characteristics but also medical geographical and hygienic grounds. He accepted that "infectious agents" were part of the cause of both diseases, and at the same time reconciled this causal element with medical geographical ones. That is why, in Esguerra's scheme, the causal "agents" could not alone determine the specificity of malaria or yellow fever: malaria was a "telluric" disease, whereas yellow fever depended upon the maintenance of the appropriate "culture" for the imported agent, that is, the bad hygienic conditions of the city.

I mentioned that Carlos Esguerra wrote his thesis for the Paris medical school. He did so when Pasteurism had reached a certain acceptance among the clinicians of said school.[300] The idea of an "infectious agent" that needed an appropriate "culture" to develop was indeed a Pasteurian notion. Interestingly, Esguerra did not consider any of the yellow fever parasites or microorganisms proposed by Latin American and French doctors during the early 1880s in his analysis, nor did he discuss the prophylactic inoculations applied in Colombia in 1887. It seemed as if Esguerra did

[298] Ibid., p. 139.
[299] Ibid., pp. 128-130, 138-139.
[300] According to Moulin, except for Peter, the four chairs of internal medicine at the Paris medical school were occupied by Pasteurians by 1886. Other Pasteur followers were Brouardel, the dean of the medical school; Cornil, the professor of anatomopathology; Chantemese, the professor of comparative medicine; and Grancher, the chair of pediatrics. Esguerra's advisor was Paul Georges Dieulafoy, who replaced Peter in 1886. I do not know Dieulafoy's position with regards to Pasteurism. Moulin, "Bacteriological Research and Medical Practice", p. 329.

not know about this Pasteurian interpretation of yellow fever, despite having listed Gabriel Gutierrez's thesis, where there was an exhaustive account of this subject, in his bibliography. Esguerra either deliberately ignored that discussion or simply did not know of it. Whatever the case, Esguerra's book can be seen as an example of how a medical geographical-minded physician from Colombia understood the germ hypothesis from within the Paris medical school in the early years of the diffusion of Pasteurism.

Bacteriology at the core of medical geography

In 1891 Luis Cuervo Marquez's book, *La fiebre amarilla en el interior de Colombia*, was published.[301] In contrast with the two medical theses that I have analyzed so far, this book was based upon several years of medical practice by the author in some of the warmest regions of Colombia. Indeed, after Cuervo graduated from the Bogotá medical school in 1884, he moved to Cúcuta, where he practiced medicine for at least four years, including the year of the controversy over prophylactic inoculations for yellow fever. Cuervo also spent eight months as an army physician, crossing the Magdalena and the Caribbean coast during one of the civil wars and spending several weeks in Ocaña and some of the Caribbean cities.[302]

Cuervo had been interested in fevers before the appearance of his 1891 book. In 1886, he had addressed the problem of the identity of fevers when the germ hypothesis for yellow fever was not yet seriously considered by the Colombian medical community. His aim, during that year, was to explain why Magdalena's fevers were climbing the Eastern *cordillera* and causing epidemics along the way to Bogotá. Cuervo suggested that those fevers were non-paludic fevers, different from the paludic fevers of the Magdalena River. In Cuervo's view, these non-paludic fevers were of typhoid nature but were not yellow fever (also considered to be of the typhoid group). His reasoning was that yellow fever could only occur along the coastlines. But Cuervo soon became convinced that yellow fever could occur in the

[301] Cuervo Márquez, *La fiebre amarilla.*.
[302] Cuervo Márquez, *La fiebre amarilla*, p. 3.

interior of the country after his experience with the 1887 epidemic of Cúcuta (see Chapter 2). This was precisely the subject of his 1891 book: to explain why and how it was possible for yellow fever to occur and persist in the *interior* of the country, that is, along the Magdalena river banks and in Cúcuta. This time, Cuervo aligned himself with both Pasteurian bacteriology and immunology and the medical geographical framework.

The problem of yellow fever in Cúcuta

As I said above, the main purpose of Cuervo's book was to explain why and how it was possible for yellow fever to occur and persist in the *interior* of the country. He quickly summarized the history of yellow fever overseas in order to set up the argument about its origin in the Antilles basin, and then went on to detail how the epidemics moved to the interior.

Cuervo's premise was that "yellow fever is native to the Antilles sea basin, outside of which it only develops via importation". So the disease might have remained in a latent state there, he said, until susceptible individuals arrived. That was the case with the European conquerors that first arrived in the Antilles in the sixteenth century. From the Caribbean breeding ground, yellow fever would have then extended to the northern and southern American coastlines following the expansion of commerce during the next centuries. By the end of the eighteenth century, the disease would not only have spread to European coastlines, but also to the interior of the continents. Rivers would have been the main gateway for the disease, provided that there were susceptible people along the way.[303] In Colombia, the history of yellow fever ran along the same lines, according to Cuervo. Thus, it first occurred in the Caribbean ports of Cartagena and Santa Marta, imported from Portobello in 1729.[304] By the late eighteenth century the disease would have attacked other cities like Colon and Panama.[305] These maritime sources would have been the

[303] Ibid., pp. 6-16.
[304] "Although," Cuervo said, "it is natural to suppose that it had already existed in all of our cities on the Atlantic Coast". Europeans arriving in Cartagena for the first time during the sixteenth century would have been attacked by a disease "that was special to them" called *La Chapetonada* (derived from the word *chapetón*, used to refer to Spanish people during the Colonial years). According to Cuervo Márquez, that name was still used in some Colombian towns to refer to yellow fever and malaria. See Cuervo Márquez, *La fiebre amarilla*, p. 19.
[305] Colon and Panama City were located in Panama, which belonged to Colombia until 1903.

point of departure of the epidemics into the interior of the country, via the Magdalena River and the Zulia River, in the nineteenth century. [306]

According to Cuervo, once the maritime regions were affected by yellow fever, they became a constant source of the disease; the fever stayed in a latent state, waiting for "non-acclimated" people to arrive.[307] That was the case with the Colombian Atlantic port of Cartagena. However, Cuervo wondered if riverbanks could also become permanent sources of yellow fever. Cuervo thus analyzed in detail whether Honda, the village in the Magdalena River, and Cúcuta, located near the River Zulia, became "continuous sources of the [yellow fever] infection" or if yellow fever "need[ed] to be imported from the coastal lines". [308] This is the puzzle to which his book is devoted.

Honda was the main intermediary port for goods that traveled between the Atlantic world and the inland regions in the Andes Mountains via the Magdalena River. Cuervo claimed that Honda "cannot be considered as a permanent source of yellow fever", since there had always been a coastal source for the epidemics (1856/7, 1865, 1880 and 1888) and for other related circumstances, such as the arrival of soldiers from the Atlantic coast in the 1885 civil war. Cuervo himself had witnessed the development of the fever after its importation in 1888. Soon after he arrived from the coast to Honda by steamboat, another steamboat arrived from Cartagena carrying a woman with yellow fever. She died in the Honda hospital, and a few days later the disease spread through the hospital and then to the whole town. In conclusion, in Cuervo's view, cases of yellow fever in Honda had always occurred due to the importation of a "morbid agent", and lasted until that agent was "exhausted". This idea of the "exhaustion" of the agent is a bacteriological notion, loosely used here to explain the course of yellow fever.[309]

Although, in Honda, yellow fever came and left following the pattern of importation and exhaustion of the "morbid agent", the dynamic of the disease in Cúcuta seemed much more complex to Cuervo. As I showed in Chapter 2, Cúcuta was located on the Colombian border with Venezuela and had recently been

[306] Cuervo Márquez, *La fiebre amarilla*, pp.7-21.
[307] Ibid., pp. 18, 21.
[308] Ibid., p. 25.
[309] Ibid., pp. 25-6.

connected to the Zulia River by the railway built between 1879 and 1888. Cuervo agreed with Aristides Salgado, the envoy sent by the JCH to study the 1889 epidemic,[310] that the disease was first imported in 1883 from the Caribbean port of Maracaibo (in Venezuela), via the Zulia River. But contrary to Salgado's view, Cuervo believed that the second epidemic of 1886 occurred "without any trace of a new importation of the disease", as if "the yellowish agent had not weakened" since its 1883 importation and had instead stayed in a latent state until "unknown causes that favor its development" caused it to reappear in epidemic or endemic form (1886 and 1889). In other words, Cuervo claimed, "Cúcuta has been a permanent source of yellow fever for some time". In his search for an explanation for the persistence of yellow fever in Cúcuta without the need for importation, Cuervo struggled to reconcile the medical geographical and bacteriological perspectives.[311] I will now look more closely at Cuervo's analysis of the Cúcuta epidemics in order to highlight his struggle to bring these two models together.

Basing his argument on the experience of the local physician Elias Estrada, who had been working in the city for decades, Cuervo explained that yellow fever had never occurred in Cúcuta before 1882. Estrada could not have mistaken the diagnosis, declared Cuervo, since he "was familiar with yellow fever of the Antilles and Maracaibo". The fever appeared in December 1882. Cuervo summarized several medical geographical hypotheses that had circulated in Cúcuta according to which yellow fever developed "*in situ* under the influence of local causes". These alleged local circumstances were the 1875 earthquake, winds, and the construction of railways. According to the first explanation, the 1875 earthquake's casualties (about 1,000 animals and 2,000 people) produced putrid emanations that would have generated the disease. However, Cuervo reasoned that since the earthquake had occurred eight years before the first epidemic episode, yellow fever would have appeared much sooner if those emanations were really the cause. As for the winds hypothesis (the theory that the lack and inconstancy of the healthy southern winds which kept the air clean was what caused the epidemics), Cuervo partially accepted it, but reflected that if it were the only cause, the fever should have disappeared soon after the winds arrived again. Finally, of the railway works hypothesis (according to

[310] See chapter 2.
[311] Cuervo Márquez, *La fiebre amarilla*, p. 27.

which the removal of soil for the construction of the railway caused the fever), Cuervo observed that when the first epidemic appeared, work on the railway was still far away from the city, while construction had not been needed near the city excavations because the region was flat. [312]

Once he discarded these "localist" or medical geographical hypotheses, Cuervo developed his arguments in the direction of the microbiological hypothesis. He was told that the first casualty in Cúcuta was a photographer, a native of the *cordilleras* of the Andes, who visited the coastal city of Maracaibo in Venezuela in 1882 and traveled via the Zulia River to Cúcuta "in only four days". He fell ill a few days later with fever, headaches, pain, hemorrhages, and jaundice. The nurse who attended him also died of the same symptoms. Although people initially diagnosed these cases as a strange form of paludism, the deaths of 213 locals and many foreigners of a similar fever convinced people that it was yellow fever.[313] For Cuervo, this story demonstrated that yellow fever did not develop spontaneously in Cúcuta, but that a "morbid element" was instead imported from Maracaibo. Interestingly, Cuervo added that this element was transported quickly enough by the recently established steamboat and railway transport not to lose its "virulence"; and that the agent successfully established itself since it found the "most powerful transmitting agent" in the human body.[314]

It is evident that up to this point Cuervo had been increasingly introducing bacteriological notions of yellow fever into his arguments, and had also discarded medical geographical arguments concerning the local origin of yellow fever. But he still struggled to reconcile both perspectives. He affirmed that some climatic elements acted as "precursors" of the 1882 epidemic and as facilitators of the propagation and multiplication of the "specific" agent during that epidemic and the ones that followed. He described a dynamic of unhealthy and healthy winds which he associated with the presence or absence of yellow fever and malaria. The northern unhealthy breezes, he said, came from the hot and unhealthy lands of the Zulia, Catatumbo and Sardinata Rivers. The valleys of those rivers had exuberant vegetation which exhaled "aerial lakes" or "aerial marshes rich in infectious germs",

[312] Ibid., p. 46-7.
[313] Ibid., pp. 47, 51-52.
[314] Ibid., pp. 47-48.

acting upon the body as actual swamps and terrestrial lakes do. When the northern breezes came to Cúcuta, saturated with these "miasmas", infectious diseases like yellow fever and paludic diseases thus predominated.

The southern winds would come to balance the negative effects of the northern breezes: they came from the "healthier" lands of the *cordilleras* and renovated the air by mechanically pushing not only the unhealthy elements brought by the northern breezes but also the "products of decomposition that vitiate the atmosphere" towards the north. Thus, when those healthy winds dominated, yellow fever and paludism would tend to disappear. For Cuervo, the weakening of these healthy southern winds was both the "precursor" to the yellow fever epidemics and a vehicle that spread the agent.[315]

Even though Cuervo considered that winds had a key role in spreading yellow fever over short distances like rooms, houses, neighborhoods and cities, he argued at length that yellow fever was a contagious disease. The disease, he affirmed, was mainly transmitted by the stools of the sick person as with typhoid fever, but also through objects that belonged to the ill person or through merchandise.[316] He did not clarify how the contagion and winds would work together to spread the disease.

Finally, Cuervo also considered that there may have been other atmospheric changes that played a role in causing yellow fever, whose effects could only have been noticed indirectly via the simultaneous occurrence of other diseases such as dengue or chicken cholera-like diseases in animals, or the increase in the number of mosquitoes.[317] The idea of the simultaneity of different epidemics or epizootics during a particular atmospheric state was a medical geographical notion, as I showed in chapter 1.

In conclusion, Cuervo considered that the first epidemic of Cúcuta of 1882 was caused by a microorganism imported from Maracaibo, but that there were also some preceding climatic changes in the prevailing winds and the local medical constitution that helped to account for the development of the disease.

Cases of fever occurred in Cúcuta up until 1884. In his explanation of the reasons why yellow fever disappeared in 1885, Cuervo's effort to reconcile the

[315] Ibid., pp. 37-8, 42-5, 47-8.
[316] Ibid., pp. 83, 87, 89-91.
[317] Ibid., pp. 42-5.

bacteriological and the medical geographical perspectives is again evident. Cúcuta was the center of military activities during that year, he said, and the city hosted a large number of troops from the cold climates of Cundinamarca, Santander and Boyacá. Cuervo was convinced that people from the highlands were more susceptible to yellow fever than lowlanders. How was it, he wondered, that there were no cases of yellow fever during that year? Cuervo pointed out that even though the troops were living under dreadful hygienic conditions such as poor ventilation, and were exposed to sudden changes of temperature and bad nourishment, yellow fever did not develop. The reason, he said, "is out of our means of research",[318] but he hinted at two possible explanations, one climatic, the other bacteriological. The first – which he applied not directly to Cúcuta but to a neighboring village - was the persistence of the southern healthy winds, which might have cleaned the air of the contagious agent. The second was that the morbid agent became "attenuated" or "exhausted" in Cúcuta.[319] Unfortunately Cuervo had still not offered any definite answer, but it is clear that up to this point his argument included two key characteristics of germ theory: virulence and attenuation.

The second epidemic (1885-7), the subsequent cases of endemic or sporadic fevers, and the third epidemic (1889) represented the real enigma, for according to Cuervo there had not been any evidence of a new importation of the infectious agent during any of those episodes. The second epidemic lasted from December 1885 to September 1886, and the disease remained endemic until May 1887. In the following years, periods with no cases were interspersed with periods with sudden cases, despite the fact that people from cold lands went down to Cúcuta continuously. For this reason, Cuervo decided that in Cúcuta the disease had not disappeared; that after its first importation in 1883 it remained there for several years, waiting for susceptible individuals to arrive.[320] This was a serious problem as Cuervo had assumed that yellow fever, being a disease native to the Antilles, could only exist permanently in the coastlines; why, he wondered, did Cúcuta, located far from the seaside, become a lasting source of the disease after it was first imported in 1883? As I showed above, Cuervo pointed to the fact that the disease occurred after some

[318] Ibid., p. 54.
[319] Ibid., pp. 54-55.
[320] Ibid., p. 59.

"precursor" climatic changes, including the weakening of the healthy southern winds and a particular atmospheric constitution. But he also suggested that germs could gain virulence or attenuate. Cuervo gave a definite and complete answer to the question by building a "causal triad" scheme for explaining infectious diseases.

The causal triad

Cuervo affirmed that any etiological conception of any infectious disease had to include the triad formed by "the morbid agent", the "favorable soil for its development" (meaning the ethnic and individual constitution of the local people), and "the external milieu" or "cosmic" influences which facilitated the spread of the disease.[321] Cuervo explicitly wanted to distance himself from the more restrictive bacteriological perspectives held in Colombia, for instance, by Daniel Gutierrez, who rejected the climatic arguments typical of medical geography, and in France by Pasteurians in the Paris medical school such as Cornil and Babes, whose work Cuervo knew. These French authors, referring to the *terrain* or "soil" to indicate the hygienic and environmental conditions that predispose to diseases, affirmed for example that:

> It is true that one has to take into account the question of the soil … the acclimatization, the overcrowding, and all the vices of hygiene that predispose individuals to be easily impregnated by morbid germs with which they come in contact. But it is not less true that the determinant initial agent, the fertilizing germ, comes from outside, and that *the question of soil is a secondary one*.[322]

Cuervo, on the contrary, considered that "the study of the causes that without motive have been called *banal* is as important as the study of the specific agent that produces these diseases".[323] Consequently, Cuervo summarized the current theories

[321] Ibid., p. 60.
[322] The original text says: "*Il et bien certain qu'il faut tenir compte de la question du terrain, des conditions d'inferiorité, d'inégale résistance ou se trouvent certaines espèces animales, et en pathologie humaine, de l'acclimatement, de l'encomrement, de tous les vices de l'hygiène qui prédisposent les individus à être imprégnées plus facilement des germes morbides avec lesquels ils se trouvent en contact. Mais il n'en est pas moins vrai que l'agent initial, déterminant, le germ fécondant, vient du dehors, et que la question du terrain est secondaire*." in A-V. Cornil et V. Babes, *Les bactéries et leur role dans l'anatomie et l'histologie pathologiques des maladies infectieuses* (Paris: Félix Alcan Editeur, 1886), p. 10.
[323] Cuervo Márquez, *La fiebre amarilla*, p. 60.

of the causal triad (agent, individual, and environment) for yellow fever with the intention of providing an answer to the question of the persistence of the disease in Cúcuta. In this effort, Cuervo struggled to attach equal weight to each of these three causal elements.

There were four theories about the "morbid agent" of yellow fever, according to Cuervo: malarial, microbial, chemical and toxaemic.[324] The malarial theory, which defined yellow fever as one of the most severe forms of paludic fever, had been discredited in Europe since 1860 and in Colombia since the early 1880s, as I showed in Chapter 2. Cuervo simply reiterated the arguments against the idea that yellow fever was a malarial fever: that yellow fever occurred away from any malarial influence or paludic soils; that yellow fever's symptoms belonged to the group of typhoid fevers; that malaria and yellow fever had different anatomopathological lesions; and, finally, that they responded differently to quinine.[325]

Cuervo agreed with the microbial theory of yellow fever. He was acquainted with the Latin American research that had proposed various germs since the early 1880s. Cuervo himself had made microscopic observations of various bodily fluids and organs of yellow fever patients during the inoculations of *Peronospora lútea* in Cúcuta in 1887, as I showed. He claimed to have found microorganisms in the intestine, as well as micrococcus in the livers and kidneys of corpses. But in his view, none of those microorganisms could be considered as the cause of yellow fever unless the requirements for a microorganism's pathogen status were accomplished: constant and exclusive presence of the microorganism either in the ill person or the corpse; pure culture of the microbe; reproduction of the disease with the isolated microorganism; and, finally, the verification that the inoculated microbe has been produced. Cuervo took these norms from Robert Koch's communication to the International Medical Congress of Berlin of 4-9 August 1890. Cuervo not only transcribed those rules but also Koch's idea that "there are certain bacteria in which those proofs cannot be found at once: for example, there are cases in which the inoculation of pure cultures into animals do not reproduce the disease, yet one can not deny that they are the cause of the disease in question." That was the case,

[324] Ibid., p. 60.
[325] Ibid., p. 61.

continued Koch, with leprosy, cholera, typhoid fever and diphtheria. Cuervo suggested that similar arguments could be applied to yellow fever. [326]

According to Cuervo, as a reaction to the premature and exaggerated extension of the microbial doctrines to yellow fever, some physicians reverted to the chemical theory based on the analogy between the symptoms of yellow fever and those of mineral or animal poisoning. He followed Armand Marie Corre, in his *Traité des fièvres bilieuses et typhiques des pays chauds, etc* (1883), in this argument. For Corre, symptoms produced both by snake venoms and by yellow fever, such as the tendency towards hemorrhages and jaundice, were sufficient arguments for assuming a chemical cause for yellow fever. Cuervo argued, however, that this theory explained neither the transmission of the disease over long distances nor the development of epidemics; in short, this theory was unacceptable for a transmissible disease.[327]

Cuervo finally considered the toxemic theory, according to which the immediate cause of yellow fever was not microbes but rather their secretions. Cuervo took this idea from Charles Bouchard's essay on a theory of infection presented at the Berlin congress of 1890. According to Bouchard, microbial secretions blocked the center that controlled the dilatation of small blood vessels, thereby halting exudation and *diapedesis* and thus phagocytosis, the foundation of cellular immunity. This condition left microorganisms free to grow and secrete toxic substances that would act against the organism. At the same time, said Bouchard, microbes would secrete a vaccine-like substance, producing a bactericidal state which would debilitate microorganisms and reverse the process mentioned above. Bouchard was trying to offer an intermediate solution between the humoral and cellular theories of immunity (from the French and the German school respectively) by stating that both mechanisms supported each other.[328] Cuervo agreed with Bouchard, but considered that there were diseases in which only one of the two mechanisms seemed to work. Cuervo affirmed that "the theories of infection [cellular] and of vaccine [humoral] should not be generalized to all diseases, as we cannot say that the microbial theory

[326] Ibid., pp. 62-66.
[327] Ibid., p. 67.
[328] Anne Marie Moulin, *Le dernier langage de la médicine: histoire de l'immunologie de Pasteur au sida* (Paris: Presses universitaires de France, 1991), p. 69.

on its own explains all infections". Cuervo was against blunt generalizations; he believed that it would be very premature to apply the toxemic theory to yellow fever, since there was no demonstration of the actual microorganisms of yellow fever, nor of any specific *leucomainas* associated to it. [329]

In conclusion, the first element of the causal triad of the infectious disease yellow fever, the "morbid agent", was, in Cuervo's opinion, a microbe. Despite his doubts with regards to the toxemic hypothesis, Cuervo believed that the yellow fever microbe would act through its toxins. Its identity was still unknown, but it was probably similar to those of yellow fever's counterparts in the typhoid group.[330]

The second element of Cuervo's causal triad of infectious diseases and hence of yellow fever was the "favorable soil" or individual influences. Without providing much detail, Cuervo described some of the old individual predisposing causes such as constitutions, temperaments and certain behaviors as associated with susceptibility to yellow fever.[331] He also analyzed the medical geographical idea of acclimatization and the new idea of immunity. Here, again, we find tensions between the medical geographical and bacteriological perspectives.

Cuervo defined acclimatization as the adaptation of organisms to the environment in which they developed; it was the property by which an individual became immune to a disease just by having lived in an infested place. Cuervo translated this property in terms of the emergent field of immunology. Immunity to yellow fever could be inherited, or acquired by either growing up in an infested place (acclimatization) or surviving a previous attack of the disease, he said. Immunity that was acquired by the second mechanism, that is, by growing up in an infested place, Cuervo explained, was produced by a slow poisoning which did not produce any observable reaction but whose prophylactic results were the same as those of acute poisoning. The biological mechanism supporting acclimatization, he suggested, could be the cellular immunity proposed by Élie Metchnikoff, the Russian zoologist

[329] Cuervo Márquez, *La fiebre amarilla*, p. 67-9.
[330] Ibid., pp.70-1.
[331] He affirmed that individuals with robust and plethoric constitutions were more disposed to contract yellow fever than the *diatesicos* or *caquecticos*. Sexual excess was a strong "determinant" cause among foreigners, since "it is obvious that such a strong cause of weakening in organisms allows the entrance of the exterior morbid agents". Yellow fever being a contagious disease, professionals such as doctors, priests and nurses were among the most affected. See Cuervo Márquez, *La fiebre amarilla*, pp.73-4.

and microbiologist from the Pasteur Institute. White blood cells would not only destroy bacteria under determined circumstances, but were also in the habit of destroying bacteria of a similar genre; it is precisely because of this habit that the base of immunity was acquired by growing up in an infested place, that is, becoming acclimatized. Cuervo supposed that organisms during childhood were more ductile than those of adults, and therefore the impressions received during that period would produce a permanent organic reaction of defense against the diseases of that particular climate. In addition, this resistance could somehow be transmitted by heredity to further generations. Foreigners would never be acclimatized in this way, and therefore, the only way for them to develop resistance to yellow fever would be via acquired immunity.[332] Thus, Cuervo selected and incorporated the "theory of phagocytosis" by Metchnikoff to explain the problem of acclimatization, that is, the resistance of natives to yellow fever, a clinical fact that was already common knowledge.

Cuervo paid less attention to the other three theories of immunity: the theory of "*contraveneno*" by Chauveau, the theory of "exhaustion" by Pasteur, and the "toxemic" theory by Bouchard that was already examined. In his theory of "contraveneno", Chauveau proposed the idea that while microbes developed in organisms, other substances were produced that made the organism resistant to similar microorganisms; Cuervo objected, on the grounds that not only had this theory not been proved but the fact was that organisms usually eliminated all waste products of cellular life. The "theory of exhaustion" by Pasteur, said Cuervo, was based on the observation that substances that had been successfully used for cultivating microbes became useless for second cultures; if applied to the body, it meant that when the pathogenic microbe consumed bodily substances, new microorganisms would not survive, thus preventing a second attack of the disease. Cuervo did not agree with this theory, since "it has the inconvenience of assimilating human organisms into vials with culture media." Cuervo opined that none of these theories, including that of phagocytes by Metchnikoff and the toxemic theory by Bouchard, offered a completely satisfactory answer to the problem of immunity.[333]

[332] Cuervo Márquez, *La fiebre amarilla*, pp.74-9.
[333] Ibid., pp.77-9.

Cuervo's explanation of acclimatization and natural immunity was contrary to some French ideas that had linked resistance or susceptibility to yellow fever with race. Susceptibility to yellow fever, declared Cuervo, was inversely related to the temperature of the place of origin of the individual and not to the whiteness of their skin. For example, black people were more resistant to yellow fever not because they were black but because they worked and lived in the hotter lands of the coastlines or river banks, thus becoming acclimatized through phagocytosis; any black person from the interior of the country would be as susceptible to the disease as a white person from the same region. Some indigenous races of America like the Guajiro Indians, who had tanned skin color – somewhere between white and black - were even more susceptible to yellow fever than whites because they lived in a cold climate, in the hills of the *Serrania* of the Guajira peninsula. A similar argument explained why, according to Cuervo, natives of the *cordillera* that came down from the *Altiplano* to Cúcuta and to the villages of the Magdalena River were defenseless against yellow fever despite their dark skin color. [334]

These opinions regarding the relationship between climate and race suggest that Cuervo was drifting away from one of the main medical geographical arguments held by the Colombian doctor, Antonio Vargas Reyes, since the 1860s. As I showed in Chapter 1, Vargas Reyes took the idea of a functional relationship historically created between local climates and plant distribution (transformationism) from plant geography and botany, and extended it to explain differences in races and diseases. Cuervo, although he accepted the influence of climate on disease production, did not link this influence to a wider theory of transformation of species; rather he reconciled it with the bacteriological explanations of acquired and natural immunity developed in France.

In conclusion, the second element of the causal triad, the "favorable soil" of yellow fever, meant (in Cuervo's work) certain characteristics of the individual constitution that could favor or halt the development of yellow fever; but a more relevant point is the way that he reconciled the medical geographical problem of acclimatization for locals with the cellular theory of immunity .

[334] Ibid., pp.71-4.

The third and last element of the causal triad in Cuervo's perspective on infectious diseases and thus of yellow fever was the "external milieu". As I mentioned above, Cuervo was convinced that winds were precursors of the yellow fever epidemics. Their influence was indisputable in his view. However, he recognized that "winds or the absence of wind are not the primary cause of fever; they are elements of *secondary order* which facilitate or halt the diffusion of fever in a locality in which there *already exists* an infectious germ".[335] Here Cuervo explicitly expresses that there is a subordination of climatic elements to the first element of the causal triad, the agent. However, climate could modify the agent. For example, the popular observation that the probability of acquiring yellow fever was higher in the mornings and in the evenings was explained, according to Cuervo, by the bactericidal power of sunlight. Most importantly, he drew an analogy between the influence of culture media upon the vitality and morphology of bacteria and the influence of climate on diseases. Cuervo affirmed that the influence of a culture medium upon the vitality and morphology of bacteria was an evident fact: Pasteur had demonstrated that an attenuated bacterium can recover its virulence when cultured in an appropriate milieu; Toussaint had demonstrated how heat attenuated the anthrax virus; and Pasteur had showed the modifications of the same microorganism under 40 degrees Celsius. That being the case, continued Cuervo, "can it not be believed that under the influence of meteorological or telluric changes, present in the majority of epidemics, the yellow fever agent can attenuate or recover its virulence thus producing long periods of calm or *recrudescence*?"[336] With this question, Cuervo finally answered the problem of the persistence of yellow fever in Cúcuta despite the lack of importation: the yellow fever germ was first imported from Maracaibo in 1882 and remained in Cúcuta in an attenuated state until environmental conditions enhanced its virulence, thus causing subsequent epidemics.

Historian Andrew Mendelsohn has pointed out that virulence was a key concept in the early years of bacteriology. The 1880-1881 years of Pasteur's work not only resulted in the invention of vaccines and the beginning of immunity; Pasteur's science was reinvented, says Mendelsohn, as a science of experimental variation of virulence. But the most relevant point for my argument is highlighted by

[335] Ibid., p. 82.
[336] Ibid. pp. 82-6.

Mendelsohn: that the attenuation and augmentation of virulence were conceived to operate continuously in the world by Pasteur himself.[337] In 1880 Pasteur revealed his method for attenuation, exemplified by his method of creating the fowl cholera vaccine: prolonged exposure of pathogenic bacterial cultures to atmospheric air. Mendelsohn argues that this paper simultaneously proposed a correlation between the laboratory and the world and thus a way to explain disease. In effect, Pasteur suggested that his vaccine-making method might hold the key to understanding epidemics; that atmospheric oxygen must be the agent of attenuation, not only in the laboratory but also in the world outside. Pasteur even addressed a case that resembled the situation in Cúcuta, namely plague epidemics for which no source of contagion had been found. Mendelsohn quotes Pasteur: "in all countries, its attenuated virus ought to exist, ready to retake its active form when conditions of climate, of famine, of poverty, show themselves anew" and that "attenuation of viruses by the influence of the air should be one of the factors in the extinguishing of great epidemics".[338] According to Mendelsohn, contemporaries immediately put this hypothesis to work in explanations of the epidemic curve. Doubtlessly, Cuervo used the idea of virulence of attenuation by the environment to explain the persistence of yellow fever in Cúcuta despite the lack of importation of the agent.

