“The First Murmurings of a Discourse”: Initial Theory Invention at the Beginning of the AIDS Epidemic

by

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Abstract

In 1981, at the first recognition of the illness/es that would eventually be named "AIDS," clinicians took what knowledge was available at hand to create several hypotheses as to the pathogenesis and etiology of the then mystery illness.

The first major hypothesis, proposed by Michael Gottlieb and colleagues in December 1981, centered on the perceived prevalence of cytomegalovirus (CMV) within the "homosexual population." The clinicians reasoned that the reactivation of the latent CMV virus coupled with constant re-exposure to the CMV pathogen gradually destroyed the cellular immune system of the host. This proposed cause quickly proved to be untenable. Subsequent explanations simultaneously refuted the CMV/overload hypothesis, yet at the same time altered the basic logic to propose other forms of withering or overload.

Using close textual analysis this thesis traces the invention of these initial hypotheses ("first murmurings") to see how they were interrelated and how, despite their differences, they entail a coherent logic. This reading utilizes Michel Foucault's archeological method in conjunction with Derrida's deconstruction of invention, and aims to identifying what Foucault calls a 'rule of formation'.

Keywords: HIV/AIDS, Theory, Biomedicine, Foucault, History, Epistemology

Dedication

This work is dedicated to my father. Only through his encouragement did I decide to go back to school and finish my undergraduate degree. Otherwise, this thesis would never have existed.

He will be missed.

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I would like to thank my supervisor Cindy Patton. It is in those reading groups that we held at the lab years ago that I truly learned how to read theory and philosophy and it is only through her training that I learned to focus my ideas and write at an academic level. She is the best supervisor that anyone could ask for.

Table of Contents

Chapter 1. Introduction

1.1. The Mystery Illness

1.1.1. The First Cases

As early as 1979, unusual cases of pneumocystis carinii pneumonia (PCP) started to appear in gay men from San Francisco and New York, a strange and unexpected occurrence, since this rare and potentially deadly type of pneumonia had previously been confined to those with clinically[1] induced immunosuppression due to chemotherapy treatments or those undergoing renal transplant procedures. Additionally, this pneumonia was usually highly treatable with one or two courses of antibiotics (like TMP-SMZ). Beyond the handful of cases where rare genetic causes of congenital immune system malfunction, it was unheard of for PCP to occur outside of the clinical setting. The, then-existing, knowledge of PCP made it hard for clinicians to imagine why it was starting to occur in young and previously healthy men.

A second perplexing epidemic was occurring in other parts of the U.S. – increase in a rare type of skin cancer called Kaposi's sarcoma (KS) that had previously been almost exclusively appearing in either the elderly or as an immune system side effect among individuals undergoing clinical immunosuppression. As with PCP, KS was usually slow-acting, treatable and seldom deadly. The new cases among young men were both aggressive and impervious to treatment and, along with the PCP, KS was causing these men to die.

[1] The term 'clinically' (as in 'clinically acquired') usually designates conditions or illnesses that are an outcome of treatment or of medication and can include conditions such as p. carinii pneumonia, side effects of medication, or other conditions which a person develops as a result of ongoing treatment. Diseases of this type are often said to have an iatrogenic cause. In contrast, the term 'community' (as in 'community acquired') usually designates conditions that a person develops outside of the hospital setting, out in the wider world.

Aside from PCP and KS, these men also developed a variety of infections with different low grade pathogens such as herpes simplex virus infection, Epstein-Barr infection, cytomegalovirus infection and candidiasis among others. The patients also suffered from a growing list of symptoms and conditions such as severe and unrelenting fever, lymphadenopathy, leukopenia, weight loss, and anemia[2].

Many of these patients quickly deteriorated and died in what seemed to be an extremely rapidly progressing condition. Even patients who at first responded well to treatment, and in whom the fever and pneumonia seemed to initially subside, found themselves back at the hospital later on with the same set of infections and symptoms. Out of the first 159 cases of this illness, the mortality rate was nearly 40 percent. Only in July 1982, at a meeting in Washington, DC, was the name AIDS (acquired immunodeficiency syndrome) officially 'coined' to designate this new illness. Until a cause was identified, the newly named syndrome remained a mystery illness: doctors were slow to understand that they were seeing patients at the very end of a largely asymptomatic immune system decline that had likely happened over five or ten years. The medical community was unaccustomed to cases of this extreme uncertainty and newness– there had been no unsolved medical mysteries of this magnitude for a very long time. Even the initial mystery surrounding the outbreak of Legionnaire's disease in 1976 did not last long. The pathology and epidemiology of the disease were arguably easier to establish since the cases were not symptomatically diverse and the cases were geographically isolated to the 221 convention attendees.