In conclusion, Cuervo reconciled the medical geographical and the bacteriological causal framework in a very pragmatic way in his analysis of yellow fever in Cúcuta. He used an etiological triad of causal elements which included the agent (the microbe), the soil (the body) and the environment (cosmic elements). He followed the Pasteurian notion of variable virulence and its extension to epidemics in explaining the persistence of yellow fever in Cúcuta, an idea through which he reconciled the first and third elements of the causal triad. Cuervo also found other ways in which these two elements interacted: for example, the bactericidal action of sunlight that could explain why yellow fever was usually caught in the mornings and evenings, and the role of winds with their predisposition to epidemics. Cuervo also addressed the old problem of acclimatization - the resistance to yellow fever due to living in places where it was endemic - incorporating the cellular theory of

[337] Andrew Mendelsohn, "'Like All That Lives': Biology, Medicine and Bacteria in the Age of Pasteur and Koch", *History and Phiosophy of the. Life Sciences*, 24, 2002, pp. 4-5.
[338] Ibid., p.8

immunology and inheritance into his answer, an idea that linked all three elements of the causal triad.

Magdalena's fevers and paludism

Physicians who dealt with yellow fever during the 1880s in Colombia usually did so by referring to the "Magdalena's fevers". As I explained in the two first sections of this chapter, by the 1880s Colombian physicians were consolidating the divide between yellow fever and malaria from the umbrella notion of Magdalena's fevers. I showed that, for Gutierrez, yellow fever was different from the other Magdalena's fevers (he did not name them as malaria), though he suggested that both were caused by similar microorganisms which he believed were found in the intestine. Esguerra, on the other hand, was also convinced that yellow fever and malaria were two different infectious diseases but, contrary to Gutierrez, considered that malaria was a telluric disease whereas yellow fever was determined by hygienic causes. Cuervo also addressed the problem of Magdalena's fevers. Like Gutierrez and Esguerra, he insisted that Magdalena's fevers included "two clinically well characterized groups: the malarial and the yellowish group". But while he struggled to reconcile medical geographical and bacteriological arguments when explaining the cause of yellow fever, he did not find any problem with explaining malaria through medical geographical arguments alone.

Cuervo started by describing the clinical, therapeutic and epidemiological differences between the two diseases.[339] Along with these disparities, Cuervo also insisted that malaria and yellow fever had etiological differences related to their relative dependence on climate. The malarial group was limited to the hot and humid lowlands, no higher than 800 meters and no colder than 24 degrees Celsius. The "yellowish" group could also occur within those limits, but was not confined to those

[339] Paludism occurred in acute and chronic forms. The acute forms were of intermittent, remittent and continuous fevers and the chronic form was the paludic cachexia. This latter state was supposed to be the result of repetitive attacks or of the slow and constant action of the miasma on the organism. To the paludic form belonged the intermittent, remittent, hematuric, bilious fevers and those fevers of ill-determined features or of typhoid aspect (true typho-malarial fevers). The yellowish infection presented a well defined clinical spectrum, always the same. The malarial group was an endemic disease, non-transmissible, and responsive to quinine, whereas the yellow group was epidemic, contagious, non-recurrent and of dubious or no responsiveness to quinine. See Cuervo Márquez, *La fiebre amarilla*, pp. 289-93, 296-7, 299-301.

boundaries; in fact it had climbed up to 1170 meters in Ocaña and to other villages like Tocaima and Guaduas in the *eastern cordillera*.[340]

With regards to malaria, Cuervo implicitly applied his triadic causal frame (agent, body, and environment), though his first line of explanation was the medical geographical model. Cuervo considered malaria to be determined by climate and caused by miasmas. He followed the medical geographical divide between the lowlands and highlands and the associated geological, botanical and pathological phenomena in explaining the geographical distribution of malaria in the two main sections of the Magdalena river, the *Alto Magdalena* (the Magdalena river valley from Neiva to Honda) and the *Bajo Magdalena* (between Honda and the Atlantic port of Barranquilla).

Thus, Cuervo stated that in the lower areas of the *Alto Magdalena*, the development of paludism was associated with the geological constitution of the soil and with the decomposition of organic matter. According to him, the sun decomposed the organic matter left after floods (cattle corpses and fish), which eventually poisoned water and air, thus producing paludism. If one moved up to the higher areas of the *Alto Magdalena,* near the *cordilleras*, malaria would proportionally diminish. Since there were no floods in those regions and the climate was drier and milder than that of the lower areas, paludism was rare.[341]

In the *Bajo Magdalena,* there were only lowlands where the river formed streams that sometimes flooded nearby lands, forests and towns. Like the lower areas of the *Alto Magdalena*, Cuervo declared, the *Bajo Magdalena* was "formed by soils in which temperature, humidity and luxurious vegetation favor the development of paludism". [342]

Cuervo held to a miasmatic identity for paludism. He vaguely described the paludic miasma as "unique in its essence and multiple in its manifestations"; it needed organic matter (preferably plant matter) in the process of decomposing, humidity, a temperature not lower than 15 degrees Celsius, and air, if it were to be produced. The idea that "the paludic miasma does not spread [in that] it develops and dies in the same place where it born", is the landmark in the climatic determinism of

[340] Cuervo Márquez, *La fiebre amarilla*, pp. 289-93, 296-7, 299-301.
[341] Ibid., pp. 289-90.
[342] Ibid., pp. 292-3.

fevers of the warm lands. Interestingly, Cuervo said that even though low Colombian lands were paludic, "it does not mean that the highlands are completely protected against malaria". It was not exceptional, he said, in Bogotá or in Pamplona, for the simple removal of debris during the repair of a building to produce a typhomalarian fever, intermittent fevers and remittent fevers, which receded with quinine.[343] Cuervo was echoing the old nosological flexibility among fevers, according to which the benign type of intermittent fevers could happen in the highlands provided that conditions for miasmatic productions were present, whereas the malign or pernicious type developed in the lowlands by virtue of heat, humidity and organic matter. The idea that the same kind of intermittent fevers – later called malarial fevers – could happen in Bogotá (benign intermittent fevers) is nothing but another demonstration of the persistence of the medical geographical argument in Cuervo's account of malaria. This persistence remained, despite Cuervo's knowledge of the parasites and microbes that had been proposed to replace the paludic miasma (Salisbury, the *Gemiasma palmella*; Eklund, the *Limnophysalis hyaline*; Klebs and J. Crudeli, the *Bacillus malariae*; Laveran, a parasite he found in feverish people). Scientists like Salisbury, Cuervo remembered, had hypothesized different parasites for explaining the diversity of presentation of malaria, but Laveran, Colin and Roux believed that there was a single specific agent. For Cuervo, the organic predisposition or the amount and toxic power of the miasma, rather than those other agents, would explain these multiple manifestations.[344]

In conclusion, Cuervo struggled to reconcile the medical geographical and bacteriological views of yellow fever and malaria. He set up the triad of the agent, body and environment, but struggled to treat each of them as if they had an equivalent role in the production of disease. In the case of yellow fever, he found one way of connecting the two models in the bacteriological notion of virulence and its application to epidemics. He even gave winds the role of the precursor of yellow fever epidemics, and linked the problem of acclimatization to the cellular theory of immunity; however, he recognized that environmental conditions were secondary to the agent. A different picture was applied to malaria: it still depended on environmental conditions. To sum up Cuervo's divergent position with regards to

[343] Ibid., p. 298.
[344] Ibid., pp. 299-301.

yellow fever and malaria, it is best to quote Cuervo himself: "given certain climatic conditions of a locality", he said, "one can a priori predict whether or not malaria prevails in that place".[345] Conversely, yellow fever "has as [its] etiological character … the absence of determined climacteric conditions".[346]

Reflections on the problem of the transition from medical geography to germ theory

I showed in the first two sections that Daniel Gutierrez and Carlos Esguerra both accepted that yellow fever and Magdalena's fevers were caused by hypothetical microorganisms or "infectious agents". But why did Gutierrez, from the medical school in Bogotá, reject the climatic determination of yellow fever and Magdalena's fevers? Why did Esguerra, on the contrary, writing from the medical school in Paris, retain the telluric nature of malaria and highlight the role of hygienic conditions as the "milieu of culture" of the agent in the development of yellow fever?

Both had been trained in the medical school in Bogotá, but they had different experiences of fevers and framed their work in different ways. Gutierrez was inspired by his professors' bacteriological work in the laboratory of histology and micrography; he did not have clinical experience of the fevers of warm climates, and perhaps neither did his supervisors. He confessed to have been only an "eyewitness" of the Magdalena's fevers.[347] But clinical experience seemed unnecessary for someone who declared that the only way of knowing the true nature of yellow fever and Magdalena's fevers would be through bacteriological studies, and that all previous knowledge of these pathologies should therefore be re-evaluated, as he indicated in his thesis. In contrast, Carlos Esguerra, although in Paris, followed the medical geographical tradition in his main approach to fevers, drawing especially on his clinical experience in the Magdalena river village of Honda, prior to his stay in Paris. Esguerra highlighted the value of empirical observation *in situ* as the basis of

[345] Ibid., p. 361.
[346] Ibid., p. 297.
[347] Gutierrez, *Los microorganismos de la fiebre amarilla*, p. 10.

authority in the study of diseases,[348] an argument that the Colombian physicians who consolidated the medical geographical perspective of diseases had been using since the mid-1850s to claim authority over medical European knowledge.

The different approaches taken by Gutierrez and Esguerra provide an interesting addition to our understanding of the relationship between bacteriology and medicine. Historians Russell C. Maulitz and Anne Marie Moulin have followed the tensions that bacteriology generated among clinicians in America and France. In America, Maulitz says, bacteriology not only offered clinicians new tools but also threatened them by suggesting that scientific values might move them from the bedside to the bench. The central question, says Maulitz, was the proper path by which science might be assimilated into medicine.[349] In France, according to Moulin, the pathologist Charles Bouchard (for example) actively advocated the introduction of microbiology into the medical curriculum, but clarified the terms of the collaboration between microbiology and medicine: clinicians should retain control over questions related to the pathogenesis of diseases, and microbiologists could only provide a partial answer.[350] Colombian doctors seem to have taken a more positive view of bacteriology. By the end of the 1880s, some physicians like Gutierrez were beginning to use bacteriology to justify the epistemological and social authority of their profession, displacing medical geography from this role; some also began to stress the importance of laboratory work on bacteriology in medical training.

I showed in Chapter 1 that from the 1850s onward, physicians in Bogotá sought to differentiate themselves from traditional healers and to gain social legitimacy by consolidating the weak medical body. The scientific argument they used for this purpose was the need to create a national medicine based on knowledge of the local pathologies, i.e. medical geographical works. They argued that it was necessary, for example, to study Magdalena's fevers because they affected the economic growth of the country. Since the medical geography perspective implied that diseases were produced locally, by geological, climatic and other geographical factors, physicians

[348] Esguerra, *Contribution a L'étude*, pp. 11, 14, 76.
[349] Rusell C. Maulitz, "Physician versus Bacteriologist': The Ideology of Science in Clinical Medicine" in Morris J. Vogel and Charles E. Rosenberg (eds), *The Therapeutic Revolution* (University of Pennsylvania Press, 1979), p. 92.
[350] Anne Marie Moulin, "Bacteriological Research and Medical Practice in and out of the Pastorian School" in Ann La Berge and Mordechai Feingold (eds), *French Medical Culture in the Nineteenth Century* (Amsterdam/Atlanta GA, 1994), pp. 331-332.

also expected that the kind of knowledge that they could produce about Colombian diseases would be superior to European knowledge; therefore, the project of a national medicine based on medical geographical works would also mean that the Colombian medical community would make original contributions to science.

By 1886, physicians had more or less succeeded in their goal of gaining social recognition. As I showed in previous chapters, by the mid-1880s the medical body centered in Bogotá had consolidated their position as the scientific authority in medical and hygiene matters in Colombia – a status which was partially crystallized in the creation of the Central Hygiene Bureau in 1887. Calls for the creation of a distinct national medicine based on medical geographical works thus began to decline and bacteriology began to take its place, which was consistent with the more internationalist ambitions on the part of the local medical profession. For example, Gutierrez, who criticized the medical geographical approach to yellow fever, argued in 1888 that bacteriological studies would "put our national medicine in the place it deserves."[351] At the same time, the SMCN began to discuss the importance of bacteriology in medical training. In the same year (1888), the physician Proto Gomez, for example, highlighted that bacteriology had produced a revolution in medical training; he insisted that the time had come to unify theoretical and practical medical training in laboratories, including not only the study of normal and pathological histology but also that of bacteriology.[352] It is no accident that earlier that year the government officially recognized the histological and anatomopathological laboratory of the medical school in Bogota, the same one in which bacteriological studies were first performed.

Gutierrez had complained of the slowness of the introduction of bacteriological studies into medical training in Bogotá. He declared that a number of established professors were well aware of the development of bacteriological studies in Europe, but that they had "expressed a lot of distrust" of the certainty that had been attributed to bacteriology in the old continent, mainly because they could not see any practical use for such knowledge.[353] But this skepticism seemed to be receding, as Castañeda's

[351] Gutiérrez Arango, *Los micro-organismos de la fiebre amarilla*, p. 10.
[352] Proto Gómez, "Importancia de los studios bacterialógicos", *Revista Médica*, XII, No. 128, Agosto 28, 1888, pp. 131-2.
[353] Gutiérrez Arango, *Los micro-organismos de la fiebre amarilla*, p. 11.

and Borda's experiments for creating a chemical vaccine against yellow fever in 1889 illustrate. The historian Diana Obregón has shown how during the following decade, Colombian physicians would use bacteriological studies on leprosy as the touchstone for the prestige and authority of the profession.[354] I will return to this problem in Chapter 5.

When Colombian physicians constructed their various interpretations of the germ hypothesis of yellow fever, they had to refer to the pre-existing tradition of climatic theories of fevers. The books that I analyzed reveal how physicians sought to reconcile the new bacteriological hypothesis of disease causation with the pre-existing medical geographical perspective, in a process that helped to consolidate the emerging differentiation between yellow fever and malaria. I showed that Daniel Gutierrez's thesis focused on the microorganisms of yellow fever and repudiated the medical geographical tradition, considering microorganisms alone to be the key element that would define yellow fever and malaria; however, he reluctantly invoked environmental conditions in order to justify the geographical distribution of yellow fever. Carlos Esguerra, on the other hand, adjusted the two perspectives by drawing an analogy between the bacteriological notion of culture and the old idea of hygiene as the necessary condition for the development of the yellow fever agent, and thereby preserved older telluric arguments for explaining malaria. Luis Cuervo tried to reconcile the two perspectives by using the Pasteurian concept of variation of virulence caused by temperature in the laboratory, and extrapolating that phenomenon to explain yellow fever epidemics, while simultaneously maintaining a medical geographical and miasmatic identity for malaria.

These different approaches to yellow fever and malaria also convey different interpretations of germs or microorganisms. Thus, for Daniel Gutierrez, germs were voracious agents that fully explained epidemics of either of the two diseases; Esguerra used germs and miasmas almost indistinguishably, as if it made no difference to him with regards to the clinical and epidemiological distribution of these fevers; and finally for Cuervo, microorganisms varied in their virulence,

[354] Diana Obregon, "Sobre epidemias, endemias y epizootias: algunos aspectos del desarrollo de la bacteriología en Colombia", *Biomédica*, 18, 2, 1998, p. 118.

resisted, became exhausted, produced immunity, and were the first element of a causal triad along with bodily and environmental elements, all of which were necessary in order to account fully for yellow fever but seemed unnecessary for explaining malaria. Gutierrez, Esguerra, and Cuervo constructed different accounts of yellow fever and malaria and of germs partly because of their different medical experience with fevers. Thus, for example, Daniel Gutierrez, who defended microorganisms as the only cause of yellow fever worth considering, did not have any clinical experience of the fevers of the warm climates; he did however have some laboratory experience in Bogotá. Esguerra, who followed the medical geographical analysis of the Magdalena's fevers, justified his work on the basis of his clinical experience in the Honda, a village on the Magdalena River. Finally, Cuervo dealt with fevers of the Magdalena River as an army physician traveling along that river and to the Atlantic coasts during 1885; he also worked in Cúcuta during the epidemics of the second half of the 1880s.

Cuervo had first approached fevers from the medical geographical perspective in 1886, but after experiments with inoculations against yellow fever in Cúcuta in 1887, he began to include the bacteriological hypothesis of yellow fever. Not only did he analyze the inoculations of the *Peronospora lútea*, but he also employed some bacteriological methods in his analysis of yellow fever and Magdalena's fevers. Cuervo's broader clinical and laboratory experience and his commitment to medical geography would explain his struggle to reconcile both perspectives.

Whatever the differences of interpretation of germs and of yellow fever and malaria among the Colombian medical community, it is clear that the physicians of the 1880s had to refer to the pre-existing tradition of climatic theories of fevers when dealing with the bacteriological hypothesis of yellow fever and malaria. Explicitly or implicitly, they needed to reconcile the germ hypothesis with the medical geographical framework of disease causation that had been established since mid-century. Germs may have then acquired various meanings in Colombia, because they were first understood within the medical geographical framework.

The idea that in the early years of medical bacteriology there was not "one germ theory" but several germ theories (at least before a consensus was reached, according to Worboys) was debated in the 1997 special issue, "Rethinking the Reception of the

Germ Theory of Disease", in the *Journal of the History of Medicine*.[355] As I have shown in this chapter, when the "parasitic" or germ hypothesis began to be seriously considered as the most likely cause of yellow fever in Colombia, physicians simultaneously used different meanings of germs: germs could be virtually equivalent to miasma, or agents with the ability to vary their "virulence" by environmental changes, as suggested by the Pasteurian account of the interaction between the environment and microorganisms. But there is a problem. As I mentioned before, Mendelsohn argues that Pasteur proposed a correlation between variation of virulence in the laboratory and the world outside, explaining how climate, famine and poverty could activate an attenuated virus and thus produce disease. Mendelsohn asserts that, for this very reason, historians cannot assume a conflict between nineteenth-century appeals to climatic factors and appeals to germs as the cause of diseases, since both were part of Pasteur's and Roux's etiological perspectives.[356] Mendelsohn criticizes Michael Osborne's paper on French military epidemiology (1992) in particular, where Osborne shows that for the army doctor Louis-Felix-Achille Kelsh (1841-1911) germs were not sufficent to explain epidemics, leading him to effect a synthesis between his medical geographical approach and the bacteriological point of view. This division, Mendelsohn implies, is in Osborne's head and not in the theory used by the nineteenth-century French doctor.[357] In Mendelsohn's view, Osborne is using a static and incorrect idea of "germ theory".

Mendelsohn is right in so far as nineteenth-century physicians, like Cuervo in Colombia, actually used the Pastorian notion of virulence for explaining epidemics. But Mendelsohn's argument does not apply to cases like that of Gutierrez, who repudiated any climatic argument and saw any attempt in that direction as opposition to the bacteriological perspective. In other words, I agree that there was not a fixed or simple "germ theory" in the early years of medical bacteriology. But I also believe

[355] See Nancy J. Tomes and John Harley Warner, "Introduction to Special Issue on Rethinking the Reception of Germ Theory of Disease: Comparative Perspectives", *Journal of the History of Medicine*, 52, 1997, pp. 7-16. Michael Worboys, *Spreading Germs. Disease, Theories and Medical Practice in Britain, 1865-1900* (Cambridge: Cambridge University Press, 2000).
[356] Mendelsohn, "'Like All That Lives', p. 12.
[357] Michael A. Osborne, "French Military Epidemiology and the Limits of the Laboratory", Andrew Cunningham and Perry Williams (ed.), *The Laboratory Revolution* (Cambridge: Cambridge University Press, 1992), pp. 189-208.

that the ideas around germs developed in Pasteur's years could have held different meanings for general practitioners, hygienists and army physicians. For the meaning of objects varies depending on previous knowledge traditions and people's experience, as I hope to have demonstrated in this chapter.

CHAPTER 4

"THE STRANGEST FORMS OF BOGOTÁ'S *PIRETOLOGÍA*". TYPHOID FEVER AND TYPHUS IN THE TRANSITION FROM MEDICAL GEOGRAPHY TO GERM THEORY

Introduction

The last two chapters were devoted to an analysis of the way that Colombian physicians transformed their understanding of periodic fevers (yellow fever and malaria) during the transition from medical geography to bacteriology. This chapter follows this transformation in the case of continuous fevers (typhoid fever and typhus). I will explore how Colombian physicians mobilized medical, geographical and bacteriological arguments in their discussions of the nature of continuous fevers in general, and of those of the capital city, Bogotá, in particular.

In nineteenth-century Colombian medicine it was considered that typhoid fever was a contagious disease not primarily determined by climate. However, I included this fever in my analysis because physicians approached it from the neo-Hippocratic and medical geographical perspective, even while declaring its non-climatically determined nature. Certainly, in the discussions of typhoid fever, physicians included one of the main proposals of medical geography, Jean Boudin's laws of geographical coincidence and antagonism between diseases, as I will show.

Another reason for considering typhoid fever within this work is related to bacteriology. Historians who have worked on the nineteenth-century knowledge of typhoid fever have found that debates were concentrated on the distinction between typhoid fever and typhus, and on its methods of transmission, including the identification of the "healthy carrier".[358] And although the identification of the

[358] Some of them are, in alphabetical order, Vincent J. Cirillo, "Fever and Reform: The Typhoid Epidemic in the Spanish-American War", *Journal of the History of Medicine and Allied Sciences*, 55, 2000, pp. 363-397; Margaret Humphreys, "A Stranger to Our Camps: Typhus in American History", *Bulletin for the History of Medicine*, 88, 2006, pp. 269-290; Michael P. McCarthy, *Typhoid and the politics of public health in 19th-century Philadelphia* (Philadelphia: American Philosophical Society, 1987); K. David Patterson, "Typhus and its Control in Russia, 1870-1940", *Medical History*, 37,

typhoid bacillus and its association with typhoid fever took a decade to complete (between c.1880 and c.1890), it seems from these stories that doctors from different latitudes easily accepted the Eberth bacillus as the cause of typhoid fever. Early supporters of Pasteurism in Colombia also embraced the new cause, as I will show. However, there were some for whom typhoid fever was not primarily determined by microorganisms but was spontaneously produced in the body – an anti-Pasteurian theory - or for whom typhoid miasmas and germs meant the same thing. This reluctance to accept the microbiological cause of typhoid fever contrasts with the ease with which most of the same physicians accepted the bacteriological hypothesis for yellow fever. As I showed in Chapter 2, between 1887 and 1889, Colombian doctors performed inoculations and sought a chemical vaccine against yellow fever following the Pasteurian model, during a time when the nature of the microorganism was strongly contested. In contrast, by the time the international community had agreed that the Eberth bacillus was the specific cause of typhoid fever (in the second half of the 1880s), Colombian physicians considered germs more relevant for practical purposes than for causal speculations, and some even downplayed their causal role using anti-Pasteurian arguments.

This chapter begins with a brief description of what typhoid and typhus meant to mid-century Colombian doctors and how they used neo-Hippocratic ideas to shape those meanings. In the following three sections I will analyze the medical geographical arguments that physicians used to understand typhoid fever, and how they used clinical thermometry and notions about ferments in those debates. In the last three sections I will explore arguments in favor of, and against, the bacteriological identity of typhoid fever; and also look at how clinicians, physicians-hygienists and local authorities used bacteriology in discussions of, and practices around, the typhoid epidemics of 1887-1890.

1993, pp. 363-381; Lloyd G. Stevenson, "A Pox of the Ileum: Typhoid Fever among the Exantemanta", *Bulletin of the History of Medicine*, 51, 1977, pp. 496-505; Dale C. Smith, "Gerhard's distinction between typhoid and typhus and its reception in America, 1833-1860", *Bulletin of the History of Medicine*, 54, 3, 1980, pp. 368-385; Ibid., Dale C. Smith, "The Rise and Fall of Typhomalarial Fever", *Journal of the History of Medicine and Allied Sciences*, 37, 1982, 182-220; 287-321; and Leonard G. Wilson, "Fevers and Science in Early Nineteenth Century Medicine", *Journal of the History of Medicine and Allied Sciences*, 33, 1978, pp. 386-407.

Typhus and/or typhoid fever in the first half of the nineteenth century

One of the earlier accounts of continuous fevers in nineteenth-century Colombia appeared in José Félix Merizalde's book, *Elementos de patologia general*, published in 1831. As I mentioned earlier, this book was written as a textbook for medical students in Bogotá.[359] In *Elementos de patologia*, Merizalde approached the problem of causes of diseases following the neo-Hippocratic framework of specific, predisposing and occasional causes. With regards to what he called *tifo*, Merizalde pointed out that it was a contagious disease whose specific cause was an animal vapor emerging from human bodies. This vapor would contaminate the atmosphere, thus affecting healthy people surrounding the sick. That is why Merizalde considered that living in cities was a general predisposing cause of *tifo*; terror or excesses were counted among the occasional causes that could trigger *tifo*. [360]

If *tifo* was associated with some predisposing causes, claimed Merizalde, then there were also some conditions that made individuals resistant to *tifo*. Without specifying the underlying mechanism, Merizalde said that one attack of *tifo* would protect against a second attack, though only temporarily. Some people would be protected just by virtue of the reinforcement of the vital force that debilitated the destructive power of the poison. Physicians surrounded by the sick in hospitals would not necessarily get *tifo*, because of this latter mechanism.[361]

It is very likely that Merizalde's scheme of predisposing, specific and occasional causes was the conceptual frame used by doctors during the typhoid fever epidemic of Bogotá in 1836. I have not found contemporary accounts of that epidemic, but according to Antonio Vargas Reyes's report two decades later, it was of typhoid fever and killed 300 "important" people, twelve of whom were his classmates and probably professors at the faculty of medicine.[362]

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[359] I also mentioned in Chapter 1 that Merizalde's textbook was extensively based on the first edition of the French Auguste Françoise Chomel's work, *Élémens de pathologie générale* (1817).
[360] Jose Felix Merizalde, *Elementos de Patologia general* (Bogotá: J. N. Barros, 1831), pp. 13, 16.
[361] Ibid., pp. 19, 32-33.
[362] Antonio Vargas Reyes, "Mis observaciones sobre la fiebre thyfoides", *El Tiempo*, No 13, marzo 17 de 1855 and No. 14, abril 3 de 1855 (pages not numbered).

A second epidemic occurred in Bogotá in 1855. Antonio Vargas Reyes claimed it was again of typhoid fever. But before I describe the causes that he and his contemporaries associated with typhoid fever at that time, I shall show how Vargas Reyes understood typhoid fever in relation to typhus, and to eruptive fevers, the other kind of continuous fevers.

First of all, Vargas Reyes reinterpreted all previous cases of continuous fevers in Bogotá as typhoid fever (he was referring to the *tifo* of Merizalde, and to what people called *peste* and *tabardillo*). Furthermore, he affirmed that the *gastro enteritis* (Broussais) and the *dothinenteritis* (Bretonneau) of the French, on the one hand, and the *typhus* of the English, on the other, were also typhoid fever.[363] This opinion contrasted with the growing consensus among the French, English and Americans on the different nature of typhoid and typhus fevers. Historians Leonard Wilson (1978) and Dale C. Smith (1980) have shown how the first step in constructing that distinction was taken by Pierre A. Louis in his work *Reserches anatomiques, pathologiques et thérapeutiques sur la maladie connue sous le noms de gastroentérie, fièvre putride, adynamique, ataxique, typhoide, etc* (1829).[364] Following his numerical method (that is, the idea that new and valid medical knowledge could be derived from aggregated clinical data), Louis established that the continued fever of Paris was a disease characterized in life by rose-colored lenticular spots and diarrhea, and in death by pathological changes in the Peyer's patches of the small intestine. He was convinced that all the important continued fevers of other locations, including the typhus of the English, were just typhoid fever.[365]

According to Wilson and Smith, during the following years, many practitioners sought to establish whether their epidemics of continuous fevers were of that type. Whereas the British did not see anything that could identify their continuous fevers in Louis's findings, two Americans and pupils of Louis, James Jackson and William Wood Gerhard, found in pathological anatomy the key to differentiating between the

[363] Ibidem.
[364] Leonard G. Wilson, "Fevers and Science in Early Nineteenth Century Medicine", *Journal of the History of Medicine and Allied Sciences*, 33, 1978, pp. 386-407 and Dale C. Smith, "Gerhard's distinction between typhoid and typhus and its reception in America, 1833-1860", *Bulletin of the History of Medicine*, 54, 3, 1980, pp. 368-385.
[365] Smith, "Gerhard's distinction between typhoid and typhus", pp. 370, 375-6

fevers that they encountered back in the USA – typhoid fever - and the typhus described by the British. James Jackson followed Louis's method in the wards of the Massachusetts General Hospital, and William Wood Gerhard did the same in the Pennsylvania Hospital in Philadelphia in the early 1830s. They first established that the common continued fever of the eastern USA, often called typhus by American practitioners, was the same disease as the typhoid of Louis. But the problem remained that there were certain American epidemics that were not typhoid fever, and were instead more similar to the British typhus. During the 1836 fever epidemic in Philadelphia, Gerhard's use of autopsies allowed him to conclude that it was not typhoid fever. Though it is not clear if Gerhard himself affirmed that it was typhus, Smith suggests that because Alfred Stillé reported in 1838 that his observations in Philadelphia as Gerhard's resident were identical to the typhus in Dublin, Edinburgh and London, Gerhard was responsible for establishing the non-identification of typhus with the typhoid fever commonly seen in America.[366] According to this account, the responsibility for the distinction between typhoid and typhus (on anatomopathological grounds) was that of the Americans.

In Britain, William Jenner (1815-1898), professor of pathology at the University College, London, carried out work in late 1849 to confirm the work of Gerhard. Only after his analysis of cases in the London Fever Hospital in 1847, and the post-mortem examinations, was the distinction between typhoid fever and typhus accepted.[367] In France, on the other hand, one of Gerhard's students, Alfred Stillé, presented Gerhard's works, and his own experiences in Naples, London, Dublin and Edinburgh, to the Medical Society in 1838, where he compared typhus fever and typhoid. Stillé's paper, and the subsequent translations of Gerhard's works in French journals, led French practitioners to understand that what they called *dothinenteritis* was a different disease from the British typhus.[368] But in 1841 Pierre Alexander Louis still insisted that typhoid and typhus were one and the same disease. In America, Elisha Barlett's 1842 monograph discussed this distinction, but practitioners across the USA persisted in considering typhoid fever and typhus the same disease, probably because of their lack of experience in post-mortem

[366] Ibid., pp. 375-6.
[367] Ibid., pp. 382-3.
[368] Wilson, "Fevers and Science in Early Nineteenth Century", pp. 404-405.

examination, or even their doubts about the reliability of the distinction in the face of a great mass of British pathological studies favoring the unity of continued fever. More favorable attitudes to Louis's method developed among American practitioners after the second edition of Barlett's book in 1847.[369]

The consensus on typhoid and typhus being two different fevers developed one or two decades later in other places. In Russia physicians fully accepted the distinction by 1860, but statistical tables sometimes continued to include significant numbers of cases as typhus-undifferentiated well into the 1890s.[370] In Colombia the 1862 classification of fevers by Vargas Reyes included typhoid fever but not typhus. However, Vargas Reyes recognized that "I could have placed the typhus of the British to the side of the typhoid fever", in the group continuous fevers.[371] Physicians and local authorities frequently used *tifus* and *tifoidea* as interchangeable terms in the following decades. The distinction between the two kinds of fever only became crucial during the 1887-89 fever epidemics in Bogotá, as I will show, but until at least 1895 clinicians still used the category "continuous fevers" along with typhoid fever and typhus in hospital statistics.[372]

Before considering which ideas were held by physicians about the causes of typhoid fever in this period, I need to outline the relationship between typhoid fever and the other continuous fevers. In Antonio Vargas Reyes's scheme, typhoid fever was an eruptive fever like smallpox. According to Vargas Reyes, "typhoid fever, like smallpox and measles, has invasive symptoms ….. periods of eruption, supuration and descamation". These periods happen "following the *sudamina*, the pink spots, petechias, and the swelling, suppuration and ulceration of the Payer patches".[373]

Since the seventeenth century, doctors in Europe had been able to place typhoid fever among the "exanthemata", based on the analogy between pox and the lesions in the ileum found with this fever. This idea was first proposed by Pierre Bretonneau

[369] C. Dale Smith also argued that the lack of experience with typhus, which would only arrive on American soil with Irish immigrants in the 1840s, may explain this "delay" in "understanding" Gerhard's work. See Smith, "Gerhard's distinction between typhoid and typhus", pp. 376-385.
[370] K. David Patterson, "Typhus and its Control in Russia, 1870-1940", *Medical History*, 1993, 37, p. 363.
[371] Antonio Vargas Reyes, 'Discusión sobre la fiebre amarilla', *Trabajos Científicos del Doctor Antonio Vargas Reyes recopilados en obsequio de la humanidad doliente i de la juventud estudiosa de Colombia.* Tomo Segundo (Bogotá: Imprenta la Nación, 1862), p. 54.
[372] Gabriel J. Castañeda, "Patología de Colombia. Continuación", *Revista Médica*, 235, Noviembre de 1897, p. 125.
[373] Vargas Reyes, "Mis observaciones sobre la fiebre thyfoides", pages not numbered.

(1778-1862) in 1929. During the early 1850s the French Academy debated if enteric fever was just an internal variola, and the English William Budd (1873) also saw, in the lesions of the Peyer patches, a true exanthema by analogy with smallpox.[374] In Colombia physicians also made an analogy but of a different order. Vargas Reyes classified typhoid fever as an eruptive disease, based not only on the similar course of symptoms as I mentioned above, but also on the analogy between the skin lesions of smallpox and the rose-colored lenticular spots of the skin of typhoid patients. He did not declare that typhoid was the pox of the ileum; he did not see the same lesion in smallpox and the ileum, for he considered typhoid fever and smallpox to be two different kinds of fevers. What Vargas Reyes did notice was that lesions in the ileum appeared when the skin spots were absent: "Eruption is a less significant phenomenon in typhoid fever than in eruptive fevers, because when it does not happen, in compensation, the inflammation of intestines is more intense."[375]

To sum up, by the time that the 1855 epidemic of continuous fever occurred in Bogotá, doctors still thought that typhoid and typhus were the same disease, and that typhoid fever was an eruptive disease like smallpox and therefore contagious. What about its causes?

Typhoid causes were framed within the scheme of predisposing, specific, and occasional causes. Some physicians considered, for example, that the main cause was the insalubrity of air, caused by an excess of humidity and also by deleterious miasmas escaping from filth. Others associated the epidemic with the accumulation of individuals, wounded people, and corpses in Bogotá during the 1854 civil war when around 10,000 troops besieged Bogotá.[376]

Vargas Reyes considered those ideas mistaken. He claimed that neither miasmas in the air nor humidity were necessary for the fever to develop. Filth and war could not account for the fever; he argued that during the previous epidemic (1836) Bogotá had suffered worse hygienic conditions and that there was no war. He seemed to be

[374] Lloyd G. Stevenson, "A Pox of the Ileum: Typhoid Fever among the Exanthemata", *Bulletin of the History of Medicine*, 51, 1977, pp. 501-3.
[375] Vargas Reyes, "Discusión sobre la fiebre amarilla", pp. 53-54.
[376] Vargas Reyes, "Mis observaciones sobre la fiebre thyfoides". On the civil war of 1854 see Fabio Zambrano, "El golpe de Melo de 1854", *Las guerras civiles desde 1830 y su proyección en el siglo XIX* (Bogotá: Museo Nacional de Colombia, 1998).

suggesting that meteorological or hygienic conditions were irrelevant as causes of typhoid fever. How, then, did he explain its causes?

Following the causal model of predisposing, specific and occasional causes, similar to the one Merizalde had used in his 1831 book, Vargas Reyes affirmed that when the body had somehow weakened, it became predisposed to disease. The miasmas from the accumulation of individuals, which, for Merizalde, had been the specific cause of typhoid fever, were, for Vargas Reyes, just the predisposing one: they would debilitate the organism, thus making it vulnerable to typhoid fever. Vargas Reyes realized, however, that this predisposing cause may predispose to many other diseases; in other words, that it was also necessary to identify a specific cause for typhoid fever. But Vargas Reyes did not have any idea of what it might be; he considered that this was a difficult issue, similar to searching for the "primary or essential causes of things."[377]

This skepticism regarding a specific cause would soon be replaced by a miasmatic cause. In 1862 Vargas Reyes recognized that "everybody agrees that typhoid fever develops in besieged cities, in prisons and camps - that is, in places of accumulation of people and animal matter in decomposition - and that the fever develops because of the absorption of miasmas that those places exhale."[378] This shift appeared in Vargas Reyes's scheme of the whole family of essential fevers. In this scheme, he portrayed fevers as a family united not only by the symptoms known as "fever" but also by a common cause, miasmas, and a common lesion, the poison of the blood caused by miasmas. Differences among genera, species, varieties, and types of fevers depended on the way that symptoms occurred, but also on the circumstances in which those miasmas were produced. Thus, while periodic fevers were caused by miasmas from putrefaction of plant and animal matter, continuous

[377] Antonio Vargas Reyes, "Mis observaciones sobre la fiebre thyfoides". For "common people", he said, occasional causes such as cold or hot air, winds, suppression of natural evacuations (sweat, milk, and menstruation), prolonged sleepless nights, moral emotions and retrocession of the exanthema were the real causes of the typhoid. Sometimes, some occasional causes like drunkenness, frequent sexual intercourse and hard mental work could debilitate the organism, thus acting as predisposing causes.

[378] Vargas Reyes, "Discusión sobre la fiebre amarilla", p. 40.

fevers too could originate from miasmas produced from animal matter, but also from places with an accumulation of people like jails, camps and surrounded cities. [379]

What about the role of climate? Was typhoid fever dependent on altitude, humidity and heat, as periodic pernicious fevers were?

Physicians agreed on the non-climatic determinism of typhoid fever, as I will show in the following sections. However, at least one physician strongly supported its geographical cause. Agustin Uribe, professor of hygiene at the Bogotá medical school, wrote a paper on the hygienic conditions of Bogotá in 1873. His purpose was to deny the causal role of miasmas, and argue that altitude was the main cause of typhoid fever in Bogotá. In Uribe's view, Bogotá's climate was unhealthy and had fatal effects on people's health. Given Bogotá's altitude and the consequent diminution of atmospheric pressure, physiological functions slowed down. This condition, added to sudden changes in temperature, formed the main causes of typhoid fever. Filth only acted as a predisposing cause. Thus, for Uribe, the disequilibrium between the exterior forces (altitude, atmospheric pressure, and temperature) and the physiological functions was the area where physicians should look for the morbid agents of typhoid fever, and not the miasmas.[380] But as I mentioned earlier, this view was less widely accepted than the idea of the non-climatic determination of typhoid fever.

Medical geography and typhoid fever: antagonism between typhoid and intermittent fevers - I

In previous chapters I described how the Colombian medical community had consolidated the medical geographical approach to diseases by 1860, and explained how doctors saw, in the intermittent fevers of the warm lands (yellow fever and malaria), the best cases of climatic and geographical determination of diseases. They linked the study of the local pathologies, from the medical geographical perspective,

[379] Similar views appeared in Antonio Vargas Reyes, "Señor doctor Eloy Ordóñez. –Paris". *Gaceta Médica*, Serie I, No. 4, 1 de septiembre, 1865, p. 14.
[380] Agustin Uribe, "Condiciones hijiénicas de Bogotá", *Diario de Cundinamarca*, Año IV, 999, 9 de abril, 1873, p. 572.

to their project for creating a national medicine that would grant them the sort of social legitimacy that they lacked at that time. Following the same rationale, they also supported the idea that typhoid fever "shows a peculiar character in Colombia with regards to the aetiology, age, and climate."[381]

For example, Antonio Vargas Reyes and Abraham Aparicio claimed that French authors' idea of typhoid as rare in individuals over the age of 40 years was contradicted by their experience in Colombia. Through autopsies, Vargas Reyes was said to have found typhoid fever in people of all ages.[382] Posada Arango calculated that approximately 14% of typhoid cases occurred in people in their 50s or above. Manuel Vicente de la Roche had also observed that Louis's principle, that typhoid did not occur in individuals after their 50s, did not describe what he had seen in Colombia. [383]

Another peculiarity of typhoid fever in Colombia highlighted by physicians was the role of climatic or local conditions. According to the French, changes in climate and habits would account for the higher susceptibility of foreigners, and men from the countryside that came to Paris, compared to Parisians. In Bogotá, Vargas Reyes said, the opposite was the rule: locals in Bogotá were the main victims of typhoid, whereas foreigners were rarely affected.[384]

The third, and probably the most important, argument in favor of the peculiarity of typhoid fever in Colombia relates to medical geography. The best-known French medical geographer of the time, Jean Boudin, had stated that where intermittent fevers occurred - e.g. swamps - typhoid fever was scarce. This was because of what Boudin called the laws of the geographical distribution of diseases. In his 1843 *Essai de Géographie médicale*, Boudin explains these laws:

> We would name by the law of *geographical affinity or coincidence* the situation in which two morbid forms are endemic in one locality and occur either parallel or consecutively. We would name by the law of *geographical antagonism* ... the situation

[381] Vargas Reyes, "Señor doctor Eloy Ordóñez", p. 13.
[382] Ibidem.
[383] Andres Posada Arango, "La fiebre tiofidea en Medellín", *Gaceta Médica*, Serie I, No. 10, 2 de marzo, 1866, p. 39.
[384] Vargas Reyes, "Señor doctor Eloy Ordóñez, p. 13.

> in which some pathologies do not occur in the same locality because of their own endemic characteristics. [385]

So, the more that one finds an endemic disease in a particular place, the greater the chances of finding related diseases and the lesser the chances of finding antagonistic diseases. The overlap of occurrence of yellow fever, cholera and plague would be an example of the law of geographic coincidence; the relationship between marshy fevers and tuberculosis and typhoid fever would be an example of the law of geographic antagonism. In the last case, the geographic or pathologic antagonism is a reference to the old idea that the marshy localities may have a healing effect on those who suffer tuberculosis, and a preventive effect with respect to typhoid fever. Marshy lands would somehow modify the organism, thus making it immune to tuberculosis and typhoid fever.[386]

The evidence that Boudin presented for the antagonism between intermittent and typhoid fevers came from the experience of the French military expedition to Morea in 1828 and his own experience in several regions of Europe and Africa:

> If we consider that M. Roux, head of the medical expedition to Morea, does not mention anything about it [typhoid fever], one can conclude that *dothiénenterie* [typhoid fever] is extremely rare in localities where marshy fevers occur. This opinion, based on observations in Africa and on careful examinations of thousands of people evacuated from Morea, Algeria, and Senegal to France, seems to authorize us to convert this opinion into law.[387]

For the Colombian doctors, Vargas Reyes and Posada Arango, Boudin's law of antagonism between marshy fevers and typhoid fever was not applicable in Colombia. Typhoid fever, Vargas Reyes said, "goes hand in hand with periodic fevers"; furthermore, he affirmed that the fever was not determined by climate:

> Typhoid fever has become generalized in the *República* and climate does not have any influence in its production; because it is as common in this savannah, where

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[385] J. Ch. M. Boudin, *Essai de géographie médicale or études sur les lois qui président a la distribution géographique des maladies, ainsi qu'a leurs rapports topographiques entre elles. Lois de coïncidence et d'antagonisme* (Paris: Germer-Bailliére/Ladé, 1843), pp. 76-77.
[386] Brunache, "Recherches sur la phtisie pulmonaire et la fièvre typhoïde considérées dans leurs rapports avec les localités marécageuses" in J. Ch. M. Boudin, *Etudes de géographie médicale. Notamment sur le question de l'antagonisme pathologique* (Paris: J.-B. Baillière, 1846), pp. 21-22.
[387] Boudin, *Essai de géographie médicale*, p. 20.

> temperatures are cold, as it is in the Cauca and Antioquia provinces, of moderate temperament, and the Coasts and the Magdalena, of burning climate.[388]

Observations by physicians living in other regions added to this argument against Boudin's law of antagonism between typhoid and marshy fevers. Andres Posada Arango showed that in rural areas surrounding Medellín - the capital of the Antioquia Province - where the "paludean" influence was strong, typhoid fever was also endemic. When both fevers occurred simultaneously in one person, he said, the fever took the form of remittent fever. Thus, concluded Posada Arango, Boudin's geographical antagonism did not exist in Colombia.[389] With regards to causes (filth and agglomeration of people), symptoms and prognosis, Vargas Reyes and Posada agreed that Colombian typhoid fever was similar to the European form.

Mainstream doctors indirectly demonstrated the non-climatic/non-geographic determinism of typhoid fever by describing cases that contradicted Boudin's law of geographical antagonism. But at least one doctor found possible answers in medical geographical determinants as to why typhoid occurred in places where causes such as the agglomeration of people and filth were absent. Antonio Vargas Vega claimed in 1866 that, at the beginning of his career, when he only had medical experience of typhoid fever in Bogotá, he accepted the causal association between filth, agglomeration and the fever. But after having traveled and lived in many other places, this opinion had been challenged: he found that typhoid fever occurred not only in unhealthy places like Bogotá, but also in healthy places with clean water like the Savannah of Bogotá, the Sogamoso valley and the Santa Rosa Valley. Conversely, he never encountered typhoid in other filthy towns such as Socorro. Socorro was a settlement of around 15,000 people, located in the province of Santander in the north-east of Colombia. Vargas Vega practiced medicine there for three years, and described the town as of a humid climate, where people not only threw filth in the streets but also lived under unhealthy conditions: houses were low, humid, ill-ventilated and infected. In addition, the inhabitants of Socorro were weak and of sickly complexion, undermined by cretinism and anemia. While syphilis, elephantiasis and smallpox occurred in the town, intermittent fevers, dysentery and

[388] Vargas Reyes, "Señor doctor Eloy Ordóñez, p.13.
[389] Posada Arango, "La fiebre tiofidea en Medellín", p. 39.

typhoid fevers were totally unknown. Given these circumstances, Vargas Vega wondered if the hypothesis that typhoid was associated with a certain kind of soil of modern alluviums, launched in the Academy of Science of Paris, was correct.[390]

Antagonism between typhoid and intermittent fevers - II

The debate on the geographical antagonism between typhoid and periodic fevers lasted throughout the 1870s and early 1880s in Colombia, but had a peculiar twist. Physicians were not concerned by typhoid occurring alongside periodic fevers in the marshy lands (lands like those of the French colonies), but, conversely, they struggled with the possibility of periodic fevers occurring in the highlands (Bogotá) alongside typhoid fever.

Thus, the physicians of the medical society of Bogotá (SMCN) participated in an intermittent debate on that issue, beginning in 1874, which lasted until 1882. Several problems were put forward: the issue of the antagonism between paludic and typhoid fevers; the strange possibility of periodic paludic fevers in Bogotá; the usefulness of clinical thermometry; and, finally, the role of "ferments" in causing typhoid fever. The debate began with a case presented by the physicians Pizarro and Aparicio before the SMCN in May 1874. It was of a 22-year-old man "of robust constitution, bilious-sanguineous temperament and good habits" who had chills followed by fever, headache and discomfort. At the physical examination he presented with a pulse of 120, dry and "burning" skin, dilated pupils, pain in the iliac cavity, reddish conjunctiva, photophobia, vomiting and headache. Pizarro and Aparicio diagnosed typhoid fever of bilious form and treated him with emetics. Symptoms worsened, with delirium and stupor followed by a short improvement. A few days later his symptoms recurred, with vomiting, headache, a pulse of 110 and delirium.[391] Having failed to find any organic lesion that could explain this second episode of fever, Pizarro and Aparicio diagnosed remittent fever of pernicious character and treated

[390] Antonio Vargas Vega, "El colera morbo", *Gaceta Médica*, Serie I, No. 8, 3 de enero de 1866, pp. 31-32.
[391] A. Pizarro and A. Aparicio, "Observacion de los señores doctores Aparicio y Pizarro", *Revista Médica*, Serie II, No. 15, 18 de julio, 1874, p. 123.

the patient with quinine sulfate.[392] Despite this treatment, the symptoms continued until the young man died. The most striking issue for Pizarro and Aparicio, and their reason for presenting the case before the medical society of Bogotá, was that it was a case of continuous fever followed by remittent and pernicious fever. This very fact contradicted Boudin's antagonism law:

> We were very surprised with this case … because it consisted of two fevers occurring consecutively in one person and some authors consider that these fevers exclude each other. This truth [of antagonism between intermittent and typhoid fever] proclaimed by M. Boudin as demonstrated by experience, would have led him to argue that the longer and more sustained the miasma action, the stronger the typhoid fever. Our case contradicts this appreciation.[393]

By displacing the context of Boudin's law of antagonism from the marshy lands of the French colonies to the highlands of the Colombian Andes, Aparicio and Pizarro declared that Boudin's antagonism was false.[394]

Pizarro's and Aparicio's diagnosis was opposed by members of the medical society. Proto Gómez and Rocha Castilla believed that the case was not of typhoid followed by a remittent fever, but of typhoid with recrudescence. Gomez argued that the failure of quinine to cure the remittent fever was proof enough to conclude that the patient never suffered such a disease.[395] Castilla agreed with this inference but added further arguments. In his view, the narrow variation of 10 units in pulse did not justify the diagnosis of two different diseases; besides, he said, typhoid fever was frequently preceded and followed by intermittency which had nothing to do with the "paludic infection" or deleterious actions of effluvia. Remittent bilious fever was, in his view, very rare in Bogotá.[396] Indeed, by the 1860s, doctors had more or less agreed that intermittent or remittent pernicious or paludic fevers were very rare in Bogotá's climate; they were supposedly typical of the low warm lands of the Magdalena River. There was a possibility that benign intermittent fevers might occur in the uplands, but pernicious intermittent (paludic) fevers would not.

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[392] Sociedad de Medicina y Ciencias Naturales-SMCN, "Sesión del día 9 de mayo", *Revista Médica*, Serie II, No. 19, 1 de agosto, 1874, p. 131.
[393] Pizarro and Aparicio, "Observacion de los señores doctores", pp. 123-4.
[394] SMCN, "Sesión del día 9 de mayo", p. 133.
[395] Proto Gómez, "Señores miembros de la Sociedad de Medicina de Bogotá", *Revista Médica*, Serie II, No. 15, 18 de julio, 1874, pp. 124-125.
[396] SMCN, "Sesión del día 9 de mayo", pp. 131-2.

Pizarro's and Aparicio's diagnosis was only supported by one member of the SMCN, Plata Azuero. Azuero actually saw, in the case of the young man, an illustration of the fact that pernicious fevers could indeed occur in the highlands: in his view, the case was typhoid followed by cerebral paludic fever. He argued that when a case of fever and cerebral symptoms occurred in the warm climates it could be readily diagnosed as "pernicious" fever, but the same case in a colder climate such as Bogotá's could very possibly be pernicious fever (so long as continuous fever and cerebral inflammation had been ruled out). Azuero's rationale was that just because pernicious fevers were uncommon in the cold climates of the highlands, it did not mean that they could never happen in those places; similarly, it could not be affirmed, he continued, that just because typhoid was rare in warm countries, it could not occur in such places. He also discredited the argument about quinine. If quinine failed to treat the intermittent fever in the case discussed, it was not because the young man had not suffered intermittent fever, but because quinine was not an infallible and specific treatment for such a fever. In other words, quinine did not have diagnostic value. [397]

The members of the SMCN were divided and the problem remained unsolved. None of them considered the causes of the fever to be a valuable element to the discussion.

In the following years, some physicians continued to address the problem but introduced a new element: clinical thermometry. Karl Wunderlich, the founder of this method, had published his famous monograph on clinical thermometry in 1871, which included twenty years of research into temperature and fever in Leipzig.[398] It is difficult to say when clinical thermometry was introduced into Colombia, but there is evidence that by 1877, it was a common practice in the wards of the university hospital, *San Juan de Dios*, in Bogotá.[399] Clinical thermometry convinced Colombian doctors that clinically, typhoid and typhus were two different diseases,

[397] Ibid., p. 133.
[398] Dale C. Smith, "The Rise and Fall of Typhomalarial Fever", *Journal of the History of Medicine and Allied Sciences*, 37, 1982, pp. 294-6.
[399] Abraham Aparicio, "Los baños fríos en el tratamiento de la fiebre tifoidea", *Revista Médica*, Serie IV, No. 42, 10 de diciembre, 1877, pp. 343-346.

and that they were both endemic in Bogotá.[400] Nicolás Osorio explained in his 1878 lectures that "thermometric lines in paludic, typhoid and typhus fever are different"; in his view, "thermometry would give us a new sign that would differentiate between *dotinenteria* [typhoid fever] and typhus fever, as well as providing a new way to demonstrate the diversity of fevers that could exist in a locality. That method would reduce any bias derived from the theoretical choices of the observer."[401] Although Nicolás Osorio claimed that he had demonstrated that typhoid and typhus were endemic in Bogotá by performing autopsies since 1866, it seems that it was only with the adoption of clinical thermometry as an everyday practice in the *San Juan de Dios* hospital that doctors began to diagnose both diseases, as I will show later.[402]

Physicians applied clinical thermometry to the problem of the coincidence of paludic and typhoid fever in both the warm lands and the highlands. For example, José Tomás Henao, working in Sonson (a temperate village in the Province of Antioquia), treated a 44-year-old doctor who had both remittent pernicious fever and typhoid fever. Henao diagnosed the latter mainly on the basis of Wunderlich's axioms: "any disease that has not reached 39.5ºC by the afternoon of the fourth day is not typhoid fever" and "any disease that reaches temperatures of 40ºC on the first or second day is not typhoid fever". For Henao, this case illustrated that typhoid and pernicious fevers could be present at the same time; Boudin's law of geographic antagonism between these two fevers was, in his view, contradicted by this case.[403]

Referring to the same problem, but in colder climates similar to the highlands, Evaristo Garcia wrote in 1876 from Paris (where he was studying medicine) that intermittency in fevers did not equal paludic influence. Typhoid fever could occur with intermittency, especially at the beginning, as it had been confirmed by the thermometric line. Fever was angular and went down in the mornings and then up in the afternoons. It was only after five or six days that temperature reached a maximum

[400] Nicolás Osorio, "Trazados termométricos de la fiebre tifoidea (dotinenteria) y del typhus fever (tifo-exantemático). Diferencia de estos trazados", *Revista Médica*, No. 43, 25 enero, 1878, p. 353. Osorio believed that the cause of both fevers seemed to be the same; however, special conditions were found in the organisms that caused them to change in effect, which was why whenever typhoid fever developed, typhus also occurred.
[401] Ibid., p. 351.
[402] Ibid., pp. 351-2.
[403] José Tomás Henao, "Observación de una fiebre tifoidea seguida de remitente perniciosa de forma diaforetica", *Revista Médica*, Serie IV, No. 47, 26 de mayo, 1878: 386-387; No. 48, 27 de julio, 1878, pp. 395-397.

and sustained its level, only to descend slowly until it disappeared completely. This intermittency was not associated with vegetable miasmas that caused paludism, since, for Garcia, typhoid occurred in cold places where those miasmas did not exist.[404] Abraham Aparicio held similar opinions. He believed that typhoid fever was not a miasmatic disease and that miasmatic fevers did not occur in Bogotá.[405] By the end of the 1870s, the balance seemed to incline in favor of the impossibility of the simultaneous occurrence of paludic fevers and typhoid fever in Bogotá.

Typhoid and intermittent fevers: medical geography and ferments

The SMCN readdressed the problem of typhoid and intermittent fevers in Bogotá in 1878. By then the SMCN had agreed that in Bogotá there had been cases of typhoid fever with remittance, demonstrated by thermometric measurements, and that this remittance had nothing to do with paludic miasmas, as I discussed above. However, in contradiction to his statement from a year before, Aparicio returned to the question by declaring that "this does not mean that intermittent and remittent or paludic fevers cannot originate in Bogotá".[406] Debates on paludic and typhoid fever were no longer framed in terms of Boudin's geographical coincidence and antagonism of diseases. Instead, older medical geographical ideas and Pasteurian notions of fermentation were brought together to make sense of the possibility. This happened before the ideas of the "golden age of Pasteurism" (that is, before Pasteur and Koch aimed to extend the germ theory to diseases other than anthrax in 1878) arrived in Colombia.

The second round of the debate began with Rocha Castilla's report to the SMCN in 1878 of possible cases of typhoid fever in Bogotá. Owing to the lack of typical symptoms (pain in iliac cavity, eruptions and skin spots) Rocha Castilla finally presented them as of the remittent fever of warm climates. Other physicians claimed to have seen similar cases, and Renjifo believed, for example, that it was not just the remittent bilious fever of the warm climates but "the intermittent fevers of the kind

[404] Evaristo Garcia, [Carta a la Revista Médica, Paris, abril 7 de 1876], *Revista Médica*, Serie IV, No. 38, 15 de junio, 1876, p. 315.
[405] Abraham Aparicio, "Los baños fríos en el tratamiento", p. 345.
[406] Abraham Aparicio, "Sociedad de Medicina. Sesión del día 4", *Revista Médica*, Serie V, No. 49, 14 de octubre de 1878, p. 411.

that from the pathological point of view we could reasonably believe to be in the riverbanks of the Magdalena River".[407]

How could this be possible? Insofar as the diagnosis of both diseases, typhoid and paludic fevers, were based on symptoms, either of the two diagnoses could be considered plausible by doctors. Liborio Zerda alone offered an explanation for this possibility; he focused on the relationship between miasmatic diseases and the geological and climatic characteristics of the city. Although Zerda attained his medical degree from *El Colegio Mayor de Nuestra Señora del Rosario* in Bogotá in 1853, he devoted his research to the chemical and physical sciences and taught them at the medical school.[408] This interest would explain why, in his 1875 paper "*Física Médica*" in which he adopted the medical geographical view of the hygienic conditions of Bogotá, he used Pasteurian notions of ferments. Interestingly, however, when he participated in the debate on paludic or typhoid fever in Bogotá three years later, Zerda did not use ferments or any other Pasteurian notions probably to emphasize the role of medical geographical factors, as I will show.

The main focus of Zerda's 1875 paper was air and its effects on health. In his view, the altered constitution of the atmosphere could weaken people's constitution and make them sickly. The accidental modification of the composition of the air might produce diseases, or give a distinct quality to all the illnesses that developed in a given moment. What kind of alteration of the air would produce such an effect? Here, Zerda reminded his readers of Tyndall's observation that air contained inorganic "atoms" and organic "*sporulos*", difficult to see with the naked eye, which traveled back and forth from the earth in rain. These organic "spores" were "animate fermentations", which produced astonishing transformations in organized bodies wherever they settled, helped along by heat and humidity.[409] "If we analyzed our atmosphere in the way that Tyndall and Pasteur did", he continues, "we would discover an important increase of organic matter in air and other abnormal

[407] Ibidem.
[408] Cáceres, Humberto and Cuéllar-Montoya, Zoilo, *Academia Nacional de Medicina de Colombia. Sus Miembros* (Bogotá: Academia Nacional de Medicina, 1998), pp. 5-6.
[409] Liborio Zerda, "Física médica. Hechos Científicos." [Folletín], *Revista Médica*, Serie III, No. 29, 15 de agosoto, 1875: 236-242.

substances during this period [between February and April]."[410] During those months, rain was scarce and heat increased, and therefore the fermentation of animal and plant matter took place, mainly at the bottom of swamps when their detritus was exposed to evaporation. What produced this transitory state in Bogotá's atmosphere? Zerda linked what was happening in the "industry" of the lowlands with Bogotá's environment. In the lowlands, he described, forests were replaced by extensive meadows, artificial prairies, and new crops. For this purpose, forest, dry grass and other corps were all burned. The smoke would ascend from those valleys to the highlands in the Savannah de Bogotá where the atmosphere was lighter, and would cover the city with a blue veil that made it hard to breathe. The smoke not only introduced improper matter into the body, but also heated and expanded the air, and the consequent delay of rain, dryness and swamp fermentations all had dreadful consequences. The matter in air that lungs could not filter may have passed directly to the blood, poisoning it and developing side effects that were generically labeled "miasmatic diseases". Breathing in an ill atmosphere also gave a destructive character to diseases that usually had a benign outcome (such as flu, angina, pneumonia, dysentery and measles).

This explanation of how miasmatic diseases were produced was restated, by its author, Zerda, during the 1878 discussions about paludic and other fevers in Bogotá. This time, he also added other medical geographical notions, such as the constitution of Bogotá's soil, in order to explain the "peculiar symptoms" of typhoid and typhus in Bogotá, as well as those of intermittent and remittent fevers. Indeed, he argued that Bogotá's geological constitution favored the putrefaction or fermentation of deposits of matter, thus periodically producing miasmas that infected the atmosphere.[411] Bogotá was situated on a permeable layer of soil which lay over an impermeable layer of clayey soil. Within these impermeable layers, rainwater formed a deposit that grew and diminished following the rainy and dry seasons; miasmatic exhalations grew and diminished alongside them. Besides, because of Bogotá's lack of a sewage system, organic matter that had been scattered in the streets and homes

[410] He also added that these microscopic beings, "which ferment and transform matter", could be halted by either filtrating them with cotton or burning them through a tube of hot platinum. Zerda, "Física médica", p. 240.
[411] Aparicio, "Sociedad de Medicina. Sesión del día 4", p. 412.

would be dragged down by rain towards the deposit, where they putrefied and fermented, thus producing miasmatic exhalations. This was how intermittent, remittent and typhoid fevers occurred. In other words, typhoid fever, like intermittent fevers, was caused by miasmas, though he did not say whether those intermittent fevers were of paludic nature.[412]

Zerda omitted ferments or any other reference to *sporulos* in his 1878 explanation. In fact, he completed his medical geographical argument by stating that the simultaneity of several epidemics could be explained by a specific atmospheric constitution. This argument was common among Colombian physicians of that period. For example, in 1879, Osorio explained the flu epidemic and some epizootics, which had developed simultaneously in Bogotá and in the savannah, using that mechanism.[413] I mentioned in Chapter 1 how, in 1881, Osorio and Gómez blamed the geological constitution (alluvium soil with vegetable matter), the seasonal conditions (the long dry season) and other climatic factors (the high temperature) for the production of the miasmas that caused two clinically different epidemics in the *sabana* of Bogotá: jaundice and *cholerina*.[414] For Zerda, similar factors accounted for the miasmatic diseases of Bogotá, among which he included typhoid and intermittent and remittent fevers.