The situation in this case was different. In its history, the development of modern biomedicine (the clinic), which began with the establishment of early hospitals in Europe, contended with diseases that already existed. During the 19th century, what was at issue was creating a better understanding and standardization of diseases that were formerly folk knowledge or old apothecary knowledge of humors and bile (see the more expanded discussion of this in

[2] What we understand now is that one of the major mechanisms by which the HIV virus destroys the immune system is by infecting the $CD4^+$ white blood cells (helper T cells) which are crucial for the regulation of the cellular immunity. Antibody dependent cell mediated cytotoxicity ($CD8^+$ suppressor cells) then recognizes these infected $CD4^+$ cells as a threat and begin to eliminate them thereby resulting in a very low number of $CD4^+$ cells and thereby an almost non-existent cellular immunity. Note, that these are theories and knowledge of the disease that only appeared later on and at the point I am studying none of this knowledge existed besides knowing the disproportion of $CD4^+$ to $CD8^+$ cells found in the patients.

Chapter 2 or refer to the source material, *Birth of a Clinic*, by Michel Foucault). The point, however, is that many of these diseases already existed and were part of the reality of life for most Europeans since time immemorial. What was different with AIDS was an urgency to say something about this mystery illness, to establish some – any – sort of etiology and pathogenesis to this apparently new condition. I would similarly argue that this presented a very unique case not just for the medical community but for the scientific enterprise[3] itself.

One of the prevailing narratives of scientific progress tells a story of science where consistently 'good' science leads to an ever evolving (positive) understanding of our world and how it functions. This narrative of scientific progress presents a sort of linear upward movement of scientific progress ad infinitum or at least towards some sort of infinitely far away idea of complete knowledge. This narrative helps science separate itself from its own historical past and extend itself past the boundaries of human civilization[4] as something timeless and transcendent.

Similarly, such a narrative is a representation of science where science is not something that can be rushed or forced. The process of scientific and technological advancement is a gradual one and scientific advancement and discovery is something that will be made in due time. Even if we look at some of the most rapid advancements of technology in the 20th century like the 'space race' between the Soviet Union and the United States, we can argue that the state of competition that developed between the two opponents may have forced the development of better space travel technologies, but the idea of space travel was already something that existed (as early as the turn of the century, in cinema) and wasn't itself something that was created by the competition. Similarly, the race to develop nuclear weapons was something that began only once

[3] I tend to use the term 'scientific enterprise' instead of just 'science' because I think referring to it as just 'science' is deluded. Science is not a thing, it is not an ostensive object and so one cannot refer to it as if it was a thing that materially exists and can be touched or felt. Science is a process, a methodology, a set of standardized procedures that yield an expected result. A term like 'scientific enterprise', however, can encompass those things like the legions of scientists, researchers, doctors, laboratories and large hadron colliders which use or help with the scientific process.

[4] A child in elementary school does not learn of Pythagoras himself or of his philosophy when learning the basics of the Pythagorean Theorem. Neither is the high school student forced to read *Philosophiae Naturalis Principa Mathematica* when learning Newton's laws of motion or, indeed, forced to read about Newton's particle theory of light. Scientific facts are presented as timeless and devoid of context – as simply there as if they have been there forever. This creates a special relation to this type of knowledge in humans from a very early age.

the physics allowing for an understanding of mass/ energy equivalence were established and those discoveries of general relativity were made well before the outbreak of conflict in Europe.

This thesis considers what happens when the scientific enterprise is forced to make a discovery, forced to say something – anything – in the face of a rising death toll and an illness that is completely unprecedented. Where do researchers turn and how do they "think" when the natural flow of scientific progress is no longer viable because of the pressure to figure out a puzzle? A later section reviews debates about how science progresses, in particular, how to conceptualize rapid intellectual change, or change that occurs after foundational hypotheses are proven incorrect. My overarching interest is in moments when we witness an 'accelerated' effort of the scientific community to make a discovery. The initial research on "AIDS" is a well-documented case of scientific transformation and rapid (though often unsuccessful) scientific application.

In the following chapters, I trace the thought processes by which doctors and clinicians generated the first hypotheses as to the etiology and pathogenesis of this mystery illness. I investigate the first hypotheses that were generated and published in well-known medical journals within the first eleven months from the initial publication about the first five cases in the *Morbidity and Mortality Weekly Reports*. I hope to show how a handful of interested researchers took up and then quickly discarded the initial hypothesis on scientific grounds. As is well-documented, activists also critiqued the early hypothesis on political grounds, and were happy when apparently better science prevailed. However, the combination of social ideas that allowed the emergence of the quickly disputed hypothesis did not entirely disappear with the initial hypothesis. I hope to show exactly how, at the level of rhetorical practices that reflect an intellectual style, a trace of the ideas that the activists objected to remained embedded in the scientific enterprise's logic.

In the remainder of this chapter, I will briefly describe the role of sociology and social theory in the analysis of science, focusing primarily on the approach taken by Bruno Latour and Steve Woolgar in their seminal work *Laboratory Life*, and then connect my project to the important early works that emerged in the social study of HIV/AIDS.