Zerda's model left the door open for the possibility of typhoid and paludic fever in Bogotá. Before physicians introduced bacteriology into this debate, they provisionally resolved it by creating a new category: typhoid remittent bilious fever, which covered both fevers.[415] Indeed, in 1881, an epidemic occurred in Bogotá and nearby villages. The envoy of the SMCN who studied the epidemic, Alberto de J. Roca, diagnosed typhoid remittent bilious fever without using a thermometer or performing an autopsy. He was sure of the diagnosis because "if, like the majority of

[412] Ibid., pp. 412-3.
[413] Nicolás Osorio, "Epidemia de Gripa en Bogotá, en 1879", *Revista Médica*, Serie V, No. 56, Junio de 1879, pp. 467-471; 468-470.
[414] Nicolás Osorio y Proto Gómez, "Epidemias de ictericia y colerina en Bogotá y pueblos vecinos. – Fiebres epidémicas de la hoya del Magdalena.- Naturaleza de estas fiebres", *Revista Médica*, Serie VI, No. 61, 20 de mayo, 1881, p. 41.
[415] By the mid-nineteenth century, American physicians had also created a category that mixed typhoid and malaria fever, the typhomalarian fever, for the fevers typical of the region to the south of the Mason-Dixon line. This fever continued to be diagnosed and studied well into the twentieth century, between the Civil War and the World War I. See Smith, "The Rise and Fall of Typhomalarial Fever".

physicians, we believe that this is a typhoid remittent bilious fever, all cases must have the same intestinal alterations typical of that disease."[416]

The interesting aspect is that Alberto de J. Roca brought back the association between typhoid fever and filth. This association implied, in his view, that typhoid fever was not associated with the geological constitution of the soil as Liborio Zerda had concluded a few years before. For Roca, miasmas associated with a particular geological constitution were only "paludic" miasmas. However, Roca assumed that typhoid fever may have had a paludic element, probably because he needed to explain the remittent character of the typhoid fever; he placed typhoid fever alongside the bilious remittent fever of the warm countries. He did not discard the possibility that typhoid and the "true intermittent fevers" or paludic fevers could develop simultaneously or consecutively.[417]

To sum up, between 1874 and 1882 the debates over the continuous fevers of Bogotá centered on the problem of whether or not typhoid fever and paludic fever could occur in Bogotá, thus refuting Boudin's law that posited a geographical antagonism between them. The diagnosis of fever was based on symptoms; although physicians acknowledged that typhoid fever was distinguished by the lesions of Peyer's patches, autopsies were not always performed; clinical thermometry was not always practiced except in the university hospital (and only from 1877). With regards to causes, doctors either considered typhoid fever to be associated with filth (Roca) or to be a miasmatic disease associated with the climatic and geological conditions of Bogotá's soil (Zerda). Only Zerda, who was interested in chemistry, drew the analogy between miasmatic putrefaction and the Pasteurian notion of fermentation in order to explain not only typhoid fever but all "miasmatic" diseases. However, this analogy was not taken up by any other member of the medical community. And in Zerda's scheme, ferments were incorporated within the medical geographical rationale, without requiring any modification.

Germ theory in the debate around typhoid and intermittent fevers in Bogotá

[416] Alberto de J. Roca, "Fiebre tifoidea remitente biliosa. Estudio clinico", *Revista médica*, 76, 29 septiembre, 1882, pp. 146-147.
[417] Ibid., p. 148-151.

The SMCN commissioned Gabriel J. Castañeda to study Alberto de J. Roca's report on typhoid in 1882, but Castañeda waited until 1883 to respond to this request. In the meantime, the *Revista Medica* had been publishing extracts of debates in the Medical Academy of France, as well as a letter from a Colombian doctor studying in Paris about the latest discussions regarding typhoid fever.[418] Although these extracts, and the letter, focused on the therapeutic practice against typhoid fever, they also included some ideas on the novel cause that French doctors called the "ferment" or "microbe" and its possible route of transmission (mainly from the feces of sick people).

By the time Castañeda presented his report, he had converted to Pasteurism (see Chapter 5). He firmly supported the idea that typhoid fever was a "parasitic disease", "if it is assumed", he said, that "it is produced by a miasma which works through the microscopic organisms that they have in suspension". Castañeda gathered several arguments in favor of that hypothesis. For example, he remembered Bouchardart's pronouncement that microbes of typhoid fever were found in the skin spots. Most important for him was Klebs's work reproducing typhoid in rabbits, and his assertion that the microbe was only found in typhoid fever patients. The *bacillus* "*typhosus*", according to Klebs, would enter the body through the digestive and respiratory systems, producing the abdominal and chest forms of the fever.[419] In Castañeda's opinion, the "panspermic theory" would come to clarify many dark issues about the

[418] Avelino Saldarriaga, "Epidemia de fiebre tifoidea en Paris", *Revista Médica*, No. 81, 12 de febrero, 1883, pp. 395-6; Revista Médica-RM, "Etiología y tratamiendo de la fiebre tifoidea. Academia de Medicina de Paris. Sesiones del 7, 14, 21, 28 de noviembre y 5 y 12 de diciembre, 1882", *Revista Médica*, No. 82, 12 de marzo, 1883, pp. 438, 441-3; Juan E. Manrique, "Tratamiento de la fiebre tifoidea", *Revista Médica*, No. 83, 20 de abril, 1883, p. 488.

[419] Gabriel J. Castañeda, "Informe sobre el trabajo de E. de J. Roca titulado 'fiebre tifoidea remitente biliosa", *Revista Médica*, No. 87, 20 septiembre, 1883, pp. 83-4. The standard story is that in July 1880, Carl Eberth (1835-1926) published his discovery of short rodlike bacteria in the tissues of fatal typhoid cases. Neither Castañeda nor Eberth isolated the organism in pure culture. In 1881 Robert Koch published photomicrographs of rodlike bacteria from the tissue of fatal typhoid cases. Eberth and Klebs agreed that they and Koch had all seen the same organism. In 1881 Klebs reported that he had grown an organism in culture media that, when inoculated into laboratory animals, produced lesions. In 1884 Georg Gaffky was able to isolate the bacterium using gelatine culture media but was unable to produce evidence of disease when he injected the pure cultures into animals. Despite the failure to satisfy the 1882 criteria established by Koch, the rodlike bacterium – *bacillus typhosus* - became generally accepted as the causative organism of typhoid fever. See Smith, "The Rise and Fall of Typhomalarial Fever", p. 299.

development of miasmatic diseases, given the extraordinary variety of germs that are disseminated on the surface of the earth.[420]

In the view of several members of the SMCN, Castañeda's report missed the point. Osorio insisted, for example, that Castañeda did not address key questions such as "do palustre fevers exist in Bogotá? Could typhoid fever take the form of an intermittent fever? Is there any form of typhoid fever that is not produced by the *miasma tifico*?"[421]

Osorio insisted that the main problem was not the identification of the cause of typhoid fever, as Castañeda reported, and he showed a strong skepticism of Pasteurism. For Osorio, Pasteurians placed too much emphasis on antiseptic medication. Castañeda responded to this accusation, insisting that typhoid fever should be treated by antiseptic methods even though it had not been fully demonstrated that it was indeed caused by bacteria; although the disease had been reproduced in animals but not in humans, Castañeda said, "I completely support Mr. Pasteur's theories".[422]

So, despite Castañeda's confidence in the "panspermic" doctrine, none of the members of the SMCN considered that this theory was able to provide an answer to the main problem: whether the type of fevers in Bogotá was typhoid or paludic. They were right: Castañeda did not address that problem, and neither had he extended the "panspermic" theory to paludic fevers.[423]

A provisional closure of this debate occurred in 1884 when Aparicio finally incorporated germs as the criteria that would clarify the nature of Bogotá fevers. He summarized the "parasitic doctrine" as the theory of live elements which followed the universal laws of life (they were alive themselves and would also take life from other organisms). This fight with other organisms was the pathological process of virulent and contagious diseases.[424] After summarizing the basic idea of vaccines, and the therapeutic and hygienic implications of the "parasitic doctrine", Aparicio stated that "pathology has found in it [the parasitarian doctrine] the explanation for

[420] Castañeda, "Informe sobre el trabajo de E. de J. Roca", p. 84.
[421] Ibid., p. 93.
[422] Ibid., p. 94.
[423] Ibid., p. 95.
[424] Abraham Aparicio, "Discursos pronunciados en la sesión solemne del 27 de febrero de 1884", *Revista Médica*, N. 93, 20 de marzo, 1884, pp. 377-8.

many apparent disagreements with the clinic", referring to the nature of fevers in Bogotá. Indeed, Aparicio suggested that the variety of climates, temperatures, humidity and atmospheric pressures of the country explained why the atmosphere played a key role in the variety of diseases, for all those elements would influence the "many germs which develop in it": "that is probably why Bogotá's *piretología* takes the strangest forms", he continued. Germs in the atmosphere varied according to the environment, and also according to the organism's conditions, which was why (continued Aparicio) it was not surprising that "two different germs develop in the same organism and therefore we would never again reject the idea that typhoid fevers could be followed by remittent pernicious fevers". [425]

The evidence available suggests that the discussion surrounding Aparicio's idea that pernicious fevers, like typhoid fever, were also caused by germs and therefore could also be found in Bogotá was the last time that physicians in the city discussed the possibility of pernicious fevers occurring in Bogotá. It is possible that the growing differentiation that doctors were establishing between paludic fevers and the epidemic fevers of the Magdalena river, identifying the former as paludic fevers typical of the lowlands (and the latter mobile), might have contributed to the decline of the debate. In 1893, Professor Juan de Dios Carrasquilla would state that because Bogotá did not have the necessary temperature for the development of Laveran's "protozoario", intermittent (or paludic) fevers could not happen in the city.[426] As such, for the rest of the 1880s and the 1890s, the discussion of fevers in Bogotá centered on typhoid fever and typhus.

Germs as miasmas in clinical and hygiene practices in Bogotá

Although by the mid-1880s doctors were still using the notion of *miasmas tifogenos* in referring to the cause of typhoid fever, and some even associated them with the removal of old cemeteries,[427] the available evidence suggests that by the time the

[425] Aparicio, "Discursos pronunciados", pp. 381-2.
[426] Juan de D. Carrasquilla, "Consideraciones acerca de la etiologia y de la profilaxis del paludismo", *Anales de la Academia Nacional de Medicina*. Tomo I-Entrega Primera (Bogotá: Imprenta de "La Luz, 1893), p. 6.
[427] JCH, "Sesión del día 11 de julio de 1887", *Revista de Higiene*, No.5, 14 de julio, 1888, pp. 75-6.

third epidemic of typhoid fever occurred in 1887, they had incorporated the "parasitic theory" into their clinical and hygiene practices. I have described how, in previous decades, some physicians had associated typhoid fever with filth. This association became common after the 1887 epidemic; the hygienic measurements that were recommended and put into practice by physicians and physicians-hygienists (as well as by local authorities) during the epidemic revolved around this association as well as Pasteurian methods against infectious diseases.

The epidemic was announced by the mayor of Bogotá, Higinio Cualla, in September 1887 before the recently created central hygiene bureau (JCH). He reported an increase in cases of typhoid fever in the *San Juan de Dios* hospital. Before asking for advice from the JCH, the mayor had ordered police inspectors to improve the cleaning of the city and the disposal of rubbish, because he was convinced that filth, feces and garbage were strongly associated with any epidemic in the city. Since the 1870s, the cleaning and salubrity of the city had been the responsibility of the police force, a body of inspectors who also had to take care of the security of the city. Although considerations regarding hygiene issues had been part of the police code since 1829, it is very likely that an actual body of inspectors for Bogotá was created around the 1870s, when the city council was allowed to tax properties and industry and thus had money to hire private contractors or to pay functionaries.[428] During the 1870s and 1880s the local council and police force worked from the idea that filth and organic putrefaction produced miasmas, which caused almost all of the city's epidemics. In 1877, for example, the police force of the city had to check sewers and streams containing stagnant material "whose putrefaction produce fetid or deleterious miasmas".[429] Throughout the 1880s people usually threw corrupted waters, filth and feces onto the streets.[430] In 1879 the inspector of the *Aseo*, *Ornato*, and *Salubridad* areas of the city prohibited people to throw filth into streams and streets since "as a consequence of the lack of cleanliness

[428] Eujenio Gonzalez Benito, "Informe del Tesorero del Distrito sobre la situacion fiscal de este", *Rejiistro Municipal*, Ano I, No. 1, Bogota, 1 de octubre de 1874, pp. 2-3.
[429] Registro Municipal-RM, "Acuerdo numero 22 de 1877 que organiza la policia de la ciudad", *Rejistro Municipal*, No. 54, Bogota, 1º de agosto de 1877, pp. 229-233.
[430] Manuel Solanilla, "Memoria prestada por el alcalde del distrito a la municipalidad de Bogotá en el día de su instalacion", *Rejistro Municipal*, No. 204, Bogota, 10 de marzo de 1884, pp. 843-4; Manuel Forero, "Informe del Inspector general", *Registro Municipal*, No. 257, Bogota, 30 de octubre de 1885, pp. 1056-7.

in the streets, houses, and plazas, epidemic and deadly fevers have emerged causing excessive mortality".[431] The mayor of the city was worried about similar issues during the 1887 epidemic.

Higinio Cualla blamed the *Hospital San Juan de Dios* for the increase in the "typhus" death toll (14 out of 22 casualties died in the hospital). Cualla denounced the lack of disinfection in the hospital wards and also the fact that corpses were left unburied in the anatomy amphitheatre for seven days in one case, ten in another. The cart that transported corpses to the cemetery also left traces of blood, flesh and bones which produced an unbearable smell.[432]

Liborio Zerda explained to the mayor that the majority of cases of fever occurred not in the hospital, but in the surrounding neighborhoods. Since the disease mostly affected poor people, who did not have anywhere else to go, they were sent to the hospital already infected.[433] The professors of anatomy, on the other hand, responded to Cualla's accusations by claiming that they did not dissect corpses of those who died from dysentery, typhoid or any other contagious diseases[434] and that the wards in the hospitals were disinfected. Josue Gomez remembered that chamber pots were all washed with iron sulfate, and that beds, once vacant, were cleaned with carbolic acid.[435] These practices were based on the germ theory perspective: the use of antiseptic substances for disinfecting feces, which were then considered the main route for the transmission of typhoid fever.

A similar approach was recommended by members of the JCH to the mayor of the city, in order to halt typhoid transmission. Not only should there be public latrines, for those who lacked sewage systems, but the authorities should also encourage people to get rid of filth, even if it were by means of throwing it into the

[431] Javier Bergara Esguerra, "Resolución por la cual se ordena el cumplimeinto de ciertas disposiciones de policia", *Rejistro Municipal*, No. 86, Bogotá, 1 de febrero de 1879, p. 363.
[432] JCH, "Sesión del día 28 de noviembre de 1887", *Revista de Higiene*, 6, 25 de agosto, 1888, pp. 124-5.
[433] JCH, "Sesión del día 5 de diciembre de 1887", *Revista de Higiene*, 6, 25 de agosto, 1888, pp. 125-30.
[434] JCH, "Continúa. Sesión del día 5 de diciembre de 1887", *Revista de Higiene*, 7, 25 septiembre, 1888, pp. 131-2.
[435] JCH, "Sesión del día 5 de diciembre de 1887", *Revista de Higiene*, 6, 25 de agosto, 1888, pp. 127-9.

nearest running water. For those who were ill, Osorio recommended compulsory disinfection of feces with carbolic acid.[436]

The hygienic actions mentioned above were, like those in the hospital, authorized by bacteriological principles of disinfection; however, in the realm of causal speculations, physicians and members of the JCH continued to use the old miasma terminology. For example, Jorge Boshell, in his 1889 thesis on the continuous fevers of the *Hospital San Juan de Dios*, claimed that "everybody knows that the fever originated in the emanations awoken by the removal of land during the construction of sewers", from where it expanded by infecto-contagion and inoculation.[437] And in September 1888, the JCH claimed that "the city is under the influence of typhoid miasmas and others of exceptional gravity".[438] In the same year, however, the *Revista Médica* published a summary of the bacteriology of typhoid fever. The summary offered information on Eberth's and Kleb's typhoid bacillus and their findings from autopsies of *dotinentericos*, concluding that "the specific and pathogenic role of Eberth bacillus is firmly established". [439]

After a year of diminution, the number of cases of typhoid increased again in 1889. The epidemic began in February in Facatativa, a village near Bogotá, and spread to the capital city. In April there were 114 cases of typhoid fever in the *Hospital San Juan de Dios*, out of 709 patients.[440] By this time, germs had fully entered causal considerations, but in a particular way. Castañeda, who proposed the "panspermic" cause for typhoid in 1883, believed that one of the causes of this recrudescence was the accumulation and stagnation of feces in pipes. Rain during the previous months had flushed out an immense mass of organic matter, thus throwing innumerable "tifogen" germs out of the mouths of latrines and drains and through pores in the ground , which were the specific cause of the disease.[441] Such "organic

[436] JCH, "Sesión del 1º de octubre de 1887", *Revista de Higiene*, Año 1, No. 5, 14 de julio, 1888, pp. 95-98.
[437] Jorge Boshell, *La fiebre continua del hospital de San Juan de Dios*, Tesis para el doctorado en Medicina y Cirugía (Bogotá, Imprenta Echeverría, 1889), p. 10.
[438] JCH, "Sesión del día 23 de abril de 1888", *Revista de Higiene*, 7, 25 de septiembre, 1888, pp. 160-161.
[439] RM, "El bacilo de la fiebre tifoidea", *Revista Médica*, No 123, 20 de febrero, 1888, pp. 1044-1053.
[440] JCH, "Sesión del dia 15 de abril de 1889", Revista de Higiene, No 11, 30 mayo, 1889, p. 270.
[441] JCH, "Sesión extraordinaria del 9 de mayo de 1889", *Revista de Higiene*, No 11, 30 de mayo, 1889, pp. 283-293.

germs" could stay lifeless for a long time in dry places, he said, but once the environment became humid they would recover their force and reproduce.

In Castañeda's model, the specific cause, germs, were subordinated to the neo-Hippocratic predisposing causes. In his view, "predisposing causes … could act upon the individual in order to favor the action of the specific cause". Castañeda judged that air was the main predisposing cause of typhoid fever. When it was not renewed, air was charged with effluvia, that is, with microbes and other elements, which could produce pernicious fevers in some places, and tuberculosis or *tifo* in others. Other predisposing causes were also considered by Castañeda, such as bad habits, food, and clothes. Bad intellectual, moral, sensitive and organic acts would also predispose individuals to the contagion, either by affecting their natural functions or by loosening their control over voluntary acts.[442]

Osorio and Castañeda argued that causes of the typhoid epidemic were complex. The "germ" was the specific cause, which was mainly found in suspension in currents of water. Following a foreign author, Dr. Fergus, they affirmed that two conditions were necessary for the development of the disease: not only the "germ", but also animal emanations. If water was not contaminated by those emanations, germs would not develop; they stayed sterile and therefore the disease did not appear. Thus putrefaction of filth was the pre-condition for the development of the germ, they asserted. Under these conditions, *typhic* microbes developed and were carried to the surface of water deposits by the atmosphere. Another possible vehicle was milk that had been mixed with impure waters. Bad meat,[443] leather deposits,

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[442] JCH, "Sesión extraordinaria del 16 de mayo de 1889", *Revista de Higiene*, No. 11, 30 de mayo, 1889, pp. 294-295.

[443] Nicolas Osorio y Proto Gomez, "Epidemia de fiebre tifoidea", *Revista de Higiene*, 16, 30 de noviembre, 1889, pp. 475-6. This possibility was confirmed when new increases were reported by October 1890. This time Michelsen pointed to the cattle arriving from low warm lands. This conclusion was based on observations by the official veterinary Vericel and the fact that he found macroscopic lesions in the 4th stomach which, though different to those of typhoid fever, were considered to be true ulcerations. Local authorities immediately acted, after the JCH warned against the extension of the *enfermedades tificas*, by reinforcing inspections of the public slaughterhouse and making the cleaning of houses, streets and sewage compulsory. But the opinion of Michelsen, who was then working in the Ministry of Fomentation, that cattle could be the reason for the increase in cases also resulted in a close inspection of cattle for other departments by the official veterinarian, Claudio Vericel. The problem was that there was no proper office in which the veterinarian could work, and some protested that he would not have time to examine the cattle used for human consumption. More protests about the quality of meat led Michelsen, the Inspector of Police and the veterinarian to take a visit to the slaughterhouse. In Michelsen's words, "I do not know the exact words that could describe the untidiness, filth, and inhumanity that dominate there. The public

slaughter houses and filth deposits were also suitable environments for the development of *typhic* germs. Given the bad hygienic conditions of the city (with people throwing dirty water, urine and feces into the streets), Osorio and Castañeda said, the epidemic could have been worse had it not been for the "indulgent climate" of Bogotá.[444]

Colombian doctors tended to stress the role of sewage and air, rather than water, as the vehicle of the typhoid germ.[445] Borda, for example, said that "it is an established fact that the bacillus that produced typhoid fever, described by Eberth in 1888, is transmitted not only by water but also by air which drags it in dust or in the form of spores which can remain for long periods of time without germinating according to the humidity conditions [...] If water is the main vehicle of the propagation of typhus bacillus, it can also be found in sewage." It was the emanations of sewage, he concluded, that were the main cause of typhoid epidemics in Bogotá.[446]

So by 1889 germs were fully incorporated into causal speculations about the causes and prevention of typhoid. Hygienic measures inspired by Pasteurism were also implemented. Measures recommended by Osorio against the "*agente tifógeno*", the patient and his environment, reinforced antiseptic measures. Thus, based on the idea that stools of sick people, and probably also their urine and secretions, carried

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slaughterhouse of Bogotá is the biggest insult to nineteenth-century civilization and the most pernicious infectious focus". None of the previous measures ordered by the JCH, he declared, had been applied. "Gas from the sewage makes contact with the atmosphere where cattle is boned, so meat sits there for long periods of time, bathed in the pestilent and mephitic emanations from the waters of the San Francisco river with filth coming from Bogotá […] a huge amount of dogs smash and lick meat thus infecting it with the emanations from the filthiness of their bodies". See JCH, "Sesion del 17 de febrero de 1890", *Revista de Higiene*, 18, 13 de marzo, 1890, p. 535; JCH, "Sesion extraordinaria del 23 de octubre de 1890", *Revista de Higiene*, 24, 30 de octubre, 1890, pp. 91-95; continuacion, 25, 30 de noviembre 1890, pp. 97-102; Ibid., 97-100.

[444] Nicolas Osorio y Proto Gomez, "Epidemia de fiebre tifoidea", *Revista de Higiene*, 16, 30 de noviembre, 1889, pp. 475-9.

[445] Until the establishment in 1888 of water pipes which took water from the San Francisco River, the population used the colonial system of water supply which consisted of women known as "aguateras" or carriers of water who would collect water from several fountains connected to the *San Agustin*, *Fucha*, *San Francisco* and *Arzobispo* rivers by open pipes made of blocks. The water pipes built in 1888 still had sections that were open to the air. It would not be until the third decade of the twentieth century that the system was modernized, when chemical analyses were systematically performed and chlorination was introduced. Benjamín Villegas (ed.), *El agua en la historia de Bogotá, 1538-1937* (Bogotá: Villegas Editores, 2003).

[446] JCH, "Sesion del dia 16 de Julio de 1889", *Revista de Higiene*, Revista de Higiene, 12, 31 julio 1889, p. 354 and 13,14 and 15 de agosto de 1889, pp. 356-358; Nicolas Osorio, "Alcantarillas", *Revista de Higiene*, Revista de Higiene 13, 15 de agosto de 1889, pp. 384-386.

the "*tifogen* principle", he suggested that fecal matter and urine should be collected in receptacles with carbolic acid or bichloride of mercury before being sent to latrines. Where there were no latrines, stools should be disinfected and buried away from the room. Latrines also had to be disinfected with copper sulfate or calcium chloride. Corpses of the sick should be buried and wrapped in a cloth soaked in an antiseptic substance. Clothes, blankets and instruments that had been used to clean them should be soaked as well. In hospitals, blankets and couches had to be disinfected with vapor. Rooms had to be fumigated with sulfuric acid, cleaned and whitened. Doctors should visit typhoid patients in hospitals at the end of their rounds, with different coats on. Potable water, sewage systems, and cleaning of streets were also recommended.[447]

Other measures such as the creation of a hospital for typhoid patients, or even the isolation of them within the *San Juan de Dios* hospital, were also considered based on the contagious nature of typhoid;[448] the JCH also proposed the creation of a door-to-door medical service, dividing the city into sections and appointing one physician to each of them, with free medicine for poor people.[449] However, it seems that none of those proposals were put into practice.

"Like extras in a drama": germs vs. the spontaneous origin of typhoid fever

Although, during the epidemic years between 1887 and 1889, physicians finally incorporated germs into their causal speculations and used them as the foundation of hygiene practices, there were some who not only distanced themselves from Pasteurism, but also rejected germs as the specific and primary cause of typhoid fever. They defended the idea of the spontaneous origin of typhoid fever. This theory contradicted the core ideas of Pasteurism. Indeed, in Pasteur's view, the future of

[447] JCH, "Sesión del día 22 de abril de 1889", *Revista de Higiene*, No 11, 30 mayo, 1889, pp. 274-275.
[448] JCH, "Sesión del día 30 de abril de 1889", *Revista de Higiene*, No 11, 30 de mayo, 1889, pp. 276-277; JCH, "Sesi[on extraordinaria del 6 de junio de 1889", *Revista de Higiene*, Revista de Higiene, 12, 31 julio 1889, pp. 326-327; JCH, "Sesion del dia 2 de septiembre de 1889", *Revista de Higiene*, 14, septiembre, 1889, pp. 387-389; JCH, "Sesion del dia 30 de septiembre de 1889", *Revista de Higiene*, 15, 31 de octubre, 1889, pp. 424-427; JCH, "Sesion extraordinaria del dia 8 de octubre de 1889", *Revista de Higiene*, 15, 31 de octubre, 1889, pp. 436-8; JCH, "Sesion del dia 4 de noviembre de 1889", *Revista de Higiene*, 16, 30 de noviembre, 1889", p. 462.
[449] JCH, "Sesión del día 6 de mayo de 1889", *Revista de Higiene*, No 11, 30 de mayo, 1889, p. 280.

medicine depended on a life-and-death struggle against the traditional doctrine of the spontaneity of disease.[450]

Ignacio Osorio, one of the supporters of the spontaneous development of typhoid, claimed that typhoid fever was a putrid intoxication. Following the French author Jaccoud in his *Traité de Pathologie Interne*, Osorio summarized the three hypotheses for the origin of typhoid fever: the extrinsic origin, that is, when the poison invaded the organism via air, water or other substances; the spontaneous origin, that is, when the poison was born in the organism under the influence of bad conditions; and the contagious origin, where it was reproduced by the sick and transmitted to healthy individuals. Spontaneous generation was based on the idea that the organism held material of putrid poisoning in its intestine or exhalations, whose noxious influence was normally destroyed by tissues, by their rapid elimination, or by transformation and re-absorption. If for any reason these compensatory actions did not work perfectly, the putrid matter could become a source of the typhoid poison; in which case it could be said that typhoid fever was completely generated within and by the organism itself.[451] To this interpretation Osorio added the idea that this noxious substance was the *leucomanias*, whose poisonous action was septic and analogous to the physiological poison of reptiles. Similarly, typhoid poison would produce hemorrhages, diminution of fibrin, ataxia and gangrene. [452]

It is telling that Osorio ignored Jaccoud's affirmation in the earliest edition of his book of 1871: that the extrinsic origin of typhoid had already been demonstrated, and that knowledge of this fact limited the possibilities of spontaneous generation. The spontaneous generation of typhoid, Jaccoud said, should be admitted only by exclusion, when the cause was not imputable to another one. Ignacio Osorio did not recognize the subordination of the spontaneous origin of typhoid fever to the extrinsic origin. On the contrary, he pretended to compare Jaccoud's observations with the observations made in Colombia in order to support them.

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[450] Gerald Geison, "Pasteur" in Charles Coulston Gillispie (ed.), *Dictionary of Scientific Biography*, Vol. 9-10 (New York: Charles Scribner's Sons, 1981), p. 384.
[451] Ignacio Osorio, "Observaciones que pueden servir para el estudio de la causa o causas que generan la fiebre tifoidea", *Revista Médica*, 145, 1 de enero, 1890, p. 742. It is not clear which of the six editions of Jaccoud's book (published between 1870 and 1879) Osorio read. I checked the 1871 edition. S. Jaccoud, *Traité de pathologie interne*, V. 2 (Paris: Adrien de la Haye, 1871), pp. 726-728.
[452] Osorio, "Observaciones que pueden servir para el estudio de la causa", pp. 743-746.