1.1.2. Critiques of Sociology in Science

Latour's and Woolgar's *Laboratory Life* is often cited as the book that established the field of science and technology studies (STS) and transformed the study of science from a mere 'specialization' of professors and researchers from several traditional disciplines into an interdisciplinary or transdisciplinary enterprise that could study the kinds of scientific developments that were occurring in the late 20th century. Rather than continuing the efforts to discuss an abstract "science" on a grand scale, Latour and Woolgar very carefully observed the everyday and often mundane details of scientific research at a laboratory. Rather than attempting to put into some sort of structure a range of scientific discoveries produced over hundreds of years, Latour and Woolgar looked at the ceremony-like nature of grant writing, the intra-laboratory employee hierarchies, and the creation of post-hoc narratives of discovery out of events whose sequence and transparency were far from clear. The focus on inter-relation among ideas, substances, techniques, and people laid the groundwork for Latour's actor-network theory, now a widely used frameworks in science and technology studies.

At the very beginning of their book, in a section named "The Social and the Scientific: The Observer's Dilemma", the two authors reflect on some of the difficulties and assumptions that face social researchers who hope to study science, which are similar to some of the problems I face in my own research on the initial hypotheses for the cause of AIDS. In the following section, I would like to address three major points related to the constraints that the social scientist faces when studying science.

First, they gesture to the world outside of the laboratory and note, in a typically Latourian tongue-and-cheek fashion, that "[p]ut simply, this means that there is no point in doing sociology of science unless one can clearly identify the presence of a politician breathing down the necks of the working scientists" (Latour and Woolgar 1986, 23). Latour and Woolgar point here to an established hierarchy between the technical and social aspects of the scientific enterprise where social researchers are seemingly confined to and therefore concentrate on the 'social' rather than the 'technical' aspects of science such as the impact of scientific knowledge on society or the effects of new technology on the social sphere. This 'social' is seen as the closest that sociology can validly get to studying 'true' science. In a sense, the actual work of the scientific enterprise is imagined as belonging to a sphere or mode of existence that is completely separate and detached from the social.

Such a structure and the ultimately high barriers of entry into the scientific enterprise which propagates the naturalness of scientific progress makes it doubly difficult to embark on any investigation of 'true' science which is not seen as heretical and which does not make the social researcher look like a quack, lunatic, or luddite. It is as simple as this: according to the current position of science, the only people who have the authority to study science are scientists themselves but the problem is that scientists themselves are not in the business of studying the social or historical. The reality that many citizens and not a few professional intellectuals accept scientists' accounts of progress as "accurate," reveals an acquiescence that would not be so fast if the subject was religion – we would not assume the accuracy of a historical account of a particular religion written by the clergy of that religion. I would argue, first, that even the 'technical' aspects are 'social' and are a product of human communication. The technical does not exist in a separate sphere that is shielded from the social and is open to the same type of investigation and analysis.

Almost in an effort to balance out this point so as not to seem that they are advocating for the other logical extreme of only studying the technical aspects of science, Latour and Woolgar make a reversal in the relation between the 'social' and the 'technical' in which social scientists focus primarily on the 'social' aspects of science and discuss what happens when there is too much focus on the 'technical' sphere. Here the authors point to studies of science which fully embraced the analysis of the 'technical' aspect of science but in doing so have produced results or findings that are sparsely generalizable and therefore not open to any sort of a theoretical analysis. The folly of such research is that it dives too deep into the technical aspects of science where it similarly dehumanizes them and is then not able to connect them back to the social and back to some theoretical perspective. While such research might present impressive technical descriptions, without this connection to the social it cannot be of much worth to the *social* study of science. Indeed, some balance between the social aspects and the technical aspects must be reached.

Lastly, there is the third major assumption that is made about what social scientists are allowed to study or are perceived to study. Here, we find a very narrow alternative to the study of social aspects whereby social researchers have the option to undertake a 'sociology of error'. Here the objects of study are those things that have long been discarded by 'good' science as wrong or archaic. The usual topics include phrenology and eugenics but also include even older

scientific ideas like the phlogiston theory of fire or the particle theory of light. The prevailing narrative is that this 'bad' science represents momentary lapses in reason and good judgment in the overall story of scientific progress. And while the study of such 'mistakes' does have its merits, such an approach ultimately covers ground that can be argued to have no hold on contemporary currents of human thought and civilization that have 'moved on' from these mistakes.

It might seem that by looking at these initial hypotheses as to the nature of AIDS which were ultimately proven wrong and replaced, my project inherits the problems of a sociology of error. Instead, I challenge this approach by looking in considerable detail at a moment of invention when, contrary to the assumptions of a neo-realist sociology of error approach, there was no larger scientific paradigm shift that can separate the early hypotheses from the discovery of the HIV virus – these events occurred in such close temporal proximity as to be almost simultaneous. Indeed, this is how this two year time period starting from the first MMWR publication describing an outbreak of PCP, then moving to the first hypothesis, and then to the identification of HTLV/HIV, has usually been presented. No large reconceptualization of immunology, biology, or genetics separates the events such that we could argue that had the first medical doctors been aware of subsequent facts, they would have seen the disease differently. There was nothing scientifically 'wrong' about how those hypotheses were formulated; they were not formulated during a lapse in reason or judgment within the scientific enterprise but were formulated out of a very common, ordinary, and everyday science of the day.