First, Osorio justified the spontaneous generation of typhoid fever with medical geographical arguments. In the first part of this chapter, I described how Colombian doctors, from the mid-nineteenth century onwards, did not consider typhoid fever to be determined primarily by climate. Ignacio Osorio returned to this argument and argued that since "abdominal *tifus*" developed in all continents, "its cause must necessarily be in all places, in all countries, in almost all latitudes". This "ubiquity" of typhoid fever, for example, contrasted with the localism of yellow fever: yellow fever was found in hot climates, never beyond 48 degrees of latitude or more than 2000 meters above sea level. Besides, Osorio argued, although it had been recognized that the emanations from fermentations of an animal origin caused typhoid fever, it was also true that not all fermentations of the same origin would produce typhoid fever. So, if typhoid fever was determined neither by climate nor by a specific ferment, he wondered "if the cause rests in the human organization that under certain circumstances wakes up from its latent state and become visible".[453]

Osorio claimed to have made observations in support of this theory in Bogotá and down in the Magdalena River valley, to the south as far as the central *cordillera* and up to 3722 meters above sea level. He claimed to have seen *dotinenteria* occur, always in a spontaneous form, in places of different temperature and pressures, where there was no industrial waste that might putrefy or where altitude and temperature made it impossible for animal fermentation to occur. Besides, not only had he seen it in crowded places, but also in isolated houses scattered in the countryside. For Osorio, "the parasitary doctrine" – or "the specificity doctrine" - did not explain why typhoid fever appeared in all places. Thus, he concluded, "nobody today doubts the spontaneous development of *dotinenteria*".[454]

One opinion supporting Ignacio Osorio's spontaneous generations theory of typhoid fever appeared in the *Revista Médica*. This opinion was not made regarding *leucomanias* transforming into typhus poison, but regarding germs being spontaneously produced in the body. Written by an unknown author, it affirmed that typhoid was a cosmopolitan disease that "develops with absolute exclusion of thermometer, barometer, hygrometer, and even pluviometer"; its germs lived and grew from the poles to the equator; and it occurred wherever humans went, in

[453] Ibid., p. 742.
[454] Ibid., 743-746.

crowded places as well as in isolated country houses. In short, typhoid fever was not a telluric disease but instead originated in the human organism.[455]

Gabriel J. Castañeda, who had first proposed germs as the cause of typhoid fever in 1883, attacked the spontaneous generation theory of typhoid. In his 1890 paper, about the etiology and pathogenesis of typhoid fever, Castañeda argued that the spontaneous generation theory of typhoid fever was completely discredited by the development of experimental medicine. He asserted that science had demonstrated the existence of the microbe, the Eberth bacillus, which was considered the specific cause of typhoid fever. As proof he mentioned that when this bacillus was injected into animals, the same symptoms were reproduced. He also argued that contagiousness could not be explained by the spontaneous doctrine.[456]

Castañeda also said that the fact that typhoid could happen in any place, both isolated and crowded, was not strange because microbes could resist high and low temperatures and could keep their noxious properties for a long time. The "parasitary" point of view thus contradicted every argument made by those arguing for the spontaneous generation of typhoid fever. According to Castañeda, typhoid fever was a disease originating in infection by specific microbes, which had varied levels of virulence depending on their ability to produce toxins. It also reproduced quickly. In the body the "parasite" caused chemical reactions that exhausted the organism. Ptomaines explained ataxico-adynamic symptoms. Microbes were located in the intestinal tube, if they entered via ingestion, or in the lungs, if they entered via breathing. Subsequently they traveled in the blood and collected in different organs, forming morbid foci, which is why several organs were affected in this pyrexia. For Castañeda, there was no evidence to support the spontaneous generation doctrine. [457]

Despite Castañeda's opinion in favor of the "parasitary" doctrine of typhoid fever, physicians continued to resist bacteriology as the first line of explanation for the fever. Even one of the converts to Pasteurism, Abraham Aparicion, supported a more sophisticated version of the spontaneous origin of typhoid. Earlier I described how, in 1884, Aparicio provided a provisional closure to the debate over the

[455] RM, "Crónica", *Revista Médica*, 151, Julio, 1890, p. 123.
[456] Gabriel Jaime Castañeda, "Etiología y patogenia de la fiebre tifoidea", *Revista Médica*, 152, Agosto, 1890, p. 129.
[457] Ibid., pp. 130-2.

possibility of intermittent and typhoid fever in Bogotá, arguing that they were both caused by germs and therefore they could both occur in the city. However, when Aparicio intervened in the debate over the spontaneous origin of typhoid fever in 1890 he followed Peter, professor of internal medicine in Paris at the Charité Hospital and Pasteur's well known challenger at the Academy of Medicine.[458] According to Peter, "the majority of fevers are the result of the exaggeration or deviation of our functional acts". Peter explained that after hard work, physical, moral or intellectual, the body could develop a "typhic-like" fever when the natural inorganic products were eliminated in an incomplete form. This fever could become epidemic and develop into the true "typhus". Peter called this "auto-typhisisation". The Colombian doctor Aparicio believed that this was plausible. Aparicio declared that "it is therefore not surprising that we lack faith in the idea that the Eberth bacillus is the specific agent of typhoid fever in contrast to the thorough acceptance of this idea by Castañeda." "Our faith", he says, "would weaken even more if we considered the evidence for the polymorphism of parasites" - that is, their change in form and properties.[459]

Aparicio suggested that there were some physicians in Colombian who did not follow what he called the "exclusivist approach" or the reductionism of bacteriology in their clinical practice. Those skeptics believed that the new discoveries could not stand by themselves as scientifically valuable; they needed to be related to the "traditional" (i.e. clinical) medicine. It was there, at the bedside, that truth and progress must be sought, they said.[460] Of course, Aparicio aligned himself with those skeptics who accepted bacteriology only to a certain extent and simultaneously defended the spontaneous origin of typhoid fever. In his view, the sanction of Koch's discovery of the bacillus, based on experimental and anatomo-pathologial verifications, could not be used to deny the spontaneous morbidity of the organism to catch diseases (or, as Peter said, the capacity of the organism to create its own

[458] Anne Marie Moulin, "Bacteriological Research and Medical Practice in and out of the Pastorian School" in Ann La Berge and Mordechai Feingold, *French Medical Culture in the Nineteenth Century* (Amsterdam/Atlanta: GA, 1994), p. 328.
[459] Abraham Aparicio, "Auto-tifisasión espontánea", *Revista Médica*, 152, agosto de 1890, pp. 133-137.
[460] Ibid., p. 135.

microbes). In Aparicio's view, "parasites" ended up being "like extras in a drama" whose main plot was the spontaneous origin of typhoid. [461]

Typhus or typhoid: germs and social causes

I have discussed how although Colombian physicians began to recognize that typhoid and typhus fever were two different diseases around the 1860s, physicians, physician-hygienists, and local authorities used typhoid and typhus as interchangeable terms until the 1887-89 epidemics. That this distinction became relevant at that time, and that the role of Pasteurism in making the distinction was marginal in the clinical realm, is evident in two medical theses based on the study of cases at the university hospital *San Juan de Dios*.

Jorge Boshell's *La fiebre continua del hospital de San Juan de Dios* (1889) attempted to establish whether the 1887 fever that he called "fever of the hospital" was typhoid or typhus based on their symptoms, clinical thermometry and anatomo-pathology. He concluded that it was neither typhoid fever nor typhus but instead a "hybrid" formed by the pathological lesions of the *dotinenteria* and the clinical syndrome of typhus. For Boshell, the problem of aetiology was irrelevant; he simply asserted that the hybrid had originated in the emanations caused by the removal of land during the construction of sewers.[462]

The second thesis, Ruben Rodriguez's *Apuntes sobre la etiologia del tifo epidemico de 1889* (1890), centered not only on the type of fever that was occurring in Bogotá but also on "the conditions in which the *elemento generador* develops". With regards to the type of fever, Rodriguez claimed it was typhus and not typhoid fever. He based this conclusion on 43 autopsies, five of which had found the Peyer's patches altered. By the "conditions in which the *elemento generador* develops", Rodriguez meant social conditions. He associated typhus with two social groups: "those who need to work to survive" and "those who live at the expense of others". Insufficient and poor quality of food, overcrowding, dirtiness, excess of alcohol, and

[461] Ibid., pp. 136-137.
[462] Boshell, *La fiebre continua del hospital,* pp. 10, 57.

prolonged action of cold would explain why typhus was endemic among these two groups. In other words, typhus was a disease of the poor people. [463]

This endemic typhus became epidemic in 1889, claimed Rodriguez, due to the 1885 war which had increased misery by triplicating prices and increasing overcrowding due to the movement of people to the capital. These new elements, associated with dirtiness, explained why "we live here, day and night, indoors and outdoors, in the middle of a complete impure and pestilent atmosphere" and therefore why epidemics developed.[464] Those social and political circumstances would account for the "conditions in which the causal element of typhoid develops". Unfortunately, Rodriguez never explained what that causal element was. But in his view, studying these circumstances was equivalent to the aims of the Pasteurians: "for some valid reasons the leader of the microbial school insists so much and with such enthusiasm about the importance of getting to know causes in medicine".[465] For Rodriguez, the study of the conditions in which the agent developed – the social conditions - was practically equivalent to the study of causes.

Epilogue

After the first cycle of epidemics, between 1887 and 1889, a second notable increase in the number of typhoid cases occurred ten years later, in 1897.[466] Even by that time, the language regarding the causes of typhoid fever and typhus had not changed dramatically. Nor had the hygienic conditions of the city: people still urinated and threw their feces onto the streets, water for human consumption was not filtered, sewage systems were not sufficient, etc. Physicians and members of the JCH continued to explain the diffusion of the typhoid fever germ in the very same terms used at the beginning of that decade, that is, as emanations that infect air. For example, Pablo Garcia Medina explained that

[463] Rubén Rodríguez N., *Apuntes sobre la etiologia del tifo epidemico de 1889* (Bogotá: Imprenta de la Luz, 1890), p. 36.
[464] Ibidl, pp. 36, 40-41.
[465] Ibid., pp. 5-6.
[466] JCH, "Informe presentado al Sr. Mnistro de Gobierno por el Secretario de la Junta Central de Higiene, sobre los trabajos de esta Corporacion en los años de 1896 a 1898", *Revista de Higiene*, 56 y 57, noviembre y diciembre de 1899, pp. 612-620.

> …it is an undeniable fact that gases that escape from those holes [sewers] can cause infectious diseases. They carry microbes that produce those diseases which are then deposited in the soil from where they could be transported by air. So, we absorb the microbe of typhoid fever, for example, not only from the water we drink but also from the air we breathe.[467]

While, by the end of the century, physicians in other countries were realizing that the provision of clean water for human consumption and the building of aqueducts were the most effective measures to control typhoid epidemics, Colombian doctors working as hygienists insisted on avoiding the spread of typhoid germ or miasma via infected feces in the atmosphere by disinfecting feces in the hospital or by building sewers. Air continued to be stressed as the main means of infection until the end of the century. Proto Gomez, for example, pointed out that each individual attacked by typhoid or typhus was a focus of infection, given the huge amount of bacilli in the atmosphere surrounding him. He supported this argument by arguing that other diseases such as tuberculosis or anthrax were transmitted by air.[468] Thus, it seems that Eberth's bacillus did not alter the miasmatic formula, which was expressed in the following terms by Pablo García Medina in November 1899:

> If we left sewers open, the putrefying matter that remains stagnant there would infect the city; this infection would be permanent if, as is natural, drains from homes continue to bring to those streams the matter that through putrefaction produces deleterious miasmas.[469]

While during the 1880s Colombian doctors reconciled medical geography and germ theory with regards to periodic or intermittent fevers, they still used medical geographical arguments to support the non-climatic determinism of continuous fevers, namely typhoid fever. The germ identity appeared to be an irrelevant argument to the debates over the causes of typhus and typhoid fever. Despite the incorporation of antiseptic practices into the hospital and antiseptic recommendations

[467] Pablo Garcia Medina, "Higiene de Bogota", *Revista de Higiene*, No 56 y 57, noviembre y diciembre de 1899, p. 577.
[468] JCH, "Acta de la session del dia 12 de Julio de 1899", *Revista de Higiene*, No 63, octubre de 1900, pp.129-133.
[469] Garcia Medina, "Higiene de Bogota", p. 577.

for public hygiene purposes, doctors and hygienists were more interested in debating the clinical features of the fever in Bogotá and the possibility of it occurring alongside fevers typical of the warm lands. This latter problem dominated discussions about typhoid fever, partly because doctors in the highlands feared the ascent of the Magdalena fevers (the yellow fever form). Indeed, while Bogotá was affected by the typhoid epidemic in 1889, the physicians Borda and Osorio were working on a chemical vaccine against yellow fever with the argument that yellow fever was approaching the capital city (see Chapter 2).

Until the end of the century physicians persisted in considering typhoid fever to be transmitted mainly by air, and only advocated marginally for the building of proper aqueducts to prevent it; the specific agent, no matter what they called it - Eberth bacillus or miasma - would behave like a miasma.

In the following – and last – chapter, I will explore the case of leprosy. I will show how neo-Hippocratic and medical geographical arguments were used to explain the disease. But most importantly, I will also show how and why, contrary to causal considerations with regard to typhoid fever, Colombian doctors widely embraced the bacteriological hypothesis for leprosy during the 1890s.

CHAPTER 5

LEPROSY: CLIMATE, HEREDITY AND THE CONTROVERSY ABOUT SEROTHERAPY

Introduction

In previous chapters I analyzed how the neo-Hippocratic and medical geographical approach to periodic and continuous fevers was transformed by bacteriology. In this chapter I will explore this transformation in the case of leprosy. There are three justifications for the study of this case. Firstly, leprosy, alongside fevers, was the pathology that concerned physicians and physician-hygienists the most, for professional reasons. Indeed, while the first tide of physicians to return from Paris since the mid-century used the rhetoric of creating a national medicine based on the medical geography of the country beginning with the Magdalena fevers,[470] the doctors of the last decades of the century justified the creation of the national medicine by simultaneously exaggerating the statistics of leprosy and promoting bacteriology as the scientific approach to this disease.[471] Secondly, scholars have already pointed out that until 1880 leprosy had been framed within the neo-Hippocratic scheme of causes and that this scheme synthesized - to a certain extent - with the new bacteriological model before the latter became the dominant view;[472] however, this process and the place of medical geographical arguments in relation to leprosy can be further explored. Thirdly, the debate around serotherapy for leprosy, initiated by a Colombian doctor, stimulated a group of young doctors to initiate the first bacteriological analysis of diseases.[473] Thus, in this chapter I will detail the role of climatic and other geographical elements believed to be associated with the

[470] I have summarized this argument in chapter 1; for a more detailed description see Claudia Monica García, "Las fiebres del Magdalena: Medicina y Sociedad en la construcción de una noción médica colombiana, 1859-1886", *Historia, Ciências Saúde-Manguinhos* (Rio de Janeiro), 14, 1, en.-mar., 2007, pp. 13-30.
[471] Diana Obregón has developed this argument in her book *Batallas contra la lepra, Estado, Medicina y Ciencia en Colombia* (Medellín: Eafit, 2002).
[472] Obregón, *Batallas contra la lepra*, p. 168.
[473] A general description of the serotherapy trials developed in Colombia and the local and international debate it generated can be found in Obregón, *Batallas contra la lepra*, pp. 168-171.

development of leprosy by nineteenth century Colombian doctors (the first section of the chapter). I will also analyze the controversy of the end of the 1860s regarding the parasite that originated leprosy (the second section) and the beginning of the bacteriological turn in the causal approach to leprosy in the 1880s (the third section). I will also explore the metaphor of the seed and soil in the debates of the hereditary vs. contagion cause of leprosy (the fourth section) and the persistence of climatic and medical geographical elements in explaining leprosy up until the end of the century (the fifth section). This chapter finishes with a discussion of the first trials in Colombia to apply bacteriological techniques to clinical diagnosis (the sixth section).

Before I continue, it is important to clarify the use of the categories leprosy and elephantiasis by nineteenth-century Colombian doctors. Indeed, since the late eighteenth-century Colombian doctors used the term *elefancia* and lepra as synonymous of a putrid disease of spontaneous or venereal origin that affected the face and then extended to other parts of the body.[474] It was only around the end of the 1860s that they began to use the term "Greek elephantiasis" to refer to a chronic condition of sores on the skin and disfiguring nodules in the face or arms that were associated to sensory loss and loss of parts of the body.[475] Doctors of that decade wanted to differentiate it from "Arabic Elephantiasis", another skin deformity which one of them described in 1873 as mainly located on the low arms and associated with swelling involving the scrotum and surrounding parts.[476] The scarce medical literature about Arabic Elephantiasis in nineteenth century Colombian medicine when compared to Greek Elephantiasis (I found only one source devoted to Arabic elephantiasis),[477] and the lack of any controversy regarding both conditions[478] explains why I will only refer to Greek elephantiasis or leprosy in this chapter.

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[474] Obregón, *Batallas contra la lepra*, pp. 81-85.

[475] See for example José María Ruiz, *De la elephantiasis de los griegos*, Tesis para la revalidación del grado de José María Ruiz, doctor en medicina, presentada al Consejo de la Escuela de Medicina el dia 20, i sostenida el día 25 de febrero de 1867 (Bogotá: Imprenta de Gaitán, 1867), pp. 21-24.

[476] This short description appears in the discussion of the SMCN of a case of a woman with tubercles located in the her nose and face; doctors were aware that this location was unusual for "Arabic" elephantiasis. See SMCN, "Extracto de las Actas de la Sociedad de Medicina. Sesión del día 4 de octu bre", *Revista Médica*, Serie I, No. 11, 15 de febrero, 1874, p. 81.

[477] Lorenzo Eslava M., *Contribución al estudio de la elefancia de los arabes en Colombia* (Bogotá: Imprenta de la Luz, 1892

[478] Based on retrospective diagnosis, Eslava suggested in his 1892 thesis that José Félix Merizalde might have been referring to "Arabic" elephantiasis in his 1866 description of cases of *erysipelas* or *espundia*; and that Nicolas Osorio referred to *elefancia* –implying "Arabic" elephantiasis- in his 1873 lectures at the Faculty of Medicine in Bogotá. For Eslava, "Arabic" elephantiasis was a skin

Causes of leprosy by mid century

One of the first medical analyses of leprosy in Colombia was authored by José Félix Merizalde in 1835.[479] I have already shown how Merizalde incorporated the neo-Hippocratic scheme of specific, predisposing and occasional causes to specify the type of causal action of the internal and environmental phenomena associated with diseases. Interestingly, Merizalde and some of the doctors of the first half of the century did not apply this hierarchy of causes in relation to leprosy. Merizalde refers to "general" causes that may produce leprosy and prioritized one on the basis of its frequency: sudden transitions from heat to cold. The other causes were pretty much the Hippocratic non-naturals: eating rotten food, frequent consumption of fish (especially if it came from swampy or quite rivers), drinking foul water, living near swampy places, filth in the body and clothes, alcohol abuse, the suppression of natural evacuations and finally, passions.[480]

As I will show, the most common cause of leprosy according to Merizalde, sudden changes of temperature, would somehow persist in physicians' explanations of leprosy until the end of the century. For the moment, let us say that for Merizalde, this cause was an "alteration of the atmosphere" that was very frequent in the Torrid Zone.[481] Although sudden changes of temperature were simply the most common cause – not the specific one - for leprosy, it is telling of the pre-eminence of environmental factors in explanations for diseases in the first half of the nineteenth century.

Apart from the "general causes" mentioned above, Merizalde also considered that contagion "coming from inheritance, breastfeeding and cohabitation" was causally

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hypertrophy of the lower limbs or genitalia caused by inflammation of the lymphatic and venous systems. In his view the worms of the group *filariae* whose causal association to "Arabic" elephantiasis had been definitely established by Patrick Manson in 1878-1879, were one among the other several causes of that hypertrophy. Eslava gathered descriptions of several doctors from Boyaca, Santander, Tolima and Cauca who climed to have seen cases of "Arabic" elephantiasis though all them agreed that the disaese was rare. See Eslava, *Contribución al estudio de la elefancia de los arabes*, pp. 14-20.

[479] Merizalde's work on leprosy is the *Memoria que la Facultad Médica presenta sobre los lazaretos que manda la Ley de 1834* which was copied almost entirely by J.B. Montoya y Flórez in *Contribución al estudio de la lepra en Colombia* (Medellín: Imprenta Editorial, 1910), pp. 48-9.

[480] Ibid., pp. 48-9.

[481] Ibidem.

associated with leprosy.[482] Merizalde recognized that "modern" authors considered the disease either contagious (under specific circumstances) or not contagious at all. For him, priority could not be given to either of these two opposed doctrines, for both were founded on sound though not constant facts.[483]

By around mid-century, a similar set of causal notions are found in the work of the medical-geographical oriented physician Antonio Vargas Reyes. Around the late 1850s, the Government of the State of Cundinamarca asked him his opinion on the contagion of leprosy and on how to avoid it. Vargas Reyes mentioned several causes of leprosy such as eating pork, venereal contagion, living in humid and ill ventilated places and, finally, a specific contagion. But contagion, he said, had not been demonstrated: it was not known how it was transmitted, it had not been inoculated, and there were multiple examples of people who, despite living with the sick, never contracted leprosy. As such, Vargas Reyes supported the idea that leprosy was a hereditary disease brought to America by Spaniards, which tended to affect Caucasians and Ethiopians and *Mestizos*, rarely natives.[484]

An example of the multiple causal approach with regards to leprosy, that dates from before any bacteriological theory was ever considered, can be found in José María Ruiz's medical thesis on *Greek elephantiasis* of 1867.[485] Ruiz analyzed causes of leprosy following the neo-Hippocratic hierarchy of causes: determinant or specific, predisposing and proximate causes.[486] Contagion, heredity and climate played an important role in this scheme; indeed, the specific cause was contagion. The evidence he provided was historical: the bible, he said, suggested that people should not touch lepers in order to avoid falling sick. Among the predisposing causes mentioned were heredity, climate and soil. Ruiz repeated Merizalde's idea that sudden changes of temperature could cause leprosy, and also the idea that it was therefore common in the intertropical areas of hot climate. Marshy and humid regions were also predisposing causes of leprosy. The speed with which leprosy

[482] Ibid., p. 48.
[483] Ibid., p. 51.
[484] Antonio Vargas Reyes, "Informe al secretario de gobierno sobre la elefanteasis", *Trabajos científicos del Doctor Antonio Vargas Reyes recopilados en obsequio de la humanidad doliente i de la juventud estudiosa de Colombia*, Tomo Segundo (Bogotá: Imprenta de la Nación, 1862), pp. 57-58, 60.
[485] Ruiz must have requested the official examinations in order to achieve the degree one year after the opening of the private school.
[486] Ruiz, *De la elefantiaisis de los griegos.*

developed was also affected by climate: clinical stages developed slowly in cold climates, according to Ruiz, especially in Colombia where diversity of climate was the rule. Ruiz also cited strong emotions in someone predisposed by heredity as one of the occasional causes.[487]

This neo-Hippocratism as applied to leprosy would give way to a full medical geographical view, even though it would never become the standard view. But before I explore that development, I shall discuss the first controversy regarding the germ origin of leprosy developed in the late 1860s.

Early considerations on the *parasitic* cause of leprosy

As Obregón has pointed out, one of Colombia's earliest supporters of the idea that microscopic living entities could cause leprosy (and also tuberculosis and syphilis) was the homeopath Ignacio Pereira. In 1866 he explained that leprosy was caused by mites in the tissue of patients with *Elefancia*.[488] Obregón argues that Pereira's work cannot be considered a precursor of the germ theory since he was referring to *parasites* –not germs or microbes- that had been identified in micrographic studies; in other words, because he was not referring to a bacteriological theory. However, a few years after the 1866 paper, Pereira claimed his work to be the precursor of the germ theory in the "bacteriological" sense. Indeed, in his 1870 paper "Parasitic Diseases" he reinterpreted his 1866 ideas in that direction after reading one of Tyndall's articles, translated in the *Revista Científica e Industrial* of Bogotá as "Polvos y Enfermedades" (Dust and Diseases). Tyndall explained the presence of organic particles in the air and summarized Pasteur's work on ferments, the "epidemic germ theory", and Listerism. Tyndall declared that some of the organic particles found in the air were germs that could penetrate organisms, causing epidemics. He also described hygienic measures to halt those organisms, mainly the use of cotton.[489] Upon reading this translation, Pereira declared that Tyndall's (and

[487] Ibid., pp. 10, 12-14.
[488] Obregón, *Batallas contra la lepra*, p. 99.
[489] J. Tyndall, "Polvos y enfermedades", *Revista Científica e Industrial*, No. 4, 20 de agosto, 1870, pp. 49-51.

Pasteur's) work were the material demonstration of what he had found "by induction" several years before, and that he was therefore the founder of the doctrine of "parasitic" diseases.[490] In support of this he claimed to have recommended similar hygienic measures, before Tyndall's paper, in a paper called *La Infeccion*. In that paper, he said, he had demonstrated that miasmas that produce dominant diseases were composed of organized beings.[491]

Pereira was not very successful in his claim of having discovered "by induction" the parasitic theory of diseases. He sent his work to the board of the medical school in Bogotá but they did not release any response to Pereira. Pereira's parasitic theory did not prosper among the medical community, partly because homeopaths were unsuccessful in their efforts to reach the same level of institutionalization that mainstream medicine had been forging since the mid-century. Indeed, while physicians managed to transform the private medical school of Bogotá into the medical faculty of the National University of Colombia created by the Colombian State in 1868, homeopaths' proposal to become part of the new university was rejected, as was the proposal to create a place for homeopathic practice in the university hospital.[492] The failure of the homeopath Ignacio Pereira to lead physicians' attention to a "parasitic" theory of leprosy cannot be separated from the failure of homeopaths to enter the mainstream of medical knowledge.

Doctors continued to use the multiple neo-Hippocratic scheme of causes to explain causes of leprosy; the emphasis on what was considered the most important cause depended on personal opinions and experience. Some considered the main cause to be sudden changes in temperature; others heredity; others contagion; and others a mixture of several of them. Although medical geography never became the major approach to leprosy, it is relevant to mention here how it nevertheless persisted in physicians' minds.

Medical geography and leprosy

[490] Ignacio Pereira, "Enfermedades parasitarias", *Diario de Cundinamarca*, 263, 29 septiembre, 1870, p. 1052.
[491] Ibidem.
[492] Maria del Pilar Guzmán, "La alopatía y la homeopatía en el siglo XIX: conflicto entre dos prácticas", *Anuario Colombiano de Historia Social y de la Cultura*, 22, 1995, p. 73.

According to Obregón, Samuel Duran's thesis *Elefantiasis de los Griegos* (1874) is one of the last examples of the persistence of the neo-Hippocratic conceptions of leprosy in nineteenth-century Colombia.[493] Indeed, Durán's thesis focuses on the "cosmic" causes of leprosy, meaning air, water, winds, humidity, temperature, topographic situation of countries, geological constitution of land and altitude; Durán considered these as the predisposing or the true causes of the malady.[494] Obregón reminds us that Duran insisted that a study of the local conditions under which leprosy developed was needed. Durán certainly echoed the argument developed since the last colonial years that "atmospheric conditions, altitude and geological constitution all influence the moral and physical development of people and predispose them to get diseases".[495] But I think it can be further emphasised that Durán was also one of the few that applied the core medical geographical argument to leprosy, that is, the idea of a functional relationship between geographical features and disease: "the frequency of this disease [Greek elephantiasis]" Durán said, "is always inversely proportional to high temperatures and pressure".[496] Indeed, according to Durán, high altitudes and mild atmospheric pressure would weaken all organs' functions, including the suppression of the activity of the skin. In consequence of this latter action, the nervous system would be strongly affected thus transforming the vital force of the body into a kind of "deleterious principle" that ought to escape but could not. This is why leprosy prevailed in the Torrid Zone, especially in the plains of the mountains. Durán recognized, however, that bad food could also spoil blood and produce leprosy.[497]

Besides the functional association between altitude and leprosy, another medical geographical argument in Durán's thesis is the association between the geological constitution of the soil and the disease. For example, he argued that despite the physical similarities between Zipaquira and Chia villages - both located in the high plateau of the eastern *cordillera* of the Andes - in the former, two thirds of the

[493] Obregón, *Batallas contra la lepra*, p. 104.
[494] Samuel Durán, "Elefantiasis de los Griegos" (Tesis para el doctorado en medicina y cirugía), *Anales de la Universidad Nacional de los Estados Unidos de Colombia*, 8, 67-72, 1874, pp. 466-8.
[495] Ibid., p. 468.
[496] Ibid., p. 469.
[497] Ibid., pp. 468-470.

population were affected by leprosy, whereas in the latter there was none. The reason was the salty soil of Zipaquira that cooled air thus triggering leprosy; Chia had a different type of soil. [498]

Another medical geographical argument in Durán's thesis is his anticontagionism. Based on his observations made at the *Agua de Dios* lazaretto, where he witnessed that healthy people who lived in close contact with lepers were never attacked, he argued that "cosmic" and hereditary causes far outweighed contagion as the cause of leprosy.[499]

As I suggested, the medical geographical approach to leprosy was marginal among the Colombian medical community. However, it is also true that by the time the germ theory of leprosy was reintroduced in the early 1880s, doctors still believed that climate and geography were closely associated to leprosy. I mentioned that Hippocratic-oriented doctors considered sudden changes of temperature to be one of the causes of leprosy from early that century and this idea continued to be present in one way or another throughout the century: one of the firm supporters of the germ cause of leprosy of the 1880s, Gabriel J. Castañeda, believed that doctors had underestimated the role heat and cold on the disease. In 1882 he affirmed that heat would open the skin's pores thus allowing the causal agents of leprosy to enter the body; then there was cold that would be the gatekeeper that closed those pores allowing the disease to develop. This role of heat and cold had, in Castañeda's view, however, a mechanical rather than a causal role in the pathogenesis of leprosy.[500]

The bacteriological turn

Gabriel J. Castañeda (1846-1900) graduated from the Bogotá medical school in 1867 and it seems that he never went to Paris.[501] I showed in Chapter 2 that he was one of

[498] Ibid., p. 471.
[499] Ibidem.
[500] Gabriel J. Castañeda, *Causa y tratamiento racional de la lepra de los griegos hallados por inducción*, (Bogotá: Imprenta de Echeverría Hermanos, 1882), pp. 39-40. The fact that pores of the skin of the face were wider that those of the rest of the body, would explain why leprosy tend to begins with lesion in the face.
[501] Cáceres, Humberto y Cuéllar-Montoya, Zoilo. *Academia Nacional de Medicina de Colombia. Sus Miembros* (Bogotá: Academia Nacional de Medicina, 1998), pp. 21-22.

the more enthusiastic supporters of a bacteriological cause for yellow fever during the 1887 episode of prophylactic inoculations. His interest in bacteriology had begun earlier, however. He was the first who praised Pasteur's work before the SMCN in 1882. In the same year he published a summary of the classification and characteristics of germs following Pierre Houbert Nysten's description of the "animal and vegetable infusoria".[502] There appeared the *microzoarios* and *micrófitos*, that is, small animals and vegetables respectively. *Micrococus, Bacterium, Vibrio Lineola, Bacillus, Spirillum, and Sarcina* were the general genera of the *micrófitos* some of them already associated to diseases such as Bacterium to putrefaction.[503]

In 1882 Castañeda also published a short work on the causes and treatment of leprosy based in the Pasteurian germ-theory, but interestingly, he started his work with puerperal fever. He claimed to have been led by "rigorous induction" from puerperal fever to the common ("parasitic") cause for several diseases, including leprosy, and to a "rational" treatment for this malady.[504] The induction process began with the description of a case of puerperal fever successfully treated with antiseptic substances, followed by two other cases (gangrene and abscess) also successfully treated with antiseptic medications. These clinical experiences added to the association that the international community had established between Hansen's bacillus and leprosy. The Norwegian Gerhard Armauer Hansen (1841-1912) described the *Mycobacterium leprae*, which was accepted as the causative agent of leprosy, between 1870-1874. Opposing the prevalent hereditary, miasmatic dietetic and hygienic theories about the causation of leprosy, Hansen believed that the disease was contagious. Thanks to his work, leprosy is one of the first illnesses that was causally connected with a microorganism.[505] News of Hansen's *Micobacteria* and the disputes over the priority of the discovery with the German Albert Neisser reached the *Revista Médica* in Bogotá by early 1882. The news said that "it is widely recognized that the severity of the leprosy lesions have a direct relationship with the

[502] It must have been one of the multiple editions of Nysten's *Dictionnaire de médecine, de chirurgie, de pharmacie, de l'art vétérinaire*. Unfortunately, I could not find the edition of Nysten dictionary on which Castañeda based his article.
[503] Gabriel J. Castañeda, "Descripción sucinta de los microzoarios", *Revista Médica*, Serie VII, No. 73, Junio 20 de 1882: 39-43.
[504] Gabriel J. Castañeda, *Causa y tratamiento racional de la lepra de los griegos*, pp. 7-8.
[505] Obregón, Diana. "The Social Construction of Leprosy in Colombia, 1884-1939". *Science, Technology and Society*, 1, 1 (1996), pp. 5, 19.

amount of parasites [Hansen's microorganisms]" but also that the cultivation of Hansen's bacilli has not produced good results.[506] Armed with Hansen's discovery and the cases of puerperal fever, gangrene and abcess successfully treated with antiseptics, Castañeda felt compelled to propose a "rational" cause and treatment for leprosy. Let us examine Castañeda's inductive process in detail.

Castañeda detailed the case of a woman who within a few days of giving birth suffered hemorrhages, fever, increased pulse, and weakness. She was diagnosed with puerperal fever caused by putrefaction of remnants of placenta in the uterus. Three uterine injections per day with carbolic acid were sufficient for the woman to survive.[507] Castañeda detailed nineteenth-century theories of puerperal fever (inflammation of organs related to birth, absorption of septic matters, miasmas entering via respiratory channels or an essential fever) in order to defend the current tendency to consider the absorption of "bacteria" as the cause of puerperal fever. Castañeda followed this description by turning to the medical thesis of a Colombian national, Ignacio Gutierrez Ponce, who studied in New York and published his thesis in 1874. In that thesis Gutierrez mentioned that French doctors had made the analogy between women in puerperium and wounded people. This analogy allowed them to propose that the uterovaginal mucous membrane was the gateway of the "morbid material".[508]

Castañeda not only agreed with this description but was also sure that the agent or "parasite" for puerperal fever had been already demonstrated. Castañeda provided excerpts from French authors such as Duclaux and Pasteur to support this idea, in particular Pasteur's discourse of July 15, 1880 about the extension of the germ theory to puerperal fever.[509] Pasteur had been allowed to make microscopic analysis and cultures of remnants of placenta, blood and liquid from the peritoneum of a woman who died of puerperal fever. He concluded that pus developed in the bloody parts of the uterus after birth and that it could be associated with organisms that came from

[506] RM. "Lepra", [Extracto Revista Extranjera], *Revista Médica*, Serie VI, No. 70, 20 de febrero, 1882: 461.
[507] Castañeda, *Causa y tratamiento racional de la lepra de los griegos*, pp. 11-12.
[508] Ibid., pp. 16-17.
[509] Ibid., pp. 17-21.

outside the body. These organisms, Pasteur stated, must have passed to the peritoneum via the Fallopian tubes and to the blood via lymphatic vessels.[510]

Castañeda affirmed that if "Pasteur tells the truth", the treatment of puerperal fever had to involve the immediate disinfection of septic foci and of the objects surrounding the sick, good ventilation, the administration of both a medicine for killing parasites and an agent that stimulates organisms in order to destroy them, and, finally, good nourishment.[511] The idea of using antiseptic measures to prevent and treat puerperal fever was not entirely new to the Colombian medical community. Two years before Castañeda's claims, Abraham Aparicio published in the *Revista Médica* a note about puerperal fever in which he stated that it was known that puerperal fever was due to septic matter coming from outside the body, and that antiseptic measures were recommended in order to prevent the infection caused by the hand of the surgeon and also to destroy the products of decomposition that would develop in the uterus.[512] In 1882 the editors of the *Revista Médica* declared that "if we admit the microbe to be the cause of puerperal septicemia, as it has been for surgical septicemia, it is obvious that we should use the antiseptic method in the former".[513] Thus, Castañeda recommended the disinfection of the septic foci in puerperal fever with carbolic acid, as in surgery, and to kill the parasite he recommended the "king of the therapeutic", quinine sulfate. He defended the use of quinine as antiseptic for several years probably because quinine had been produced in Colombia since the colonial years and doctors had shown wide interest on its production and therapeutic use on intermittent fevers of any kind.[514] Actually, by the time Castañeda wrote his document, quinine was in the last year of its last nineteenth-century export boom.[515]

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[510] Ibid., p. 26.
[511] Ibid., pp. 26-27.
[512] Abraham Aparicio, "Revista Científica", *Revista Médica*, Serie IV, No. 43, 25 de enero, 1878, pp. 353-356.
[513] Auvard, "Ensayo de sustancias antisépticas durante el embarazo, el parto y sus consecuencias", [Extracto Revista Extranjera], *Revista Médica*, Serie VI, No. 70, 20 de febrero, 1882, pp. 471-474.
[514] Castañeda, *Causa y tratamiento racional de la lepra*, pp. 27-28. Even by 1893 Castañeda still insisted in the killing power of quinine. See Gabriel J. Castañeda, "Estudio sobre la lepra. Conclusión", *Revista Médica*, 129, 28 de septiembre, 1888, pp. 179-181; Ibid, "Patologia. Estudio sobre la lepra en Colombia", *Anales de la Academia Nacional de Medicina*, Tomo I (Bogotá: Imprenta de la Luz, 1893), p. 179.
[515] Yesid Sandoval and Camilo Echandia, "La historia de la quina desde una perspective regional", *Anuario Colombiano de Historia Social y de la Cultura, 1850-1882*", 1986, 13-14, pp. 153-187.

Once Castañeda had made his argument in favor of a parasitic cause of puerperal fever and described his antiseptic treatment by analogy with antiseptic surgery, he then moved to a general germ theory of diseases. Again he began with clinical cases upon which he made reflections on the "community of cause" of several diseases. First, one of his relatives suffered gangrene in one leg after having typhoid fever and the leg had to be amputated with the antiseptic method by his colleague Nicolás Osorio. It is not clear when this operation happened, but, according to Castañeda, this was the first time that "the Listerian method" was used in Bogotá. They continued the treatment with quinine. The second case was Castañeda himself. He had an abscess in the liver, having suffered from "telluric" dysentery. Physician Rafael Rocha Castilla explored the abscess without success but Castañeda's life was saved because of carbolic acid injections. These two cases and the case of puerperal fever successfully treated with carbolic acid were "all caused by parasites and all were cured by the same treatment", concluded Castañeda; thus, he claimed, he was led "by induction" to apply all of these principles to leprosy.[516]

He knew that the "parasitic" theory was already established for many diseases including leprosy (I mentioned above that he was aware of the work of Gerhard A. Hansen on the leprosy microorganism and of A.V. Cornil, the French author of an early textbook on bacteriology). As Castañeda's main goal was to find a treatment for leprosy according to the new parasitic theory, he advocated the same treatment he had recommended for puerperal fever, that is, to kill the bacteria with quinine but also use steam baths with carbolic acid. Those vapors, he believed, would open the pores, allowing the parasites of leprosy to leave the body and thus reverse the process by which they had entered, that is, whe the pores were opened by the heat of the environment.[517]

Castañeda applied the anti-parasitic treatment described above to a woman diagnosed with leprosy in Bogotá in 1882.[518] He gave her quinine to kill the germ, vapors of carbolic acid, and good nourishment and wine, and he reported temporary good results for the cutaneous lesions. However, after eight months there were no

[516] Castañeda, *Causa y tratamiento racional de la lepra*, pp. 29-31.
[517] Ibid., pp. 31-32, 38-40, 42-43.
[518] Gabriel J. Castañeda, "Tratamiento parasiticida de la lepra", *Revista Médica*, Serie VIII, No. 96, 20 de junio, 1884, p. 513.

further changes. Castaneda was concerned that the failure of antiseptic treatment, which aims to kill the putative germs of leprosy, will lead people to doubt that leprosy was actually caused by microbes. This worry is particularly acute since Castaneda (and Hansen) had been unable to cultivate the putative germs, or to demonstrate their causal role by inoculating the disease into experimental animals. Consequently, Castañeda turned to Koch, who had argued that his inability to cultivate and inoculate the cholera germ does not disprove the microbial origin of that disease, and who explicitly extended this argument to other diseases such as leprosy and typhus. Following this argumentation, Castañeda affirmed that despite leprosy bacilli had not been cultivated or inoculated, it can however be consider as the specific cause of leprosy by "its way of action", meaning by the disease itself.[519]

Aditionally, he was subsequently able to reinforce this argument using histological preparations of the Hansen bacilli. In 1886, the Colombian national Emilio Alvares was working in Victor Cornil's laboratory in Paris[520] and sent 19 preparations of the bacteria of anthrax, typhoid fever, pneumococci, tuberculosis, and leprosy, among others, to the SMCN in Bogotá.[521] Castañeda was commissioned to study them. In his report he centred on the samples with the bacillus of leprosy. Castañeda summarized Hansen discovery, the confirmation of Hansen's work by other scientists, and the unsuccessful trials for cultivating it. Castañeda also summarized staining methods used by A. V. Cornil and V. Babes and the histological characteristics of Hansen bacilli.[522] As I said, despite Castañeda's antiseptic treatment failed, and despite the fact that Castañeda was aware that inoculations to reproduce the disease had been unsuccessful as well as its cultivation, he insisted that the "histological" demonstration of the bacilli –his own experience of them through

[519] Ibid., pp. 513-520.
[520] We don't know how long did he stay there or if he returned to Colombia aferwards. We know that he published three papers on the microorganisms of rinoscleroma and syphilis in the *Archives de Physiologie Normale et Pathologique*. See Cornil and Alvarez, "Memoire pour servir a l'histoire du rhinosclérome", *Archives de Physiologie Normale et Pathologique*, 17, 5, 30 Jouin, 1885, pp. 11-40 and Alvarez, "Reserches sur l'anatomie pathologique du rhinosclerome", *Archives de Physiologie Normale et Pathologique*, 18, 2, 15 Janvier, 1886, pp. 196-207 ; Alvarez and Tavel, "Reserches sur le bacille du Lustgarten", 17, 7, 30 Septembre, 1885, pp. 303-321.
[521] Gabriel J. Castañeda, "Informe relativo á la colección de preparaciones microscópicas enviadas de París por el señor doctor Emilio Álvarez", *Revista Médica*, Serie XI, No. 118, 20 de septiembre, 1887, p. 811 and SMCN, "Acta de la sesión ordinaria del 20 de septiembre de 1888", *Revista Médica*, 130, 28 de Octubre, 1888, p. 266.
[522] Castañeda, "Informe relativo á la colección de preparaciones microscópicas", pp. 807-8113.

Alvarez's preparations and as well as Cornil's and Babes's descriptions-, would account for the definite role of this bacilli in the illness.

Historian Diana Obregón has argued that Castañeda's version of leprosy illustrates the syncretism between the neo-Hippocratic theories about leprosy and the new germ theory.[523] This is the case, she argues, because Castañeda simultaneously defended the "parasitic" origin of leprosy and the role of sudden changes of temperature in the pathogenesis of the disease (heat allows the germ to enter and escape through the pores). But as I said, in Castañeda's 1882 paper the role of these changes was less causal and more mechanical.

The role of geography and climate in causing leprosy

In the later 1880s, the germ theory of leprosy acquired a new significance in the context of Colombian doctors' efforts to claim leprosy from philanthropists. Lazarettos were in the hands of charity organization since colonial times but in the late 1880s and the 1890s Colombian doctors exaggerated the amount of cases of leprosy as a strategy to medicalize it, as historian Diana Obregón has already detailed. Obregón argues that the medical profession sought to convince society of its exclusive possession of the scientific competence to treat leprosy. As such, they set out to demonstrate that philanthropy had been unable to manage the problem and thus the number of lepers had increased instead of diminished. In other words, at the end of the nineteenth century, when medicine became a profession in Colombia, leprosy was one of the means doctors used to assert their scientific authority and increase their power by medicalising what had previously been seen chiefly as the domain of philanthropic care.[524] It seems that Gabriel J. Castañeda, who since 1882 insisted in the antiseptic treatment for leprosy, sounded the alarm with regards to the increase of the amount of lepers in Colombia. In September 1888, he announced to the SMCN that he had begun informal enquiries with friends living in other cities about the amount of lepers in the country. This announcement was made one year after he reported about Emilio Alvarez's samples of the Hansen bacilli sent from

[523] Obregón, *Batallas contra la lepra*, p. 168.
[524] Obregón, *Batallas contra la lepra*, pp. 182-184; 201.

Paris as I explained in the last section. Interestingly, Castañeda had proposed in his report about Alvarez's samples the creation of a special commission within the SMCN devoted to microscopic studies. That commission was actually created in August 1887 composed by Gabriel Duran Borda, Nicolas Osorio, and Castañeda himself but it seems to have never functioned.[525] But consistent with this renewed interest on leprosy Castañeda thus began in 1888 his individual enquiries about the distribution of leprosy in the country; he considered plausible, for example, José María Buendías's opinion that 6% of the 500,000 population of Santander could be affected, that is, 30,000 people. This, Castañeda said, was a terrifying figure for a region and asked the SMCN to point out the danger of leprosy to the government.[526] Accordingly, in October of that year the JCH approved a questionnaire about the extent and causes of leprosy to be sent to physicians around the country as well as to the regional medical societies of Medellín and Cauca that had been recently created in 1887 and 1888 respectively. The questions concerned, among other topics, the number of individuals affected by leprosy, the causes that the population and physicians judged to be related to leprosy; whether any food could be considered as a predisposing, determinant or occasional cause; whether there were any climatic condition, real or believed, that had been pointed out as favorable for the development of leprosy; whether there were facts that proved contagion or heredity; and what other diseases occurred there.[527] Answers began to arrive in May 1889 and the summary of the results was presented by Castañeda in June 1890. Although he complained that the questionnaire was not answered by physicians in all cases, he did not find any problem in using some of the answers as evidence of his own arguments.

122 out of 920 municipalities sent their answers; 29 believed that leprosy was caused by heredity and 31 by contagion. Despite the minimal difference between the

[525] Castañeda, "Informe relativo á la colección de preparaciones", p. 812. In the SMCN session of August 27, 1887 the SMCN appointed Castañeda, Duran and Osorio as permanent members of the microbiological commission but I have not found evidence of their work as a group. See SMCN, "Acta. (Sesión del sábado 27 de agosto de 1887), *Revista Médica*, Serie XI, No. 119, Octubre 20 de 1887, p. 833 and Pedro María Ibáñez, "Informe leído por el doctor don Pedro María Ibáñez, secretario de la Sociedad de Medicina y Ciencias Naturales de Bogotá, en la sesión solemne que tuvo lugar el 25 de febrero de 1888 (Continuación)", *Revista Médica*, Serie XII, No. 126, 28 de junio, 1888: 78.
[526] SMCN, "Acta de la sesión ordinaria del 20 de septiembre de 1888", *Revista Médica*, 130, 28 de Octubre, 1888, pp. 262-264.
[527] JCH, "Sesión del día 1º de octubre de 1888", *Revista de Higiene*, No. 9, 25 Noviembre, 1888, pp. 207-8.

numbers, Castañeda took the latter figure as a proof of the "parasitic" theory of diseases, instead of taking the former as a proof of leprosy being hereditary. In 20 towns people considered that food was either a predominant, determinant or occasional cause of leprosy. In Castañeda's view, food only prepared the "soil" for the "seed" to get in the body, or was the vehicle of germs.[528]

30 towns considered sudden transitions of temperature to be the cause of leprosy and 40 referred to the bad influence of temperate and humid climates. From the answers Castañeda concluded that in dry climates with temperatures above 25 degrees Celsius, leprosy did not develop. The reason, he said, was that parasites expelled from the bodies of lepers in those climates were either already dead when they came out or died when exposed to the environment. Castañeda thus recognized that high temperatures might have a deleterious effect on the microbe of leprosy.[529] Conversely, he declared, humid places of mild temperatures "are favorable for the swarming of microscopic fungi and therefore figures indicate that in those climates leprosy spreads quickly".[530]

These conclusions meant that Castañeda became more open than before to a causal role of climate in leprosy and led him towards a medical geographical argument: from the mechanical role he imputed in 1882 to sudden changes of high temperatures opening the way for microbes, the survey led him to accept that temperature was a key factor in determining the geographical distribution of leprosy. In the first Colombian medical congress in 1893 Castañeda reaffirmed this idea. In his presentation he declared that "climatic conditions play an important role as determinant common causes of leprosy". By "common determinant cause" he meant the fact that climate could alter the functions of the nervous system, weaken the nutrition system and disrupt the organic resistances of tissues, all of which created adequate conditions for the leprosy germs to penetrate the body or to develop if they rested in a latent state. He went on to argue that "since Antiquity authors have unanimously agreed that sudden changes of temperature are harmful for health", an idea that early nineteenth-century Colombian doctors such as José Félix Merizalde

[528] Castañeda, "Investigaciones sobre la lepra en la República de Colombia", pp. 645-6.
[529] Ibid., p. 646.
[530] Ibid., p. 649.

had repeated, specially those changes were the most common cause of leprosy. Contagion was, for Castañeda, the efficient cause of leprosy.[531]

Although the survey did not provide overwhelming quantitative support for any one cause, even by the rather selective reasoning employed by Castañeda, he concluded from those results that the JCH should propose a law for the compulsory isolation of the lepers.[532] Castañeda's arguments in favour of a contagious, microbial aetiology of leprosy provided strong support for such policies. Obregón has extensively shown how Aparicio and other physicians exaggerated the amount of cases of leprosy in Colombia so that the disease began to be considered one of the barriers to a political and cultural project of the Colombian elites who wanted to enter the "civilization".[533] This concern supported on the idea that leprosy was caused by a specific microorganism fostered policies of compulsory isolation of lepers who were by then confined in three Lazarettos: *Agua de Dios* in Cundinamarca, *Caño del Loro* in Bolivar and *Contratación* in Santander. Doctors from the JCH wanted them to be transported to an island on the Pacific coast but this never became a reality.

Castaneda's campaign for the microbial cause of leprosy, though important, did not lead to universal acceptance that leprosy was caused by a microbe. Not only he turned to climatological and medical geographical arguments after the national survey he promoted, as I showed, but also doctors around the country clearly continued to frame leprosy referring to bacteriological and climatological notions simultaneously. Evaristo Garcia and Adolfo Tenorio from Cali, Cauca, recognized the bacteriological and contagious character of leprosy but at the same time claimed to have noticed variations of temperature as the immediate – probably meaning occasional - cause of leprosy (for example cold baths after exercise or jobs that exposed people to high temperatures). They also considered that leprosy developed in temperate and humid climates, though they recognized that there was no scientific evidence that supported a "climatic-telluric" cause. Although leprosy could be found

[531] Gabriel J. Castañeda, "Patología. Estudio sobre la lepra en Colombia", *Anales de la Academia Nacional de Medicina*, Tomo I (Bogotá: Imprenta de la Luz, 1893), pp. 156-158.
[532] Castañeda, "Investigaciones sobre la lepra en la República de Colombia", p. 659.
[533] Obregón, *Batallas contra la lepra*, pp. 180-191.

in all of our climates, they said, it was uncommon in warm and dry valleys.[534] J.B. Londoño from Medellín, Antioquia, on the other hand, also stated that leprosy was common in climates of low temperature. For him, climate prepared the soil for the development of leprosy: organisms were more prone to nervous perturbations under certain atmospheric and telluric conditions, and he wondered if leprosy was an essentially nervous disease, even though he also recognized that it was generally accepted to be a "parasitic" disease.[535] More radical was the opinion of José Narciso Garay who asserted that the expansion of leprosy was due to climatic influence, heredity, and relaxation of habits; that it was not contagious; that it dominated in places in which the topography exposed people to sudden changes of temperature.[536] One professor at the medical school was teaching, by 1888, that leprosy was not a contagious disease but was caused by sudden changes of temperature and heredity, and that it occurred in humid, rough and irregular places, exposed to the noxious influences of winds and therefore to sudden changes of temperature.[537]

During the 1893 medical congress. when around 30% of papers presented dealt with leprosy (around 40% were on Magdalena fevers and malaria together), opinions on climatic considerations of leprosy were also prevalent. Manuel Uribe Angel claimed that leprosy was a specific, microbial, contagious and chronic disease; that like the other specific affections it had only one efficient cause and two or more coadjutant causes which provoked its development such as sudden changes of temperature or strong emotions.[538]

One of the participants to that congress who had a long experience in an endemic area of leprosy wanted to call the attention of the attendants to the congress on the medical geographical elements associated with leprosy. Heráclito Gómez had worked thirteen years in Socorro and Velez villages in Santander, places with high rates of leprosy. He treated sick people, paying careful attention to the predisposing and determinant causes of the disease. In more than two thirds of the cases he found

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[534] Evaristo García y Adolfo Tenorio, "Lepra", *Boletín de Medicina del Cauca*, 29, Julio, 1889, pp. 812-813.
[535] J.B. Londoño, "Apuntamientos sobre la propagación de la elefantiasis griega en Antioquia", *Anales de la Academia de Medicina de Medellín*, 4, enero, 1891, pp. 112-125.
[536] José Narciso Garay, "La lepra de Moisés", *Revista de Higiene*, 34, 10 de abril, 1892, pp. 402-403.
[537] Manuel S. Algandona, "Contribución al estudio de la higiene. Historia suscinta de la lepra en Paipa", *Revista Médica*, 165-166, sept-oct., 1891, p. 626.
[538] Manuel Uribe Angel, "Profilaxia de la lepra", *Anales de la Academia Nacional de Medicina*, Tomo I (Bogotá: Imprenta de la Luz, 1893), pp. 71-72.

hereditary antecedents. When he could not find this antecedent, he attributed leprosy to contagion if the patient had lived with lepers before the onset of the disease. When he could find neither hereditary antecedents nor contagion, he would look for other causes in geography. Indeed, in his practice he noticed that localities where leprosy prevailed were very similar in their climate and exposition to winds. Gómez illustrated this point with two cases. First, in the valley of the River Suarez there were Puente Nacional, Guavatá and Cite, municipalities of temperate climate cooled by southern and eastern winds. These municipalities had a high rate of cases of leprosy. Nearby regions, on the contrary, had different climatic conditions that were more apt for life and therefore had few cases of leprosy, despite the commercial and social relations they had with the former municipalities. Second, in the Guadalupe municipality, continued Gómez, the high rate of leprosy cases was due to sudden changes of temperature owing to the geographical constitution of the region. Indeed, Guadalupe was formed by a high plateau surrounded by two river valleys where people would go down to cultivate rice. These valleys were unhealthy and dominated by paludic fevers. In the morning, peasants that lived in the high plateau would go down to the river valleys and work the whole day amid that unhealthy atmosphere. In the afternoon they would climb to their homes in their wet clothes. Once at the top they would be exposed to the frozen winds that came down from the high eastern mountains. This exposure to high and low temperatures in a short period of time, according to Gómez, accounted for the high rate of leprosy in the Guadalupe municipality.[539]

Gómez concluded that the etiological question of leprosy was very complicated:

> There are examples that either confirm or deny contagion; there are cases which support heredity but others that do not. I believe that both are positive but not decisive factors for the development of leprosy since it also requires some level of virulence and some personal and climatic conditions.[540]

[539] Heráclito Gómez, "Observaciones sobre la lepra griega, sus causas, y comarcas donde es más frecuente", *Anales de la Academia Nacional de Medicina*, Tomo I (Bogotá: Imprenta de la Luz, 1893), pp. 84-85.
[540] Ibid., p. 87.

The "seed and soil" metaphor in the hereditary/contagious considerations around leprosy

Historians have described how the idea that leprosy was predominantly hereditary rather than contagious was the predominant European medical explanation of the disease after the work of the Norwegians D.C. Danielssen and C.W. Boeck on leprosy in 1847 and until the discovery of Hansen bacilli in the 1870s.[541] Whereas by the late 1860s in Britain and in Europe in general, physicians endorsed those findings of Danielssen and Boeck,[542] in Colombia doctors tended to consider leprosy contagious and hereditary at the same time. I showed how Vargas Reyes (1862) considered plausible that leprosy was contagious but decided that it was hereditary only because its contagion had not been demonstrated. I also showed how for José María Ruiz (1867) contagion was the specific cause whereas, heredity and sudden change of temperature were predisposing ones. Ricardo de la Parra, the author of a book on Greek elephantiasis in 1867 was acquainted with Danielssen's work and described leprosy as a constitutional disease. But at the same time he included several predisposing, direct or occasional causes among which were contagion and heredity. Obregón argues that for Colombians of the 1860s heredity and contagion in leprosy were not contradictory facts and suggests that doctors did not follow European medical authorities on that matter (Danielssen and Boeck) because there was not a powerful medical community that could support it.[543] It was not until the 1880s that physicians would debate on heredity and contagion of leprosy, when the germ-theory of diseases had already spread. The main concern was not if leprosy was hereditary and how, but rather the implications that the bacteriological cause would have for the hereditary hypothesis. For explaining that, doctors recurred to the "seed and soil" metaphor.[544]

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[541] Rod Edmond, *Leprosy and Empire. A Medical and Cultural History* (Cambridge: Cambridge University Press, 2006), p. 45; Obregón, *Batallas contra la lepra*, pp. 118, 126-128.
[542] Ibid., p. 19.
[543] Obregón, *Batallas contra la lepra*, p. 118.
[544] Obregón explains the use of the metaphor in Juan de Dios Carrasquilla's work. Obregón, "The Social Construction of Leprosy in Colombia", p. 9. This metaphor was also common in contemporary accounts of other diseases and countries. See for example Michael Worboys, *Spreading Germs: Disease Theory and Medical Practice in Britain, 1865-1900* (United Kingdom: Cambridge University Press, 2000), p. 193 and Jo Robertson, "Leprosy and the elusive *M. leprae*: colonial and Imperial

For followers of Pasteurism, heredity was another way of transmitting leprosy like inoculation and infection; that is, children could would get the germ and the disease not like people inherit a particular constitution or predisposition but by inoculation or infection. Leprosy was not a constitutional disease. Castañeda believed that if microbes could find the appropriate "soil" they would reproduce and produce disease.[545] The answer to the question of why leprosy, being contagious, did not always attack those who came in contact with the "seed" (an argument in favor of the hereditary hypothesis), Castañeda responded that in a certain group of people whose "soil" was analogous because they belonged to the same race and lived under identical conditions in the same country, may be easily affected by leprosy in comparison with another group of a different race living in a different climate or affected by different living conditions; in the latter, contagion may come up against insurmountable difficulties.[546]

For Juan de Dios Carrasquilla (1833-1908) there were no diseases that were hereditary since diseases were not natural attributes of individuals; what could be transmitted was the predisposition to said diseases.[547] Repeating Castañeda's argument, Carrasquilla argued that resistance to contagion in some cases could be explained by the fact that microbial diseases could only develop under favorable conditions, a predisposed milieu, an apt chemical environment. This milieu varies according to organisms, races and climates, and even within a single organism it can vary from time to time.[548] Individuals could inherit the favorable soil for the development of the microbe but not the microbe itself. However, no matter how good the conditions were, without microbes, there would not be disease:

> Like a field could not give wheat crops if the seed is not watered, no matter how good or fertile the soil, no matter how excellent the fertilizers, similarly the human organism does not fall sick if it does not receive the seed, if the contagion agent does not fall into the body's trap, as Koch said, no matter how well apt the body were for the culture of

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medical exchanges in the nineteenth century", *Historia, Ciências Saúde-Manguinhos*, 10 (supplement 1), 2003, pp. 13-40.

[545] SMCN, "Acta de la sesión ordinaria del 20 de septiembre de 1888", *Revista Médica*, 130, 28 de Octubre, 1888, pp, 267-8; Gabriel J. Castañeda, "Contagio y herencia", *Revista Médica de Bogotá*, 138, 1 de junio, 1889, pp. 485-6.

[546] Gabriel J. Castañeda, "Patología. Estudio sobre la lepra en Colombia", p. 158.

[547] Juan de Dios Carrasquilla, "Disertación sobre la etiología y el contagio de la lepra", *Revista Médica*, 137, 15 de mayo, 1889, pp. 447-448.

[548] Ibid., p. 473.

> the pathogen germs. Heredity, misery, effluvia, emanations are all insufficient to produce a contagious disease if the microbe is not present.[549]

Other physicians also used the seed and soil metaphor in the sense that Castañeda and Carrasquilla did. Evaristo Garcia and Adolfo Tenorio from Cauca believed that "according to the microbian theory, the live germ, the seed of a disease must be associated with an appropriate soil in order for the disease to develop". What was inherited, they said, "is a particular aptitude, the adequate soil for the germs to develop". It is in that sense that they conceived of leprosy as an hereditary disease.[550] The discussion of the heredity of leprosy continued to be debated in Bogotá by several doctors, the majority of whom shared that view.[551]

The metaphor "seed and soil" had other uses apart from the rejection of the hereditary hypothesis of leprosy. It was used to explain why even though "we live surrounded by microbes", epidemics were temporary.[552] It was also used to criticize the emphasis that earlier followers of bacteriology put on microbes and the antiseptic treatment. Carlos Esguerra, the author of a medical-geographical oriented book on the Magdalena fevers (see chapter 3), believed that the emphasis on microbes during the first period of bacteriology resulted in a positive influence for antiseptic surgery but felt that it was dangerous for the treatment of internal diseases. Paraphrasing Peter, the famous adversary of Pasteur in the Paris Academy, Esguerra said that physicians of that period pointed to the microbe but killed the patient. The shift in the emphasis from microbes to the conditions of life of microbes, their evolution, and the proprieties of the "soil" in which the pathogenic species develop, during the second period of bacteriology, meant for Esguerra the return of clinical pathology and anatomic pathology to their rightful place within medicine, and the "liberation" of hygiene.[553] It seems that the "soil" of bacteriologists reminded Esguerra of the

[549] Ibid., p. 480.
[550] Evaristo García y Adolfo Tenorio, "Lepra", *Boletín de Medicina del Cauca*, 29 de Julio, 1889, pp. 796-819.
[551] Juan David Herrera, "Discusión sobre la herencia", *Revista Médica*, 141, 1 de septiembre, 1889, pp. 582-634; Gabriel J. Castañeda, "Las enfermedades microbianas no pueden ser hereditarias", *Revista Médica*, 142, 1 de octubre, 1889, pp. 635-670.
[552] Carlos Esguerra, "Discurso del doctor Carlos Esguerra", *Revista Médica*, 160-161, mayo, 1891, pp. 448-9.
[553] Ibid., p. 446.

hygienic conditions he considered fundamental in his previous medical geographical work on fevers.

Bacteriology and the controversy about serotherapy

The idea of leprosy being a specific disease transmitted by a specific agent would become the dominant theory in Colombia after Juan de Dios Carrasquilla's work on serotherapy in the 1890s.[554] Carrasquilla got his medical degree from the *Colegio Nacional in Bogotá* in 1852 and, according to his biographers, he never went to the Paris medical school to complete his studies.[555] Carrasquilla followed J. Héricourt and Charles R. Richet method of serotherapy, the treatment of a disease by the administration of the serum of an immunized animal. In 1888 the French tried to prepare serum for syphilis and tuberculosis without success. In 1890, Emil von Behring and Kitasato prepared serum for tetanus and diphtheria by inoculating rabbits and mice. They found that their immunity depended on the serum capacity of neutralizing toxins produced by tetanus and diphtheria bacillus.[556] According to Obregón, Dr. Carlos Putnam first applied serotherapy in Colombia to leprosy in 1895 in the Lazaretto *Agua de Dios* with good results (ulcers improved, and tubercles disappeared). But his work was interrupted since priority was given to Juan de Dios Carrasquilla's serum. Based on the analogy between leprosy and syphilis, whose etiological agent had not been cultivated, and following Héricourt and Charles R. Richet's method, Carrasquilla prepared immune serum from horses by injecting them with serum from lepers.[557] Carrasquilla applied his serum to 15 people for two months and informed the Academia Nacional de Medicina (ANM) – new name of the SMCN- in November 1895 that the lesions had receded, that the first injection of his serum had stopped the action of the bacillus; and, finally, that his procedure could cure the disease.[558]

[554] Obregón, *Batallas contra la lepra*, pp. 169-171.
[555] Eliseo Montaña, *El doctor Juan de Dios Carrasquilla*, (Imprenta y litografía de J. Casis, 1919).
[556] Ibid., p. 193.
[557] Ibid., p. 194.
[558] Carrasquilla's communications with the SMCN are found in Juan de Dios Carrasquilla, "Comunicaciones sobre el empleo de la seroterapia en la lepra, hechas a la Academia Nacional de

The ANM gave Carrasquilla priority for this treatment and rushed to send his results to Paris. Furthermore, the Ministry of Government offered their support and in December an institute and a hospital for serotherapy were created. The hospital was never established but the Carrasquilla Institute was set up in a central park of Bogotá.[559] From there the serum spread to the lazarettos and to other cities to be tested. Doctor Pedro Pablo Nates, along with five medical students, conducted experiments with the serum in *Agua de Dios* for several months. Roberto Azuero applied this therapy in 14 patients in *Caño del Loro*;[560] and it was also applied in Cali and Bucaramanga.[561] In this city, the founder of the Medical Society of Santander, Jesús Olaya Laverde, set up an institute for serotherapy in 1896 with Alejandro Peña Solano and two other physicians, with the support of the local government.[562] Instead of serum from the blood of patients, Olaya used juice of tubercles and blood for the injections in animals.[563] Olaya presented cases before the medical society of Santander and the governor of the Department who proposed to name the serotherapic institute of Bucaramanga the Olaya Laverde Institute.[564]

The ANM, carrying out the request of the national government to evaluate Carrasquilla's treatment, appointed a commission composed of Juan E. Manrique, Nicolas Osorio and Miguel Rueda in March 1896. By then Carrasquilla had treated 150 people, 60 of whom would attend his Institute.[565] The government was worried that Bogotá might become flooded with lepers who sought Carrasquilla's treatment. They blamed Carrasquilla's conclusion for this danger. One member of the ANM believed that people had been arriving in Bogotá due to Carrasquilla's conclusion –

Medicina de Bogotá (Colombia), por el señor doctor Juan de Dios Carrasquilla L.", *Revista Médica*, 205, mayo, 1895, pp. 306-307 and "Tercera comunicación sobre un procedimiento seroterápico aplicado al tratamiento de la lepra griega, presentada a la Acadamia Nacional de Medicina de Bogotá (República de Colombia), por el doctor Juan de Dios Carrasquilla L.", *Revista Médica*, 207, julio de 1896, pp. 368-9.