Now I would like to turn the attention to the topic at hand and narrow down the investigation from this abstract space of 'the study of science' to some of the more nuanced and presiding matters of the social study of HIV/AIDS. The aim is that the next section, combined with the previous discussion will make it clear what it is that I exactly intend to do in the following chapters of this thesis.

1.2. The Social Study of AIDS

1.2.1. An Overview of Literature

There is by now an extensive body of work on the social and cultural aspects of HIV/AIDS, even that which is found more strictly within STS is substantial. I leave aside the more conventional work that views HIV/AIDS as a more or less empirical reality and then describes, critiques or proposes solutions, health care delivery improvement, awareness campaigns, population health or public policy initiatives to name only a few. For my purposes, the most directly relevant works are those that link disease conceptualization with social and cultural production. I would like to begin this brief overview by first looking at one end of the spectrum – the person living with AIDS (PLWA) and the activist (often one and the same) – and then transitioning to the other side of the spectrum – the clinic and biomedicine.

The emphasis in this body of literature pertaining to the PLWA and activism is on the idea that the scientific discourse surrounding AIDS developed as a distinctly public one. Biomedical knowledge surrounding AIDS was not based on the work of a few 'heroic' credentialed specialists like Montagnier, Gallo or Fauci whose discoveries made up and completed the field, but to a great degree, involved the contribution of knowledge from laypeople in various fields of society (Epstein 1996, 5). Patton notes that public discourse surrounding AIDS quickly developed into a highly sophisticated one to the point where, "the gay press reads like a medical journal and gay readers may perceive themselves as largely able to grasp the technical aspects of HIV" (1990, 54). Activists in groups such as ACT UP became, what Patton calls, "autodidactic medical experts" (Ibid, 54).

Similarly, Treichler notes the paramount effect that such activists had on the structure of scientific research. In about 1984, when institutional medicine began to launch studies and experiments into different drugs and treatments for what was already then known to be HIV, some clinical researchers recognized that some people with AIDS were "too well informed and technologically expert" to participate in randomized clinical trials (Treichler 1999, 287). Such autodidactic medical experts, many of whom were also patients, reversed and stifled the standard doctor-patient relationship where power, operating as specialized knowledge, was usually unidirectional and flowed from the doctor to the patient (Epstein 1996, 206-207).

These changes in the individual consciousness of AIDS patients developed more profoundly into what can be called 'treatment activism'. Unofficially, such treatment activism involved underground buyer's clubs and guerrilla clinics (Treichler 1999, 287; Epstein 1996, 223-224). Officially, it included organizations such as New York's ACT UP, Project Inform of San Francisco and the Community Research Initiative (CRI) of New York. These organizations, together with more than 30 other smaller organizations, formed what was known as the ACT NOW Network (Patton 1990, 139 (Note 26)). Perhaps the best way to describe the directive of this treatment activism is to use the words of John James – the founder of the AIDS Treatment News publication. He states that, "So far, community-based AIDS organizations have been uninvolved in treatment issues… With independent information and analysis, we can bring specific pressure to bear to get experimental treatments handled properly… so far, there has been little pressure because we have relied on experts to interpret for us what is going on" (Epstein 1996, 195). What James saw, and he was far from the only one, was the need for persons with AIDS and those otherwise involved in AIDS activism to become involved in the knowledge creation process. Treichler would add that one essential element of organized treatment activism is "a vision of a power structure that calls for unleashing the power and knowledge of resistance forces; expertise about technology and science… biomedical research… [and] self-education" (Treichler 1999, 291).

While many of these developments were important it is also worthwhile to note that the spheres of knowledge produced by each of these groups (activists on one side and biomedicine on the other) were quite closed off. Patton (1990), for example, traces the discourse of the AIDS service industry which developed in the 1980's. This analysis describes how strict distinctions of 'expert', 'volunteer', and 'victim' emerged early on in the epidemic and how each of these designations was responsible for production of very distinct spheres of knowledge whereby the particular and personal story of PLWAs were eventually turned into scientific, epidemiological knowledge (Patton 1990, 52). These personal narratives by themselves could not exist within the scientific sphere and thereby had to be transformed accordingly into a form that was permissible in biomedicine. Once transformed into this scientific form, the knowledge is then once again disseminated in an 'accessible' form back to the public including those members of the public who originally provided the knowledge. Patton notes this process to be a circulation of knowledge which re-establishes the dominance of science as the first voice among many when it comes to HIV/AIDS and to knowledge production in general (Patton 1990, 52-57).

Looking further into this control by science over the discourse of HIV/AIDS, Erni presents a very detailed analysis of the many contradictions that developed within institutionalized medicine in the area of treatment and cure. Looking at events such as the azidothymidine (AZT) controversy[5], Erni analyzes the careful balance that developed within biomedicine where on one hand doctors and clinicians often spoke of a 'cure' for HIV/AIDS and on the other hand the disease was often termed incurable – that the best anyone could hope for was extending the lives of those with the syndrome (Erni 1994, 12-13). Ultimately, Erni establishes that this insistence on the possibility of a cure is not simply a narrative established out of the particularities of the AIDS epidemic but is a central narrative within the sciences where a conclusion – such as a cure for a disease – is a necessary part of the story of scientific progress and is something that is constantly re-established even when contrarian voices state that the disease is incurable (Erni 1994, 126-136).

Epstein also considers the problem of self-contradiction within the scientific enterprise and notes a very important change in directive that occurred with the early ideas of etiology and pathogenesis of the illness. Similar to what I will term the cytomegalovirus hypothesis, Epstein notes that the immune overload hypothesis followed a line of biomedical reasoning distinctly different to what was established practice at the time. Epstein describes that the established form of understanding human illness relied on the idea of 'one illness, one cause" and that "the search for the cause of illness should focus primarily on microbes, very secondarily on lifestyle issues, and only incidentally on environmental causes related to the larger organization of society" (Epstein 1996, 57). Contrarily, the initial search for the etiology of the disease was based in the 'homosexual lifestyle'. He notes that "to the extent that doctors endorsed a multicausal lifestyle model, they were going against the prevailing medical currents" (Epstein 1996, 58).

[5] AZT was a cancer drug created in the 50's and then quickly 'mothballed'. After the discovery of the HIV virus, it was brought back into use again due to its properties as an antiretroviral drug. Large scale randomized control trials were started to test its efficacy against HIV. The trials were criticized by different activist groups as being a waste of time and that they should start issuing the drug to persons with AIDS as soon as possible. On another front, the drug was also found to be toxic in the dosages in which it was prescribed. Lastly, upon its approval as a form of treatment, WelcommeBurroghs, who held the patent, made the initial per patient price extremely prohibitive for the majority of patients ($10,000 per year).

1.2.2. Focusing the Study

These foundational and influential works start from a position of embeddedness within the "real world" where the discourse of AIDS and HIV were weighing heavily on individuals and communities that lay outside mainstream science and mainstream society, and thus, these writers were in something of a privileged "outsider" position to sceptically greet a set of actors/ agents, institutions, structures, statements, and objects of discourse. But at the same time, the writing in that moment was itself pressured by political necessity and the allegation that homophobia was hampering science. These writers all tried to disentangle precursor ideas about homosexuality from the emergent ideas within AIDS science, and Epstein in particular critiques the 'viral overload' hypothesis, but perhaps quickly fast forwards into the time period in which HIV has been discovered, somewhat falling prey to the problems of a sociology of error that places social attitudes rather than scientific backwardness as the cause of the mistaken idea. Epstein notes that the name 'acquired immunodeficiency syndrome' (AIDS) was chosen because of its neutrality compared to the previous name of 'gay-related immunodeficiency' (GRID) (Epstein 1996, 55). This change to neutrality itself points to a change in judgment. In turn, this change in judgment points to a change in concept and to a change in the very foundation of how this disease was understood. In this thesis, I reopen the conceptual space that lay one step before this foundational step to an HIV discourse, in order to treat the 'mystery illness' that was "before AIDS" and AIDS itself as different objects of discourse.

This is why I would like to focus on this preliminary time period before an attempt at neutrality had become necessary. In that space when there was only GRID (and arguably even including a time before the term 'GRID' was even established), there existed a different discourse; all the involved actors operated under different spheres of knowledge and a different stock of knowledge was available to them – particularly if we look at the knowledge which the doctors possessed. The findings that are produced by this early form or stage of science make the social and the political presuppositions that went into it all the more visible. This is not to say that this early science is corrupt or tainted. On the contrary, it is in this early scientific thought that we see the 'un-doctored' story. Once this early narrative gets coopted into the primary narrative of a victorious, unfettered and ceaseless scientific progress moving humankind towards some sort of ultimate emancipation, history is destroyed. In the process, the original meanings become obfuscated and, I will argue, subjugated.

I look exclusively at the initial published hypothesis of the cause of the mystery illness, and I want to consider this hypothesis-generation as a moment of invention on pressure. For this purpose I will employ the Foucauldian archeological framework, which I will discuss in more detail in the next chapter. This approach will help to me frame the different hypotheses as statements within a discourse conforming to certain rules of formation. Indeed, a large part of the analysis will be devoted to establishing the rule of formation that regulated the creation of these hypothesis-statements within this discourse.