[559] Juan Pablo Gómez Ochoa, *Seroterapia Carrasquilla en la lepra. Resultados Obtenidos en Bogotá*. Tesis para el doctorado en medicina y cirugía (Bogtá: Imprenta Nacional, 1897), pp. 8-9.

[560] Diana Obregón, *Batallas contra la lepra*, p. 195.

[561] Evaristo García, "Seroterapia en la lepra", *Boletín de Medicina del Cauca*, 119-120, junio, 1897, pp. 98-102; Sociedad de Medicina del Cauca, "Acta de la sesion del dia 27 de abril de 1897", *Boletín de Medicina del Cauca*, 119-120, junio, 1897, pp. 102-107.

[562] Luis Fernando Otero, "Sociedad de ciencias médicas de Santander", *Revista Médica de Santander*, 5 y 6, febrero, 1897, p. 4.

[563] Jesús Olaya Laverde, "Segundo informe del director de los trabajos científicos del Instituto de Seroterapia aplicada a la lepra", *Revista Médica de Santander*, 5 y 6, febrero, 1897, pp. 10-35.

[564] Sociedad de Ciencias Médicas de Santander, "Sesión del 8 de Noviembre", *Revista Médica de Santander*, 5 y 6, febrero, 1897, pp. 5-9.

[565] ANM, "Sesión del día 18 de marzo de 1896", *Revista Médica*, 208, agosto de 1896, pp. 12-17.

which was without scientific basis, he said - that at soon as the serum was applied the action of the bacillus was interrupted.[566]

The commission faced several problems. They complained about the lack of laboratories for pursuing any bacteriological study.[567] J.D. Herrera argued that the proof of the usefulness of Carrasquilla serum had to be bacteriological, meaning that it has to be demonstrated that the the bacilli is no longer present in leprosy lesons, and that such proof would take time, especially for a chronic disease like leprosy. Besides, Herrera insisted, statistics and confirmation by competent scientific corporations were needed.[568] Lombana, another member of the ANM, added that even if it were the case that serotherapy could heal leprosy, people should be followed for several years to prove that the organism would no longer be favorable soil for the development of the disease. As such, he proposed to reduce the scope of the commission to an evaluation of the first modifications caused by serotherapy in a limited number of lepers.[569] Thus the JCH proposed to hospitalize a group of 12 sick people in order for the commission to study Carrasquilla's serotherapy. The government wanted to know whether it should continue to support the Carrasquilla Institute, whose staff cost 12,000 *pesos* per month, or use these resources to create a national lazaretto.[570]

The commission followed the 12 cases for nine months. In November 1897 they presented their conclusion: that Carrasquilla's treatment did not modify leprosy either in its symptoms, or in its clinical evolution. Thus they recommended the suspension of the serotherapy in lazarettos and the conversion of the Carrasquilla Institute into a bacteriological institute for the teaching and study of the new science.[571] The serum was not only tested at home. During the international congress in Berlin of 1897 it became evident that Carrasquilla's serum had been tried in Germany, London, Copenhagen and Paris, among other places, with diverse results. According to Obregón, the congress' judgment of Carrasquilla's work was not favorable, arguing that the Hansen's bacillus is not to be found in the blood of

[566] ANM, "Sesión del día 23 de abril de 1896", *Revista Médica*, 208, agosto de 1896, p. 21.
[567] ANM, "Sesión del día 18 de marzo de 1896", pp. 12-17.
[568] ANM, "Sesión del día 23 de abril de 1896", pp. 23-25.
[569] Ibid., p. 29.
[570] Ibid., p. 30.
[571] ANM, "Acta de la sesión del día 4 de noviembre de 1897", *Revista Médica*, 224, 28 de octubre, 1897, pp. 133-134.

patients. Thus Carrasquilla varied his strategy, and instead of using serum from the blood of patients he decided to culture the bacillus, but failed.[572]

In Colombia, the commission of the ANM evaluated Carrasquilla's serum not only on clinical grounds but also on microscopical analysis. The commission performed microscopic analysis of lesions before and after the nine months of treatment of the 12 patients and concluded that the Hansen bacillus was not modified either in its form or in its number during that period.[573] The commision's bacteriological observations confirmed its clinical conclusions.

Criticism also extended to Carrasquilla's methods. Juan Martín Restrepo, who was an intern at the Carrasquilla Institute for ten months, judged that the method for preparing the serum was defective because of the lack of aseptic and antiseptic measures: the sick were bled in the corridor of the Institute and Carrasquilla filtered the serum with only two layers of cotton and one in the middle with pulverized camphor; as a result, accidents such as abscesses in the place of the inoculations were frequent.[574] Furthermore, Restrepo denounced the Institute for a lack of microscopes and claimed that Carrasquilla could not, therefore, by any means, corroborate the full disappearance of the microbes after the treatment; besides, the clinical improvements that Restrepo witnessed did not seem superior to those obtained with the other preferred treatment, Chaulmoogra oil.[575]

A similar opinion was held by Andres Gómez, who helped with the ANM commission to study Carrasquilla's serotherapy. With the aid of Heliodoro Ospina, then professor of micrographic studies, he successfully cultivated the bacillus in serum and initiated immunization and serotherapy.[576] He said he could cultivate the bacillus in ox serum using the juice of tubercles and blood.[577] Gomez, like Restrepo,

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[572] Obregón, *Batallas contra la lepra*, p. 196.
[573] ANM, "Acta de la sesión del día 4 de noviembre de 1897", pp. 133-134. In charge of the microscopic preparations and culture of the bacillus was Heliodoro Ospina who had replaced Nicolas Osorio in the commission. Ospina had graduated in the medical school of Bogotá in 1874 and had gone to Paris to complete his medical studies, but we do not know what kind of courses he took in Paris; we only know that on his return to Colombia he taught zoology, histology and bacteriology. See José María Lombana Barreneche, "El doctor Heliodoro Ospina L.G.", *Revista Médica*, 220, agosto, 1897, pp. 19-20.
[574] Julio Martin Restrepo, *Estudo sobre la lepra y su tratamiento por la seroterapia*. Tesis para el doctorado en medicina y cirugia (Bogotá: Casa editorial de J. & L. Pérez, 1896), pp. 125-126, 137.
[575] Ibid., pp. 145-146.
[576] Andres Gómez, *Seroterapia en la lepra*. Tesis para el doctorado en medicina y cirugia (Bogotá: Imprenta Nacional, 1897), pp. X-XI.
[577] Ibid., pp. 15-17; 22-24.

was convinced that Carrasquilla's process and conclusions were problematic: Carrasquilla diagnosed leprosy without the use of microscopes, and so misdiagnoses based on clinical criteria were common. Besides, the process of production of serum was empirical, not scientific, because it had not been demonstrated that toxins were always present in the blood. Convinced that any effective serum should kill microbes, Gomez applied Carasquilla's serum directly to samples of the germ, proving that they still continued to move and therefore that the serum was useless.[578] Juan Pablo Gomez, another intern, also believed that clinical criteria were not enough to decide whether or not Carrasquilla's serum was effective and that better results had been obtained with Chaulmoogra oil.[579] He confirmed that "everything that has to do with serotherapy in the Institute has been done without any microscopic examination".[580]

Despite Carasquilla's therapeutic failure, the interest aroused by his claims served to popularise bacteriological methods among Colombian physicians, leading to their acceptance as the key criteria for investigating leprosy, and then for other diseases as well. First, it fostered the growing awareness of the need to incorporate bacteriological analysis into clinical and hygiene practices. Between 1894 and 1896 the JCH prepared two projects for the creation of a laboratory for bacteriological studies, with the aim to perform micrographic studies, hygiene studies, research on diseases of animals and plants, and to carry out work cultivating microbes and preparing vaccines and serums. The Ministry of Public Instruction had sent a professor to Europe to study bacteriology, but it would only be at the end of the century that the envoy and a laboratory material would arrive.[581] In the meantime, in Medellín, Juan B. Montoya y Florez established a theoretical and practical training course on clinical bacteriology in 1896, based on his course with Emile Roux in the Pasteur Institute. This course was initially offered as a private course in Montoya's private laboratory.

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[578] Ibid., pp. 26-8, 35-6.
[579] Juan Pablo Gómez, *Seroterapia Carrasquilla en la lepra. Resultados obtenidos en Bogotá* (Bogotá: Imprenta Nacional, 1897), pp. 20-22; 76.
[580] Ibid., p. 77.
[581] Pablo García Medina, "Informe presentado al Sr. Ministro de Gobierno por el secretario de la Junta Central de Higiene, sobre los trabajos de esta corporación en los años de 1894 a 1896", *Revista de Higiene*, 54-55, septiembre y octubre, 1899, pp. 485-486.

Second, medical students involved in testing Carrasquilla's serum in lazarettos wrote their medical thesis on the subject and others turned to bacteriology: they wrote about and performed works on the serum diagnosis of typhoid fever, the cultivation of the Koch bacillus and the identification of microbes of erysipelas, anthrax and pneumonia. These works were named by one of them as the first "experimental bacteriology" works to be carried out in Colombia.[582] They were performed in the laboratory of the student Victor Gomez with modest resources: they used plates, stains and a microscope costing 500 Francs, and used alcohol to light the lamp of the stove invented in Bogotá for the cultures. This implied that they had to keep watch day and night to keep the temperature constant.[583] They inoculated animals with cultures of the microorganisms they cultivated.[584] The lack of resources such as artificial culture mediums for this kind of work meant that they were replaced by "natural" culture mediums such as eggs.[585] Despite those efforts, bacteriology would not become part of everyday clinical practice until well into the twentieth century.

If the germ cause for leprosy was finally accepted in Colombia after the debates around Carrasquilla's serotherapy, what happened with the neo-Hippocratic and medical geographical causal scheme? The reconfiguration of these causal models by bacteriology, in the case of leprosy, is summarized in Juan N. Rondon's thesis of 1897 on the causes of the disease. Rondon participated in the inoculations of Carrasquilla's serum at the *Agua de Dios* lazaretto. He claimed that the "essential" cause of leprosy was the Hansen bacillus and that the "predisposing" causes were the same as for other infectious diseases: causes that debilitate the organism or modifiy it make it apt for the development of the specific germ.[586] Before medicine considered

[582] Gomez, *Seroterapia en la lepra*, pp. XI-XII.
[583] Luis Zea Uribe, *El bacilo de Eberth y serodiagnóstico en la fiebre tifoidea*. Tesis para el doctorado en medicina y cirugía (Bogotá: Imprenta Nacional, 1898), pp. 10-12.
[584] Belisario Castro, *Etiología de la tuberculosis. Cultivo de su bacilo*. Tesis para el doctorado en medicina y cirugía (Bototá: Imprenta Nacional, 1897), p. 21.
[585] Indalecio Camacho, "Estudio de una afección endémica en las regiones llamadas templadas en Colombia, conocida vugarmente con el nombre de *puercas* o *marranas*", *Revista Médica de Santander*, 1 y 2, septiembre, 1894, pp. 24-28.
[586] Juan N. Rondon, *Contribución al estudio de la etiología de la lepra griega.* Tesis para el doctorado en medicina y cirugia (Bogotá: Imprenta Nacional, 1897), p. 13.

Hansen bacillus to be the cause of leprosy, Rondon reminded his readers, doctors believed that poverty and bad hygienic conditions were the first and essential causes of leprosy.[587] Indeed, Daniel C. Danielsen and C.W. Boeck, who established leprosy as a nosological entity in 1847, conceived it mostly in hereditary terms but also believed that some cases were due to unhealthy and difficult environmental and living conditions.[588] In an attempt to recover the importance of the role of those "predisposing" causes, Rondon produced figures for meat consumption in Colombia and linked them with the distribution of leprosy. These figures showed that leprosy dominated in Departments where there was less consumption of meat, that is, in Cundinamarca, Santander and Boyacá, compared to places with higher rates of meat consumption such as the Departments located on the coasts. But Rondon also established a causal relationship between this distribution of leprosy and climate: the fact that leprosy predominated in temperate climates and was rare in warm climates and on the coasts could be explained because the burning rays of the sun, by increasing temperature, killed the "parasites".[589] On the eve of the nineteenth century, when the Colombian medical community finally agreed that the essential cause of leprosy was the Hansen bacillus, doctors simultaneously considered it plausible that geographical features such as temperature determined the occurrence and distribution of leprosy: temperature – as Rondon suggested in 1897 – would ultimately decide whether the bacillus lived or not.

[587] Ibid., p. 29.
[588] Obregón, "The Social Construction of Leprosy in Colombia", p. 5.
[589] Rondon, *Contribución al estudio de la etiología de la lepra*, pp. 31-34.

CONCLUSIONS

This dissertation aimed to detail how one particular medical community made sense of the conceptual transformation brought about by the Pasteurian theory of disease causation and the consequent transformation of the identity of epidemic diseases. In the introduction, I pointed to the fact that this subject has been neglected by historians. Scholars have traditionally preferred to discuss the theories of disease causation before germ theory or the establishment of the germ theory; few have addressed the problem of how the latter was incorporated and transformed by the former (as I said in the Introduction, the few who have addressed this problem in particular are Michael Osborne, Andrew Cunningham, Marcos Cueto and Diana Obregón).[590] In any case, historians dealing with one or the other have suggested ways in which that process might have occurred. Some have pointed to the continuity between the pre-bacteriological and the bacteriological notions in the sense that some of the germ theory notions or meanings resonated with, or were actually present in, previous theorizations (William Bulloch, W.A. Foster and K. Codell Carter).[591] Others have focused on the discontinuity aspects, arguing that germ theory was incommensurably different from previous theories (Andrew Cunningham).[592] Finally, some have highlighted the coexistence or synthesis between previous theories of disease causation and germ theory, at least during the first decades of the definition and diffusion of germ theory (Michael Osborne and Diana Obregón).[593] These three different interpretations depend heavily on the germ-oriented approach of historians: they choose to look for either similarities or differences between

[590]Michael A. Osborne, "The Geographical Imperative in Nineteenth Century French Medicine" in Nicolaas A. Rupke, "Medical Geography in Historical Perspective", *Medical History*, Supplement No. 20 (London: The Wellcome Trust Centre For the History of Medicine at UCL, 2000), pp. 31-50; Andrew Cunningham, "Transforming Plague. The Laboratory and the Identity of Infectious Disease" in Andrew Cunningham and Perry Williams (eds.), *The Laboratory Revolution in Medicine* (Cambridge: Cambridge University Press, 1992), pp. 209-244; Marcos Cueto, "Nationalism, Carrión's Disease and Medical Geography in the Peruvian Andes", *History and Philosophy of the Life Sciences*, 25 (2003): 319-335, and Diana Obregón, *Batallas contra la lepra: Estado, Medicina y Ciencia en Colombia* (Medellín: Eafit, 2002).

[591] William Bulloch, *The History of Bacteriology* (London, Oxford University Press, 1960 [1938]); W.A. Foster, *A History of Medical Bacteriology and Immunology* (London: William Heinemann Medical Books, London, 1970); and K. Codell Carter, *The Rise of Causal Concepts of Disease* (Aldershot: Ashgate, 2003).

[592] Cunningham, "Transforming Plague".

[593] Osborne, "The Geographical Imperative" and Obregón, *Batallas contra la lepra*.

nineteenth-century doctors' definitions of germs and the opinions that those doctors held before the idea of germs emerged. I took another perspective. I first explored pre-bacteriological meanings of disease causation in order to understand how Colombian physicians subsequently made sense of germs and transformed those meanings. My intention was to express the multiplicity of the causal elements that were involved in disease according to doctors of the pre-bacteriological era, and explain how germs came to be understood by doctors starting from that standpoint.

Although this research focuses on fevers and leprosy, and I have not considered other areas in which germ theory was applied such as antiseptic surgery and epizootics, it is possible to put forward a provisional periodization of the history of the causal considerations of human disease in nineteenth-century Colombian medicine: first, the neo-Hippocratic period which extends from 1800 to 1850, when doctors adapted the neo-Hippocratic scheme of specific, predisposing and occasional causes to the local context, and secondly, the medical geographical period from 1850 to 1882 during which French-medicine-oriented doctors incorporated the neo-Hippocratic causal scheme within medical geography. It is true that between 1870 and 1882 physicians were aware of the germ theories associated with antiseptic surgery on the one hand, and with causes of leprosy on the other. However, the Pasteurian germ theory (or the "parasitic" theory as contemporaries called it) would only begin to transform the causal approach to disease from 1882 onwards. Thus, the third period, between 1882 and 1891, was that of the intense reconfiguration of the medical geographical and bacteriological models. The fourth and last period, from 1891 until the end of the century, was that of the awareness that bacteriology and laboratory techniques should be applied in the diagnosis of other diseases, despite the persistence of medical geographical notions.

The ways in which Colombian physicians (and physicians acting as hygienists) incorporated germs into medical geography, and the ways in which the latter was transformed by bacteriology, were strongly determined by three elements: the situation of the medical community as it went through the process of professionalization; the diseases that physicians decided were most relevant to their studies according to their professional interests and the sociopolitical circumstances; and finally, the therapeutic practices suggested by germ-theory-oriented doctors,

regardless of whether or not those practices succeeded. In what follows I will refer to these elements following the periods mentioned above.

Neo-Hippocratism and medical geography

The scarce evidence suggests that during the neo-Hippocratic period (between 1800 and 1850), Colombian physicians fully embraced the neo-Hippocratic scheme of specific, predisposing and occasional causes. Most importantly, they used the climatic determinism implicit in this model to explain both the predisposition or resistance to diseases and the physical and moral differences between the *castas*, namely "whites", "blacks" and "*indios*". These ideas persisted for several decades after the political independence from the Spanish crown in 1819. The leaders of the independence wars (the "whites") not only retained the economic institutions inherited from colonial times (monopoly of production and distribution of tobacco and slavery, for example) but also preserved in practice the social distances between the *castas*. The continuity of these institutions would explain why elite doctors, such as Jose Felix Merizalde, adapted the neo-Hippocratism and its climatic determinism to explain why *indios* were docile, suitable for certain kinds of work, and acclimated (that is, resistant) to certain diseases, compared to whites and *mestizos*.

From 1850 until 1882, during the medical geographical period, doctors integrated and reformulated these neo-Hippocratic notions with the newer French medical geography, according to the new socio-political circumstances. Physicians who returned from the Paris medical school from the 1850s onwards strove to consolidate the national medical community by creating the first ever medical journals and a private school. They justified that effort by claiming that it was necessary to build a "national medicine" based on medical geographical studies. They referred to the French medical geographical concepts that emphasized the local origin of diseases. Accordingly, doctors focused their attention on the "local" pathologies and the geographical boundaries of the country's diseases, following the ill-defined division between warm, temperate and cold lands. Although explicit references to *castas* in relation to diseases reduced dramatically in medical texts at this time, doctors would

use the term "races" in order to make sense of the physiological responses to diseases in different climates. The neo-Hippocratic scheme of specific, predisposing and occasional causes, as well as the crucial role of climate as predisposing and occasional cause, survived in the medical geographical model though it was adapted to the new socio-political and professional conditions of the time.

Indeed, this period coincided with the "liberal hegemony" in politics. From 1850, the government implanted liberal reforms that were designed to end the social and economic institutions of the colonial era (slavery, monopoly of tobacco production and distribution, among others). With these reforms the rulers aimed to integrate the Colombian economy into international world economy as an exporter of primary goods such as tobacco.[594] This political situation, and the liberal spirit that dominated the elite (including physicians such as Antonio Vargas Reyes and Domingo Esguerra),[595] explain why they focused their attention upon the intermittent or periodic fevers of the Magdalena river valley where tobacco was produced. Doctors identified the intermittent or periodic fevers of the Magdalena River as diseases locally produced by the action of heat and humidity on the organic matter. Magdalena Fevers would be a case in point for medical geographical studies and the disease upon which they insisted on the creation of a national medicine during those years.

Given these socio-political conditions and professional interests, it is not strange that doctors would attribute the role of the determinant cause of Magdalena fevers to the climate, and extend this approach to other diseases. Indeed, diseases such as leprosy and typhoid fever, which physicians did not consider as important as Magdalena fevers, were also approached using the neo-Hippocratic and medical geographical model. Doctors had claimed that sudden changes of temperature were the most common cause of leprosy, but they also wrote about its contagious nature. One homeopath claimed to have established a "parasitic" cause for leprosy using Tyndall's ideas on germs in 1870, but given the opposition to homeopathy from

[594] José Antonio Ocampo, *Colombia y la Economía Mundial. 1830-1910* (Bogotá, Tercer Mundo, 1998), pp. 21-43; Luis Eduardo Nieto Arteta, *Economía y cultura en la historia de Colombia* (Bogotá, El Ancora Editores, 1983), pp. 214-228; and, finally, Luis Ospina Vásquez, *Industria y Protección en Colombia 1810-1930* (Medellín, FAES, 1987), p. 223.

[595] I developed this argument in "Las 'fiebres del Magdalena': medicina y sociedad en la construcción de una noción médica colombiana, 1859-1886", *Historia, Ciencias, Saude-Manguinhos* (Rio de Janeiro), 14, 1, ene.-mar., 2007, pp. 63-89.

regular practitioners, this theory was not taken up by physicians. Contagion, heredity and changes of temperature would prevail as the plausible causes of the disease during this period.

Doctors considered that typhoid fever was a miasmatic disease which was not clearly determined by climate. During the 1870s, however, debates surrounding the typhoid epidemics of Bogotá revolved around the law of pathological antagonism between intermittent and typhoid fevers postulated by the French medical geographer Jean Boudin. Physicians struggled to explain why typhoid fever cases occurred simultaneously or consecutively with intermittent fevers. Given the fact that doctors were interested in creating a "national medicine" through medical geographical studies (the study of local pathologies), thus claiming scientific authority over the knowledge of local pathologies, exceptions to the medical geographical laws set by the French medical geographer would give Colombian doctors the opportunity to reinforce their authority over that knowledge.

Reconciling medical geography and the germ theory

The third period, between 1882 and 1891, was that of the reconciliation between medical geography and the germ theory. Doctors turned their attention to the germ theory as a result of Pasteur's propaganda regarding chicken cholera and rabies vaccines in the early 1880s. The role of Gabriel J. Castañeda was crucial during those years. He was the first and most enthusiastic supporter of Pasteurism in that period, and he struggled to move physicians' interests from Magdalena fevers to leprosy. However, for most of the decade, physicians' main concern was still the intermittent and periodic fevers of the warm lands, particularly the variety of yellow fever. Periodic and intermittent fevers not only began to climb from the Magdalena River up to the *eastern* cordillera along the way to the capital city, Bogotá, but also affected Cúcuta. The latter was located in the region of Santander, the most important producer of coffee at the time: in Cúcuta, coffee was stoked to be exported via the Maracaibo Lake. Coffee had begun to replace tobacco as the main export when tobacco exports started to decline in 1870. The threat of intermittent or

intermittent fevers to the capital city, Bogotá, and the occurrence of fevers in Cúcuta, a city prosperous due to coffee export, were crucial in the debates and reconfiguration of medical geography by the germ theory.

Indeed, doctors reconciled the medical geographical explanation of Magdalena fevers and fevers in Cúcuta with the germ theory. A range of explanations emerged: some considered that microorganisms were the only cause (and were almost opposed to any climatic cause of diseases); others struggled to retain the idea that climate and geography were active elements in their production. Cuervo Marquez, who practiced medicine in the Magdalena River region, was aware of the international efforts to identify the germ causing yellow fever and even recognized the primary role of germs; however, he strove to retain winds and heat as precursors of yellow fever epidemics. Carlos Esguerra, on the other hand, had a very generic conception of germs which did not essentially transform contemporary conceptions of the way that miasmas worked.

In their views, germs or microorganisms related to climate in different ways. For Cuervo Marquez, heat could modify the power of germs to produce yellow fever epidemics, whereas for Esguerra, there did not seem to be any difference between germs and miasmas: the development of the "agent" strongly depended on the "culture" conditions, that is, the environmental and hygiene conditions. I only found one reference to the notion of virulence to explain the occurrence and disappearance of yellow fever epidemics in Cúcuta: climate would modify the action of microorganisms. The historian Andrew Mendelsohn has suggested that because the Pasteurian notion of virulence implied that climate played a role in the emergence and disappearance of epidemics, there was no opposition between climatic and germ-theory oriented causes of disease.[596] Mendelsohn was criticizing the historian Michael Osborne, who assumed that both climatic and bacteriological models were opposed.[597] Mendelsohn and Osborne both assume that germs or microorganisms carried an essential meaning (depending on whether they incorporate or exclude climate elements in that meaning): I found Colombian doctors that used the notion of virulence in the sense explained by Mendelsohn, and also those who saw

[596] Andrew Mendelsohn, "'Like All That Lives': Biology, Medicine and Bacteria in the Age of Pasteur and Koch", *History and Philosophy of the. Life Sciences.*, 24, 2002, p. 12.
[597] Ibidem..

bacteriology as distinct or opposed to previous climatic notions in the sense explained by Osborne.

Despite Castañeda's propaganda with regards to the microbiological cause of leprosy in 1882, when he explained that sudden changes of temperature (heat and cold) acted as a mechanism that opened and closed the doors to the microorganism of leprosy, physicians would only take that cause seriously at the end of the century. In the late 1880s, Castañeda promoted a national survey about the distribution and causes of leprosy with the intention to call physicians' and the government's attention to the disease. Obregón has shown that doctors exaggerated leprosy as a way of gaining social recognition, partially achieved during the medical geographical era. The survey showed that physicians around the country considered food, contagion, heredity and sudden changes of temperature to be causes of leprosy. Castañeda thus transformed his idea about the role of climate on leprosy: though he had started by considering it to be a mechanical factor, he ended up admitting the importance of climate to the production and geographical distribution of leprosy. This illustrates the strength of the medical-geographical-oriented thinking at that time.

The pre-eminence of fevers of the warm lands as pathologies worthy of study in the first years of the 1880s - due to their occurrence in areas of economic significance - over the problem of typhoid fever in Bogotá, and physicians' new interest in leprosy by the end of that decade, may explain why doctors thoroughly adapted the germ theory to explain yellow fever while seeming almost indifferent to the idea that the *Eberth* bacillus caused typhoid fever. The same issue could explain why the germ theory of antiseptic surgery, which had been divulged during the 1870s in the Colombian medical literature, did not affect the view of the causality of epidemic diseases.

The last and fourth period, which lasted roughly from 1891 until the end of the century, was dominated by debates about leprosy, in particular serotherapy and the first trials with laboratory techniques for diagnosing other diseases. Following the decline in epidemics of fevers of the warm lands and Cúcuta by the early 1890s, physicians' attention was concentrated on leprosy. As I have said, Obregón has explained the professional reasons for physicians to exaggerate leprosy statistics. But

I also found that the exaggeration was triggered by Gabriel J. Castañeda. He divulged the antiseptic treatment for leprosy based on the germ theory in 1882 and tried to capture doctors' attention in 1888 by exaggerating the cases of leprosy as a way of justifying the national survey on the disease.

The most systematic description of the role of climate and germs in this period of reconfiguration or reconciliation of both models was offered by Luis Cuervo Márquez. In his work on yellow fever and malaria, Cuervo Marquez proposed a causal triad: the first was the specific agent (microorganisms in yellow fever, miasmas in malaria); the second was the "soil" or bodily conditions; and the third, the "cosmic" conditions, the environment. However, until the end of the century, the majority of physicians continued to use the old scheme of specific, predisposing and occasional causes and to recur to climatic factors to explain both the geographical distribution of diseases and what could not be explained by the action of germs alone. In 1897 one doctor claimed, for example, that sun heat could kill the leprosy germ thus determining the geographical distribution of leprosy.

No matter the approach to germs that physicians might have had, I found that they invariably defended the germ theory from critiques that might arise (the failure to comply with Koch's criteria for affirming that one microorganism was the specific cause of a disease, i.e. isolation, cultivation and inoculation to reproduce the disease) by using another of Koch's ideas: that even if a germ that was supposed to be the cause of a disease could not be cultivated or inoculated, it did not mean that it was not the specific germ for that disease. In fact, I did not find open opposition to the Pasteurian cause of diseases except in the case of typhoid fever. Few doctors defended the spontaneous generation of typhoid fever using medical geographical arguments: if typhoid fever occurred independently from the climate, and there was no evidence of contagion, then it must have spontaneously originated in the body. But this idea was marginal.

About the transformation of the identity of diseases

In investigating the transition from medical geography to germ theory, this dissertation also explored the transformation of the identity of diseases. Following the scholars working in the SSK in supposing that meanings and use of concepts are capable of change because they are not pre-determined, I showed how physicians in Colombia transformed the nosology of fevers in an uneven way in the process. Although typhoid fever was understood within the frame of predisposing, occasional and specific causes, and was mostly considered to be associated with miasmas from the environment, filth and overcrowding, the *Eberth* bacilli that were proclaimed to be the cause did not cause controversy. At the time of the arrival of germ theory, Colombian doctors were not interested in the causes of typhoid fever. As I said, their interests lay in contradicting the law of pathological antagonism between intermittent and typhoid fever that had been set up by French medical geographers. Doctors did not find it useful to refer to the bacteriology in order to differentiate between typhoid fever and typhus. They chose mainly to differentiate them on the grounds of clinical thermometry well into the last decade of the century.

Yellow fever and malaria were considered clinical varieties of intermittent or periodic fevers, locally produced by miasmas and not contagious. The differentiation between the two did not begin with the germ hypothesis; nor was it deduced by comparing epidemics in the seaports, as Coleman says was the case with European and American physicians.[598] In Colombia, doctors noticed that one of the varieties of the intermittent or periodic fevers of the Magdalena River was constricted by the boundaries of the hot climates of the river's valley, while another type was liberated from that geographical limit, moving up the Andes slopes to temperate lands and breaking out in unexpected places like Cúcuta. Germs, which physicians discussed as a consequence of the controversy over the preventive inoculations, were at hand to explain the mobility of such fever (to be known as yellow fever), while the miasma served to help describe the other's dependency on climate (the malarial variety). This move consolidated the distinction between two fevers: yellow fever and malaria.

Despite the highly controversial nature of the germ of yellow fever in the international debates of which Colombian doctors were aware (the *Peronospora Lutea* and the *Criptococus Xantogenicus* among others), they easily accepted the

[598] William Coleman, *Yellow Fever in the North. The Methods of Early Epidemiology* (Madison: The University of Wisconsin Press, 1987), p. 42-45.

germ hypothesis for yellow fever and even sought a chemical vaccine. On the contrary, doctors were indifferent to the *Eberth* bacillus as the cause of typhoid fever, even though the international community was almost simultaneously recognizing it as the main cause of the disease.[599] In the case of leprosy, although the germ that caused it was launched earlier than with the other two cases (first by the homeopath in 1870, at which point it was rejected, and then again in 1882), it was only finally accepted in the 1890s. So the Colombian medical community incorporated, in different ways and different velocities, the germ hypothesis for different pathologies according to local conditions and physicians' interests. These different patterns for the introduction of the germ hypothesis followed the relative importance that doctors conferred to those diseases in both economic and professional terms, as I said above.

When I began this project, I expected that the transition from medical geography to germ theory would have been completed by the turn of the century. Indeed, the causal shift did occur in the two last decades of the century, but I was surprised by the ongoing presence of climate and geographical traits in doctors' minds. Geography was still considered a predisposing cause, and for some it was the factor that explained what germs could not: the resistance to certain diseases and the geographical distribution of diseases. Climate and geography were now at the periphery of the ideas regarding disease causation, but persisted until the end of the century. The way that climate and geography were reframed with the development of a new medical field, tropical medicine, and in a new professional and social scenario, should be the subject of another investigation.

Therapeutics and the germ theory

One of the most interesting aspects shown by this research is the impact of germ-theory-oriented therapeutics on the critiques and acceptance of the germ theory. The antiseptic treatment promoted by Castañeda for typhoid fever and leprosy in 1882, the preventive inoculations applied to yellow fever in Cúcuta in 1887, the chemical vaccine invented in Bogotá in 1889, and Carrasquilla's serotherapy for leprosy in

[599] Dale C. Smith, "The Rise and Fall of Typhomalarial Fever", *Journal of the History of Medicine and Allied Sciences*, 37, 1982, p. 299.

1895 strongly influenced (and in some cases were the turning point in) the ideas about causes of disease. They also played an important role in securing a foothold for bacteriological methods in Colombia.

The evidence suggests that Colombian doctors were initially skeptical of the germ theory because they could not see any practical use for it. Despite Castañeda's propaganda with regards to the "parasitic theory" from 1882 onwards, and his therapeutic recommendations (antiseptic treatment for leprosy and typhoid fever), Nicolas Osorio attacked his enthusiasm with regards to Pasteurism, arguing that antiseptic treatment was an excess. Some years later, Abraham Aparicio suggested that bacteriology was meaningless when taken out of the context of clinical medicine. Colombian doctors would only begin to take the Pasteurian cause of yellow fever seriously after the debates surrounding both the preventive inoculations against yellow fever in Cúcuta and leprosy after Carrasquilla's serotherapy. Inoculations for the treatment or prevention of yellow fever were not new, but were reinterpreted in light of Pasteur's discoveries. Both the national government and the local authorities in Cúcuta took the inoculation method developed by the Mexican doctor, Manuel Carmona y Valle, seriously. The national government motivated the medical elite located in Bogotá to study Carmona y Valle's book, and doctors and merchants in Cúcuta decided to apply its methods during the 1887 epidemic of fever. As I mentioned earlier, Cúcuta was the main center from which coffee was being exported, and the need to tackle the epidemic in order to reduce the economic impact of the fever surely prompted not only the application of inoculations but also the acceptance of the germ cause by locals. Despite doubts regarding the nature of the fever, doctors in Bogotá that defended Pasteurism took the opportunity to emphasize the microbiological cause of yellow fever, though they questioned the effectiveness of the inoculations. They considered that the inoculations failed because the techniques used did not follow Pasteurian methods, thus reaffirming the germ theory. Sometimes they used contradictory arguments: according to the critics, the lack of certainty about the identity of the germ that was being inoculated would explain the unsuccessful inoculations, even though they knew that Pasteur had developed the rabies vaccine without any clear identity of the microorganism inoculated. There is

no doubt that the search for a chemical vaccine in Bogotá in 1889 reflects how sure doctors were about the microbiological nature of yellow fever.

By the time that doctors turned their attention to leprosy, the conditions were already set, paradoxically, for a positive welcome of new therapeutics based on germ theory. Serotherapy, developed by Juan de Dios Carrasquilla, was a success for a while: patients flowed from different regions to be treated in Bogotá, the medical community proclaimed Carrasquilla as the inventor of the method and the national government created and supported the Instituto Carrasquilla in Bogotá (the local government of Santander created the Serotherapic Institute in Bucaramanga). But critiques of Carrasquilla's method soon appeared from the new generation of doctors that were trained in Bogotá in the 1890s, who criticized his failure to cure leprosy by arguing that there were failures in the techniques used to produce the serum. They used bacteriological criteria to evaluate those therapies, and did not question the validity of the Pasteurian approach despite the blatant failure of those techniques. On the contrary, the very same critiques of Carrasquilla's methods prompted these young doctors to study and try using the new bacteriological methods to diagnose other diseases.

Somehow, the new bacteriological methods and theory affected doctors' belief in their ability to produce original knowledge. In the medical geographical era they claimed that direct experience of diseases in the climates where they were supposed to originate was a good source of original medical knowledge and scientific authority and the basis for creating a national medicine based on the new science. Earlier supporters of Pasteurism such as Gutierrez began to claim that bacteriology would enhance national medicine, but did not mention the possibility of producing original contributions to universal medicine. I could not find any references from physicians claiming that they could make original claims based on medical geography *and* bacteriology. It seems that during the last years of the nineteenth-century transition from medical geography to germ theory, Colombian doctors felt more insecure about their abilities to produce new knowledge or challenge European medical knowledge. Geography could not compete with the laboratory as the site of production of original medical knowledge.

BIBLIOGRAPHY

Primary sources

Journals and newspapers

Archives de Physiologie Normale et Pathologique
Anales de la Universidad Nacional de los Estados Unidos de Colombia
Anales de la Academia de Medicina de Medellín
Boletín de la Sociedad de Medicina del Cauca
Diario Oficial
El Tiempo
El Pueblo
Gaceta Médica
La Lanceta
Revista Científica e Industrial
Revista de Higiene
Revista Médica
Revista Médica de Santander
Registro Municipal

Printed sources

Boudin, J. Ch. M. *Essai de Géographie médicale or études sur les lois qui président a la distribution géographique des maladies, ainsi qu'a leurs rapports topographiques entre elles. Lois de coïncidence et d'antagonisme* (Paris: Germer-Bailliére/Ladé, 1843).

Boshell, Jorge. *La fiebre continua del hospital de San Juan de Dios*. Tesis para el doctorado en Medicina y Cirugía (Bogotá, Imprenta Echeverría, 1889).

Brunache. "Recherches sur la phtisie pulmonaire et la fièvre typhoïde considérées dans leurs rapports avec les localités marécageuses" in J. Ch. M. Boudin, *Etudes*

de géographie médicale. Notamment sur le question de l'antagonisme pathologique (Paris: J.-B. Baillière, 1846), pp. 21-59.

Buendia, J.M. [informe "epidemia de angina diftérica"], *Revista Médica*, Serie I, No. 2, 2 de agosto (1873), 10.

Caldas, Francisco José de. "Preliminares para el Almanaque de 1811", *Obras completas de Francisco José de Caldas* (Bogotá, Universidad Nacional de Colombia, 1966 [First edn. 1811]), 401- 410.

---- "Del influjo del clima en los seres organizados", *Obras Completas de Francisco José de Caldas* (Bogotá: Imprenta Nacional, 1966 [1808]), 79-120.

Carmona y Valle, Manuel. *Leçons sur l'étiologie et la prophylaxie de la fièvre jaune* (México: Impr. du Ministère des Travaux Publics, 1885).

Castañeda, Gabriel J. *Causa y tratamiento racional de la lepra de los griegos hallados por inducción* (Bogotá: Imprenta de Echeverría Hermanos, 1882).

---- "Patología. Estudio sobre la lepra en Colombia", *Anales de la Academia Nacional de Medicina*, Tomo I (Bogotá: Imprenta de la Luz, 1893), pp. 151-181.

Carrasquilla, Juan de D. "Tercera comunicación sobre un procedimiento seroterápico aplicado al tratamiento de la lepra griega, presentada a la Acadamia Nacional de Medicina de Bogotá (República de Colombia), por el doctor Juan de Dios Carrasquilla L.", *Revista Médica*, 207, julio, 1896, pp. 368-9.

---- "Comunicaciones sobre el empleo de la seroterapia en la lepra, hechas a la Academia Nacional de Medicina de Bogotá (Colombia), por el señor doctor Juan de Dios Carrasquilla L.", *Revista Médica*, 205, mayo, 1895: 299-308.

---- "Consideraciones acerca de la etiología y de la profilaxis del paludismo", *Anales de la Academia Nacional de Medicina*, Tomo I-Entrega Primera (Bogotá: Imprenta de "La Luz", 1893), pp. 3-70.

---- "Disertación sobre la etiología y el contagio de la lepra", *Revista Médica*, 137, 15 de mayo, 1889: 441-484.

Castro, Belisario. *Etiología de la tuberculosis. Cultivo de su bacilo.* Tesis para el doctorado en medicina y cirugía (Botótá: Imprenta Nacional, 1897).

Cornil A-V. and V. Babes. *Les bactéries et leur role dans l'anatomie et l'histologie pathologiques des maladies infectieuses* (Paris: Félix Alcan Editeur, 1886).

Colombia, Secretaría del Estado y del Interior. “Decreto 720 [3 de Octubre, 1826], Reglamentación de la Educación Pública”, José M. de Mier, *La Gran Colombia. 2. Decretos de la Secretaría del Estado y del Interior, 1825-1826* (Bogotá: Presidencia de la República, 1983), 587-649.

Cuervo Márquez, Luis. *La fiebre amarilla en el interior de Colombia. Epidemia de Cúcuta-Fiebres del Magdalena* (Curazao: Imprenta de la librería de A. Betencourt é hijos, 1891).

Cuervo Marquez, Luis to Nicolás Osorio, Abril 14, in Luis Cuervo Márquez, *La fiebre amarilla en el interior de Colombia, Epidemia de Cúcuta-Fiebres del Magdalena* (Curazao: Imprenta de la librería de A. Betencourt é hijos, 1891): 262-268.

Chomel, Auguste Françoise. *Élémens de pathologie générale* (Paris, Crochard/ Gabon, First edn. 1817).

Duran, Samuel. “Elefantialsis de los Griegos” (Tesis para el doctorado en medicina y cirugía), *Anales de la Universidad Nacional de los Estados Unidos de Colombia*, 8 (67-72), 1874: 455-501.

Esguerra Ortiz, Domingo. *Memoria sobre las fiebres del Magdalena* (Santa Ana: Imprenta de D. Díaz, 1872).

---- *Trabajos médicos sobre las fiebres de Colombia* (Bogotá: s/e, 1877).

Esguerra, Carlos. *Contribution a l’étude de la fièvre du Magdalena* [Thesis for the medical degree] (Paris: Imprimerie des Écoles, 1889).

Eslava M., Lorenzo. *Contribución al estudio de la elefancia de los arabes en Colombia* (Bogotá: Imprenta de la Luz, 1892).

Facultad de Medicina de Bogotá, *Epidemia reinante en Bogotá, a fin de 1830; y preceptos de higiene publica* (Bogotá: Impr. de B. Espinosa por José Ayarza, 1831).

Gómez, Andres. *Seroterapia en la lepra*, Tesis para el doctorado en medicina y cirugía (Bogotá: Imprenta Nacional, 1897).

Gómez, Heráclito. “Observaciones sobre la lepra griega, sus causas, y comarcas donde es más frecuente”, *Anales de la Academia Nacional de Medicina*, Tomo I (Bogotá: Imprenta de la Luz, 1893): 84-87.

Gómez Ochoa, Juan Pablo. *Seroterapia Carrasquilla en la lepra. Resultados Obtenidos en Bogotá*. Tesis para el doctorado en medicina y cirugía (Bogtá, Imprenta Nacional, 1897).

Gutiérrez Arango, Daniel. *Los micro-organismos de la fiebre amarilla*, Tesis para el doctorado en Medicina y Cirugía (Bogotá: Imprenta El Telegrama, 1888).

Humboldt, Alexandre and Aime de Bonpland. *Essai sur la Gégraphie des plantes; accompagné d'un tableau physique des régions équinoxiales. Fondé sur des mesures exécutés, depuis le dixième degré de latitude australe, pendant les annés 1799, 1800, 1801, 1802 et 1803* (Paris: Levraut, Schoell et Compagnie, 1805).

JCH (Junta Central de Higiene) "Sesión del día 23 de mayo de 1887", *Junta Central de Higiene* (Bogotá: Casa Editorial de M. Rivas y Cía, Director, 1887): 29-33.

---- "Sesión del día 30 de mayo de 1887", *Junta Central de Higiene* (Bogotá, Casa Editorial de M. Rivas y Cía, Director, 1887): 33-43.

Levy, Michel. *Traité D'Hygiène Publique et Privée* T. I. (Paris:, J.-B. Baillière, Second edn., 1850).

Merizalde, José Félix. *Epítome de los elementos de higiene o de la influencia de las cosas físicas i morales sobre el hombre i de los medios de conservar la salud. Extractados de Estevan Tourtelle i traducidos al castellano, i añadidos con otras observaciones nuevas por José Félix Merizalde* (Bogotá: Imprenta de Pedro Cubides, 1828).

---- *Elementos de patologia general* (Bogotá: J. N. Barros, 1831), 165.

Olaya Laverde, Jesús. "Segundo informe del director de los trabajos científicos del Instituto de Seroterapia aplicada a la lepra", *Revista Médica de Santander*, 5 y 6, febrero, 1897: 10-35.

Otero, Luis Fernando. "Sociedad de ciencias médicas de Santander", *Revista Médica de Santander*, 5 y 6, febrero, 1897: 3-5.

Rodríguez N., Rubén. *Apuntes sobre la etiologia del tifo epidemico de 1889* (Bogotá: Imprenta de la Luz, 1890)

Rondon, Juan N. *Contribución al estudio de la etiología de la lepra griega*. Tesis para el doctorado en medicina y cirugia (Bogotá: Imprenta Nacional, 1897).

Ruiz, José María. *De la elefantiaisis de los griegos*, Tesis para la revalidación del grado de José María Ruiz, doctor en medicina, presentada al Consejo de la

Escuela de Medicina el dia 20, i sostenida el día 25 de febrero de 1867 (Bogotá: Imprenta de Gaitán, 1867).

Tourtelle, Etienne. *Elemens d'hygiene: ou, de l'influence des choses physiques et morales sur l'homme, et des moyens de conserver la sante*, 2v (Paris: Raemont et Fils, 1815).

Vargas, Jorge. *Programa para la enseñanza de la hijiene en las universidades de la República, formado por el Dr. Jorge Vargas, i aprobado por la dirección jeneral de instrucción pública* (Bogotá: 1845), 1-22.

Vargas Reyes, Antonio. *Trabajos Científicos del eminente médico granadino Dr. Antonio Vargas Reyes recopilados en obsequio de la humanidad doliente i de la juventud estudiosa de Colombia por Próspero Pereira Gamba* (Bogotá: Imprenta de la Nación, 1859).

---- *Trabajos Científicos del Doctor Antonio Vargas Reyes recopilados en obsequio de la humanidad doliente i de la juventud estudiosa de Colombia*, Tomo Segundo (Bogotá: Imprenta de la Nación, 1862), 37-54.

Uribe Angel, Manuel. "Profilaxia de la lepra", *Anales de la Academia Nacional de Medicina*, Tomo I (Bogotá: Imprenta de la Luz, 1893): 70-79.

Vargas Reyes, Antonio. "Informe al secretario de gobierno sobre la elefanteasis", *Trabajos Científicos del Doctor Antonio Vargas Reyes recopilados en obsequio de la humanidad doliente i de la juventud estudiosa de Colombia*, Tomo Segundo (Bogotá: Imprenta de la Nación, 1862): 55-61.

Zea Uribe, Luis. *El bacilo de Eberth y serodiagnóstico en la fiebre tifoidea*. Tesis para el doctorado en medicina y cirugía (Bogotá, Imprenta Nacional, 1898).

Secondary sources

Ackernecht, Erwin H. "Anticontagionism between 1821 and 1867," *Bulletin of the History of Medicine*, 22 (1948): 562–593.

Appel, John Wilton. *Francisco José de Caldas: A Scientist at Work in Nueva Granada, Transactions of the American Philosophical Society,* New Ser., 84, 5 (1994): 1-154.

Barry Barnes, “On the Conventional Character of Knowledge and Cognition”, *Philosophy of the Social Sciences*, 11, 3 (1981): 309.

Barnes, Barry, David Bloor and John Henry, *Scientific Knowledge. A Sociological Analysis* (London: Althlone, 1996).

Beldarraín, Enrique. “Evolución Histórica de la Geografía Médica en Cuba”, *Ilé. Anuario de Ecología, Cultura y Sociedad*, 3, 3 (2003): 93-110.

Benchimol, Jaime Larry, *Dos micróbios aos mosquitos. Febre amarela e a revolução pasteuriana no Brasil* (Rio de Janeiro: Editora Fiocruz, 1999).

Bulloch, William. *The History of Bacteriology* (London: Oxford University Press, 1960 [1938]).

Bynum, W.F. and Roy Porter, *Companion Encyclopedia of the History of Medicine*, Vol. 1 (London/New York: Routledge, 1997): 309-334.

Cáceres, Humberto y Zoilo Cuéllar-Montoya. *Academia Nacional de Medicina de Colombia. Sus Miembros* (Bogotá, Academia Nacional de Medicina, 1998).

Carter, K. Codell. *The Rise of Causal Concepts of Disease* (Aldershot: Ashgate, 2003).

Coleman, William. *Yellow Fever in the North. The Methods of Early Epidemiology* (Madison: The University of Wisconsin Press, 1987).

Caponi, Sandra. “On acclimatization: Boudin and medical geography”, *Historia, Ciências Saúde-Manguinhos* (Rio de Janeiro) 4, 1, en.-mar (2007): 13-30.

Crozier, Ivan “Social Construction in a Cold Climate”. Discussion Point. *The Society for the Social History of Medicine*, 13, 3 (2000): 535-546.

Cueto, Marcos. “Nationalism, Carrión’s Disease and Medical Geography in the Peruvian Andes”, *History and Philosophy of the Life Sciences*, 25 (2003): 319-335.

Cunningham, Andrew. “Transforming Plague. The Laboratory and the Identity of Infectious Disease” in Andrew Cunningham and Perry Williams (eds.). *The Laboratory Revolution in Medicine* (Cambridge: Cambridge University Press, 1992): 209-244.

Delaporte, François. *The History of Yellow Fever. An Essay on the Birth of Tropical Medicine* (Cambridge, MA: MIT, 1991).

Díaz y de Ovando, Clementina. *El doctor Manuel Carmona y Valle y la fiebre amarilla son noticia periodística, (1881-1886)* (México: Facultad de Medicina/UNAM, 1993).

Edmond, Rod. *Leprosy and Empire. A Medical and Cultural History* (Cambridge: Cambridge University Press, 2006).

Foster, W.A. *A History of Medical Bacteriology and Immunology* (London: William Heinemann Medical Books), 1970.

Fredj, Claire. "Cerner une épidémie: le travail des médecins militaries sur la fièvre jaune au Mexique en 1862 et 1867", *Genèses*, 38 (2000): 79-104.

García, Claudia Mónica. *Las 'fiebres del Magdalena': medicina y sociedad en la construcción de una noción médica colombiana, 1859-1886* (Bogotá: Universidad Nacional de Colombia, 2006).

--- "Las 'fiebres del Magdalena': medicina y sociedad en la construcción de una noción médica colombiana, 1859-1886", *Historia, Ciências, Saúde-Manguinhos* (Rio de Janeiro) 14, 1, ene.-mar (2007): 63-89.

Geison, Gerald. "Pasteur" in Charles Coulston Gillispie (ed.), *Dictionary of Scientific Biography*, Vol. 9-10, New York, Charles Scribner's Sons, 1981: 350-416.

Guzmán, Maria del Pilar. "La alopatía y la homeopatía en el siglo XIX: conflicto entre dos prácticas", *Anuario Colombiano de Historia Social y de la Cultura*, 22 (1995): 59-73.

Humphreys, Margaret. "A Stranger to Our Camps: Typhus in American History", *Bulletin for the History of Medicine*, 88 (2006): 269-290.

---- *Yellow Fever and the South* (New Brunswick/New Jersey: Rutgers University Press, 1992).

Hamlin, Christopher. "Predisposing Causes and Public Health in Early Nineteenth-Century Medical Thought", *The Society for the History of Medicine*, 1992: 43-70.

Hannaway, Caroline. "Environment and Miasmata", W.F. Bynum and Roy Porter, *Companinon Encyclopedia of the History of Medicine* (London and New York: Routledge, 1993): 293-308.

Harrison, Mark. "'The Tender Frame of Man': Disease, Climate, and Racial Difference in India and the West Indies, 1760-1860", *Bulletin of the History of Medicine*, 70, 1 (1996): 68-93.

Ibáñez, Pedro María. *Memorias para la historia de la medicina en Santafe de Bogotá* (Bogotá: Imprenta Nacional, 1968 [First edn. 1884]).

Jacyna, L.S.,"Au Lit des Malades: A.F. Chomel's clinic at The Charité, 1828-9", *Medical History*, 33 (1989): 420-449.

Latour, Bruno. *The Pasteurization of France* (Cambridge/Massachusetts/London: Harvard University Press, 1988).

---- "Pasteur on Lactic Acid Yeast: A Partial Semiotic Analysis, *Configurations*, 1, 1 (1993): 129-130.

Livingston, David N. "Human Acclimatization: Perspectives on a Contested Field of Inquiry in Science, Medicine, and Geography", *History of Science*, 25 (1987): 359-394.

Löwy, Ilana. *La fièvre jaune au Brésil entre science et politique* (Paris: Editions des archives contemporaines, 2001).

Márquez Valderrama, Jorge. *Ciudad, miasmas y microbios. La irrupción de la ciencia pasteuriana en Antioquia* (Medellín: Universidad de Antioquia, 2005).

Maulitz, Rusell C. "Physician versus Bacteriologist': The Ideology of Science in Clinical Medicine" in Morris J. Vogel and Charles E. Rosenberg, *The Therapeutic Revolution* (Pennsylvania: University of Pennsylvania Press, 1979): 91-107.

McCarthy, Michael P. *Typhoid and the politics of public health in 19th-century Philadelphia* (Philadelphia: American Philosophical Society, 1987).

Mendelsohn, Andrew. "'Like All That Lives': Biology, Medicine and Bacteria in the Age of Pasteur and Koch", *Hist. Phil. Life Sci.*, 24, 2002: 3-36.

---- *Cultures of Bacteriology: Formation and Transformation of a Science in France and Germany, 1870-1914*, PhD Dissertation, Princeton University, June, 1996.

Moulin, Anne Marie. "Bacteriological Research and Medical Practice in and out of the Pastorian School" in Ann La Berge and Mordechai Feingold, *French Medical Culture in the Nineteenth Century* (Amsterdam/Atlanta: GA, 1994): 327-349.

Montaña, Eliseo. *El doctor Juan de Dios Carrasquilla*, (Imprenta y litografía de J. Casis, 1919).

Montoya y Flórez J.B. *Contribución al estudio de la lepra en Colombia* (Medellín: Imprenta Editorial, 1910).

Moulin, Anne Marie. “Bacteriological Research and Medical Practice in and out of the Pastorian School” in Ann La Berge and Mordechai Feingold, *French Medical Culture in the Nineteenth Century* (Amsterdam/Atlanta: GA, 1994): 327-349.

---- *Le dernier langage de la médicine: histoire de l’immunologie de Pasteur au sida* (Paris: Presses universitaires de France, 1991).

Nutton, Vivian. “Humoralism”, W.F. Bynum and Roy Porter, *Companinon Encyclopedia of the History of Medicine* (London and New York: Routledge, 1993): 281-291.

Obregón, Diana. *Batallas contra la lepra: Estado, Medicina y Ciencia en Colombia* (Medellín: Eafit, 2002).

---- “Sobre epidemias, endemias y epizootias: algunos aspectos del desarrollo de la bacteriología en Colombia”, *Biomédica*, 18, 2 (1998): 110-121.

---- “The Social Construction of Leprosy in Colombia, 1884-1939”, *Science, Technology and Society*, 1, 1 (1996): 1-23.

Ocampo, José Antonio. *Colombia y la Economía Mundial. 1830-1910,* **(**Bogotá: Tercer Mundo, 1998).

Osborne, Michael. “The Geographical Imperative in Nineteenth Century French Medicine” in Nicolaas A. Rupke, “Medical Geography in Historical Perspective”, *Medical History*, Supplement No. 20, (London: The Wellcome Trust Centre For the History of Medicine at UCL, 2000): 31-50.

---- “‘Resurrecting Hippocrates”: Hygienic Sciences and the French Scientific Expeditions to Egypt, Morea and Algeria”, in David Arnold (ed.), *Warm Climates and Western Medicine : The Emergence of Tropical Medicine, 1500-1900* (Amsterdam/Atlanta: Rodopi, 1996): 80-95.

---- “French Military Epidemiology and the Limits of the Laboratory”, Andrew Cunningham and Perry Williams (ed.), *The Laboratory Revolution* (Cambridge: Cambridge University Press, 1992): 189-208.

Palacios, Marco. *Coffee in Colombia, 1850-1970* (Cambridge: Cambridge University Press, 1980).

Patterson, K. David. “Typhus and its Control in Russia, 1870-1940”, *Medical History*, 37 (1993): 363-381.

Pavía, Lázaro. *Reseña biográfica de los doctores más notables de la República Mexicana* (México: Imp. Eduardo Dublan, 1897).

Pelling, Margaret. "Contagion, Germ Theory/Specificity" in W.F. Bynum and Roy Porter, *Companion Encyclopedia of the History of Medicine*, Vol. 1, (London/New York: Routledge, 1997): 309-334.

Pérez López, Jaime. *Colombia-Venezuela. Economía-Política-Sociedad. Siglos XIX y XX* (Cúcuta: Offset La Opinión, 2003).

Postel-Vinay, Nicolas and Anne-Marie Moulin. "La syphilisation: ou les rapports utopiques du chancre et de la lancette", *La Revue du Practicien* (Paris), 44 (1994): 1727-1729.

Quevedo, Emilio and Camilo Duque. *Historia de la Cátedra de Medicina, 1653-1685* (Bogotá: Universidad El Rosario, 2002).

Quevedo, Emilio, Juan C. Eslava, Claudia M. García et al, *Café y gusanos, mosquitos y petróleo. La transición de la Higiene a la Medicina Tropical y la Salud Pública en Colombia, 1873-1953* (Bogotá: Instituto de Salud Pública-Universidad Nacional de Colombia, 2004).

Robertson, Jo. "Leprosy and the elusive *M. leprae*: colonial and Imperial medical exchanges in the nineteenth century", *Historia, Ciências Saúde-Manguinhos*, vol. 10 (supplement 1), 2003: 13-40.

Rupke, Nicolaas A. (ed.) 'Medical Geography in Historical Perspective', *Medical History*, Supplement No. 20 (London: The Wellcome Trust Centre for the History of Medicine at UCL, 2000).

---- "Humboldtian Medicine", *Medical History*, 40 (1996): 293-310.

Safford, Frank. "Race, Integration, and Progress: Elite Attitudes and the Indian in Colombia, 1750-1870", *Hispanic American Historical Review*, 71, 1 (1991), 1-33.

Safford, Frank and Marco Palacios. *Colombia. Fragmented Land, Divided Society* (New York/Cambridge: Cambridge University Press, 2002).

Sandoval, Yesid and Camilo Echandia, "La historia de la quina desde una perspective regional", *Anuario Colombiano de Historia Social y de la Cultura, 1850-1882*", 13-14 (1986): 153-187.

Sierra, Luis Fernando. *El tabaco en la economía colombiana* (Bogotá: Universidad Nacional, 1971).

Silva, Renan. *Los ilustrados de la Nueva Granada, 1760-1808* (Medellin: EAFIT, 2002).

Shapin, Steven. "History of Science and its Sociological Reconstructions", *History of Science*, 20 (1982): 164-166.

Smith, Dale C. "Gerhard's distinction between typhoid and typhus and its reception in America, 1833-1860", *Bulletin of the History of Medicine*, 54, 3 (1980): 368-385.

---- "The Rise and Fall of Typhomalarial Fever", *Journal of the History of Medicine and Allied Sciences*, 37 (1982): 182-220; 287-321.

Smith, Michael M. "The 'Real Expedicion Maritima de la Vacuna' in New Spain and Guatemala", *Transactions of the American Philosophical Society*, New Series, 64, 1 (1974): 1-74.

Stevenson, Lloyd G. "A Pox of the Ileum: Typhoid Fever among the Exantemanta". *Bulletin of the History of Medicine*, 51 (1977): 496-505.

Tomes, Nancy J. and John Harley Warner. "Introduction to Special Issue on Rethinking the Reception of Germ Theory of Disease: Comparative Perspectives", *Journal of the History of Medicine*, 52 (1997): 7-16.

Tomes, Nancy J. and John Harley Warner, "Introduction to Special Issue on Rethinking the Reception of Germ Theory of Disease: Comparative Perspectives", *Journal of the History of Medicine*, 52, January (1997): 7-16.

Uribe, Agustin. "Condiciones hijiénicas de Bogotá", *Diario de Cundinamarca*, Año IV, 999, 9 de abril (1873): 572.

Valencius, Conevery Bolton. *The Health of the Country: How American Settlers Understood Themselves and Their Land* (New York: Basic Books, 2002).

Villegas, Benjamín. (ed.), *El agua en la historia de Bogotá, 1538-1937* (Bogotá: Villegas Editores, 2003).

Warner, John Harley. "The Selective Transport of Medical Knowledge: Antebellum American Physicians and Parisian Medical Therapeutics", *Bulletin for the History of Medicine* 59 (1985): 213-231.

Warner, Margaret. “Hunting the yellow fever germ: the principle and practice of etiological proof in late nineteenth-century America”, *Bulletin of the History of Medicine*, 59 (1985): 361-382.

Wilson, Adrian “On the History of Disease Concepts: the Case of Pleurisy”, *History of Science*, 38, (2000), 271-318.

Wilson, Leonard G. “Fevers and Science in Early Nineteenth Century Medicine”, *Journal of the History of Medicine and Allied Sciences*, 33 (1978): 386-407.

Worboys, Michael. *Spreading Germs: Disease Theory and Medical Practice in Britain, 1865-1900* (United Kingdom: Cambridge University Press, 2000).

Wright, Peter and Andrew Treacher (ed.). *The problem of Medical Knowledge*: *Examining in Social Construction of Medicine* (Edinburgh, Edinburgh University Press, 1982).

Zambrano, Fabio. “El golpe de Melo de 1854”, *Las guerras civiles desde 1830 y su proyección en el siglo XIX* (Bogotá: Museo Nacional de Colombia, 1998).