However, the archeological framework is primarily used for the analysis of "things" whose truth effect is already established, that is, it seeks to dis-entangle and make strange worldviews that have been accepted as "real." It is by itself not a framework that can handle the analysis of things that are still only emerging – it is not as acutely focused on what Foucault himself calls the "first murmurings of a discourse." Before a discourse is established, a discourse must have a rule (or rules) which must be invented and then stabilized. To investigate how a rule of formation forms itself I draw on Derrida's notion of the deconstruction of an invention. In the next chapter, I discuss Foucault's and Derrida's respective ideas and then make the case for combining them into a coherent whole that splits some of the differences between two authors who are often seen as intellectual adversaries.

Chapter 2. Discourse, Invention

2.1. The Archeological Method

In this section, I intend to outline my particular usage of Foucault's writing. This will be split into several sections because there is a particular difference that I hope to show between the archeological method (which in itself is a particular theoretical and epistemological approach) and Foucault's empirical findings in *Birth of a Clinic*. There is also a particular way that each of these should be treated and appropriated into analysis – one would not use Foucault's particular conception of the statement and discourse the same way as his articulation of the clinical analogy – one is method, the other is finding. The former, we can say, is his theoretical labour, the latter, a description of the studied time period's knowledge. But to understand the difference, I will first try to provide the reader with an explanation of what the archeological method is and what it hopes to achieve; only then can we discuss, the empirical description and understand what is at stake within it. Then I will show why Foucault's notion of clinical analogy must be supplemented with the concept of invention, a concept I take from Jacques Derrida that helps fit the Foucauldian archeological project to the instance of a mystery illness. This case of the mystery illness abides to the schema of the clinical analogy, but at the same time is imbricated in the creation of a discourse, and is, therefore, as an inventive instance an instance during which the clinicians involved with the mystery of the new disorder propose theories which would explain the disease – its pathogenesis and etiology. Following Derrida, I will offer a sort of archaeology of invention: nothing new is ever invented, invention is not a discovery, but a signature. As I elucidate Foucault's idea of the functional interaction, the reader will already be able to see how this postulate of invention can relate to the clinical analogy.

2.1.1. Distinguishing the Empirical and Theoretical

The clinical analogy/ fruitful analogy is not, then, a theoretical tool but an empirical finding with regards to how clinicians of the Classical period conceptualized disease and the relationship between disease and the human body. Foucault does not try to create and impose a theory upon the writings of those early doctors – that is, he does not "theorize" their work -- instead he analyzes their writings to understand the theories used in their work. These theories are what is important to Foucault not the theories of some philosophers created after the fact of the matter, but the very theories which at a given time, governed the thoughts and practices in a world; the very theories that let that world be as it was at that time. The point is not to uncover the hidden or secret ideas which were kept silent during those times and which we now, with our better and more sophisticated understanding, can extract and reveal. The point is to give voice once again to that which was vocal and which was spoken during those periods. And so the fruitful analogy is not the secret and hidden logic behind medicine of the Classical period which we have now uncovered, but it is the very thing that those same clinicians knew, discussed and practiced during their time.

> "To be brief, then, let us say that history, in its traditional form, undertook to 'memorize' the monuments of the past, transform them into documents, and lend speech to those traces which, in themselves, are often not verbal, or say in silence something other than what they actually say; in our time, history is that which transforms documents into monuments" (Foucault 2011, 7-8).

The clinical analogy is not a document then, it is the reconstituted monument; a window into a world 200 years past. It is not its secret or silence which has been given voice, but on the contrary, those very vocal parts which formed that period of time not just in its intellect or rationale but with its very real and material consequences – consequences such as the very real diagnosis and treatment of people.

2.1.2. Power, Subjugated Knowledge, Archeology

For Foucault, the whole archeological and genealogical project can be described as the attempt to trace the emergence of different forms of power. As with clinical analogy, what Foucault has to say about power is empirical finding, not "theory," as it is often treated. The form of power he describes is radically different from the power described by Marxist thinkers, which

theorize power as profoundly economic and based solely in the mode of production and private property relations. This second point will be discussed later in much more depth as I introduce Derrida's work into the conceptual framework but it will suffice to say here that the dominant structuralist viewpoint of the time considered philosophy to be at the center of human thought and action. This is a postulate that Foucault rejected by showing, through stringent empirical work, that human history and human knowledge do not operate under a transcendental schema, neither do they operate according to some propositional or analytical logic; indeed; human knowledge is often based on 'historically' situated a priori knowledge that changes and disappears from the sphere of 'official knowledge' and while this knowledge disappears, the discursive formations and forms of knowledge which it gave way to, live on and exist. They live on to shape our existence and guide our experience.

Alas, the clearest and reader-friendliest definitions of the three most famous Foucauldian concepts of power, archeology, and genealogy are not to be found in his major works like *The Archeology of Knowledge* or in *The Order of Things* (they are there, to be sure, but they are difficult to extract in a denotative form; usually buried within complex philosophical or empirical discussion), they are, in fact, to be found in one of his more minor lecture series books, *Society Must Be Defended.* There, on the idea of economic power, Foucault writes,

> "Is power always secondary to the economy? Are its finality and function always determined by the economy? Is power's raison d'etre and purpose essentially to serve the economy?" and "Is power modeled on the commodity? Is power something that can be possessed and acquired, that can be surrendered through contract or by force, that can be alienated and recuperated, that circulates and fertilizes one region but avoids others?" (Foucault 2003, 14)

This critique of economic power is equally pointed towards both capitalist (the formal study of economics, for example) and Marxist analyses of power. Can power be analyzed as something that does not depend on the basis of society, on private property relations, on the means of production and on the commodity function of the economy?

Indeed, what other ways can we conceptualize power? What we see in *Society Must Be Defended* is the beginning of Foucault's investigation into state and political power – this is an investigation that will develop into a discussion of governmentality and biopolitics in further lecture series books. But what is found in these lecture books is just one form of power, as we similarly see an erudition of psychiatric power in *History of Madness*, of scientific power in the

Order of Things, and of the power of the clinical gaze within *The Birth of the Clinic*. If we look at all of these varied archaeologies and the descriptions of power that we can articulate from them, we will see that what they have in common is the ability of these power structures to subjugate knowledge – this is the central point of the all too often cited term 'power – knowledge'. Foucault defines subjugated knowledge as,

> "…historical contents that have been buried or masked in functional coherences or formal systematizations" and "…a whole series of knowledges that have been disqualified as nonconceptual knowledges, as insufficiently elaborated knowledges: naïve knowledges, hierarchically inferior knowledges, knowledges that are below the required level of erudition or scientificity." (Foucault 2003, 7)

Power, to an extent, then lies in hiding this knowledge away – in placing it in the proverbial basement. This knowledge is still a part of the formal systematization, but it is buried. It is still knowledge that helped produce the functional coherences, but it has perhaps been subjugated under the constraints of empiricism and positivism. It is not some contradiction that had been solved or eliminated and neither is it a 'secret contradiction' which must be uncovered[6], it is still there to be seen if one was to pick apart the formal system that sits upon it.

Such knowledge exists in many different forms: we are talking here as much about the subjugated knowledge of the patient as we are about the subjugated knowledge of the clinician. Both are buried within the biomedical discourse. The idea here is not exclusively about the subjugation of one group's knowledge by another group's knowledge through force or authority but the loss of knowledge as the complexity of thought within some particular discourse increases. With this increase, and as different disciplines tend to 'move on' to higher grounds of thought, certain knowledge is forgotten, other knowledge is formalized into 'facts' and presuppositions that no longer have to be checked or verified but can just be assumed. This type of knowledge is then subjugated within the formal systematization that it helped create; the systematization no longer requires that subjugated knowledge to operate, on the contrary, that

[6] In *The Archeology of Knowledge*, Foucault briefly cites this term of 'secret contradictions' when talking of established unitary discourses such as psychiatry or political science, he says, "…but I shall not place myself inside these dubious unities in order to study their internal configuration or their secret contradictions" (Foucault 2011, 29). It must be noted here that in all likelihood this is a jab at Derrida and at 'deconstruction'.

subjugated knowledge now exists solely as a faint and distant memory – a trace – within that particular formal system or discursive formation.

In many ways, this idea of subjugated knowledge is the focus of this work; the uncovering of subjugated knowledges within the clinical discourse surrounding the mystery illness which allowed the discourse to exist in the first place. What we will look at is how these subjugated knowledges, these unstated assumptions and presuppositions helped create some of the first hypotheses for the etiology and pathogenesis of what was later named AIDS.

So if the task is to unearth these subjugated knowledges, what is this archeological method by which I am to do so? The above discussion of power already lays out some of the goals of archeology but to be more specific, Foucault says quite simply that, "Archeology is a method specific to the analysis of local discursivities." The goal of archeology, in a sense, is to lay out the field, untangle and unfold the intricacies of a specific body of knowledge – to establish and define the parameters and rules of a discursive formation. The scope of the archeological analysis is therefore large. Just by looking at the sorts of analyses carried out by Foucault himself, we can see that his meaning of 'local discursivities' encompasses investigation into the overall history of madness in European civilization or of the history of European medicine – large topics indeed, and seemingly not so 'local' unless we are to conceptualize Europe as one 'locale'. This is why in contrast, my investigation will actually be of a local discursivity; the scope of the study covering but one part of a localized discourse emerging around this issue of the mystery illness that itself operates within the larger biomedical discourse of the time. Two questions may arise: (1) is a model of analysis suited from such large scale macro analyses reducible to an analysis of such a small scale? (2) Would it not be simpler to simply call the proposed analysis a genealogy?

First, while the archeological analysis can be used to analyze macro level phenomenon, it can also be used to analyze other smaller instances. Foucault writes that any discourse has several levels on which it operates. A discourse can simultaneously operate at,

> "the level of statements themselves in their unique emergence; the level of the appearance of objects, types of enunciation, concepts, strategic choices…the level of derivation of new rules of formation on the basis of rules that are already in operation…lastly, a forth level, at which the substitution of one discursive formation for another takes place" (Foucault 2011, 189).

My analysis, then, will operate at these first three levels. First, I will show how existing statements were transferred into the emerging discourse of the mystery illness, then I will outline the development of the first hypothesis (concept) which hoped to explain the mystery illness. Lastly, I will show how this concept was eventually formalized as a rule of formation for further hypotheses into the nature of the mystery illness.

Second, why not simply call this a genealogy? Well, what is a genealogy? Foucault would say that "genealogy is the tactic which, once it has described these local discursivities [once archeology's work is done], brings into play the desubjugated knowledges that have been released from them" (Foucault 2003, 11).

The reason I would not call my study a genealogy is because the focus and the direction of the study will somewhat shift after the process of establishing the 'local discursivity' is complete. What the reader will not see is a shift from bringing 'into play the desubjugated knowledges' to trying to better describe and clarify the inventive instance of the hypothesis; to understand how at this level of statement and concept, actual and real clinical hypotheses for the mystery illness in question had emerged. The goal will be to establish the conceptual framework to better understand the specific inventive instance in question. The hope is that the resulting framework can shed at least some light into a more general understanding of the process of scientific and technological invention.

The project is then not a genealogy as the goal is not as clear cut as bringing newly desubjugated knowledge into play against prevailing and established modes of thought. Neither can this be called an archeology due to the small scope – a sort of 'partial' archeology at best. Furthermore, as will be evident further on, the divide between this analysis and an archeology will widen as several different theories are brought into the fold in the effort to better understand this inventive instance in question.

But more theoretical work yet remains before we can reach the actual study itself and the empirical side of documents and analysis. The first task is to complete the argument that was made in the first section of this chapter: to articulate a proper usage of Foucault's empirical discussion – specifically the empirical discussion that will apply to this study – of the functional interaction found in *The Birth of the Clinic*. In this discussion we will find that this functional interaction is well suited for a discussion of modern medical practice. However, we will see that it

is missing certain elements which will make it functional for a discussion of invention and the inventive instance.

2.1.3. Transposing to the Modern

At first glance, using social theory seems problematic does it not? How can I use such Foucault's analyses of 18th and 19th century medicine as a way of understanding fairly modern late 20th century medicine in my research? Surely, the ways of understanding medicine within the medical compound itself have changed. Indeed, medicine has become more technologically advanced, the medical language more technical, and yet, we see that at their core, the philosophical principles of seeing the human body as a field in which disease is to be discovered and cured have not changed.

The methods of ascertaining the illness remains the field of symptoms as signs – and when there are several symptoms to be immediately noted they form a unique combination which can then be identified as a particular disorder or disease. Towards the beginning of the Classical period, disease existed within "the visible multiplicity of symptoms that signified its meaning without remainder" (Foucault 2003, 117). Similarly, the clarity of the disease did not exist within the essential generality (and likewise categorization) of the disease but within the complexity of combination which constituted the disease. The principle of intelligibility is therefore established on the combination of different vital symptoms and signs which accompany a disease. To be sure, this still remains the language of the clinicians who we will look at in this study.

When addressing the new syndrome and the appropriate response that clinicians across the country should have towards it, the *CDC Control Task Force on Kaposi's Sarcoma and Opportunistic Infections* states that,

> "[c]linicians caring for homosexual men should be alert for signs and symptoms of other infections and neoplasms associated with immunodepression, in addition to Kaposi's sarcoma and P. carinii pneumonia." (*Center for Disease Control Task Force on Kaposi's Sarcoma and Opportunistic Infections* 1982)

Similarly, we can see that the *Dorland's Illustrated Medical Dictionary* (32nd ed.) defines disease as "any deviation from or interruption of the normal structure or function of a part, organ, or system of the body as manifested by characteristic symptoms and signs; the etiology, pathology,

and prognosis may be known or unknown." This definition seems to still participate in the 19th century understanding of disease described in *The Birth of the Clinic*; the language is that of interaction and configuration, of ascertaining the particular set of symptoms and signs presented by this mystery illness and figuring out the particular combination of causes of the disease. The sign, however, is different from the symptom. While the symptom is a sign of a disease, the sign does not necessarily have to be a symptom. As we shall see in the empirical discussion, what constitutes a sign in the analogy of the disease can therefore exist outside the immediately visible aspects of the patient's body.

We have to realize, of course, that what constitutes the visible has changed with the rise of biomedicine. Technology now allows those things that were previously not symptoms, because they were invisible to the doctor's eye, to become part of the symptomology of the disease as they are rendered visible by technology. For example, in this study we will see mention of such disorders as leukopenia which can be made visible through the process of flow cytometry; where the different blood platelets in a patient's blood sample are counted and categorized with the aid of lasers, light detectors, and electrical impedance. In this way, the molecular is made visible and observable. Note that this does not change the inherent logic of diagnosis, but instead, conforms new technologies and tailors them towards an existing logic. Similar to the case of flow cytometry, many other previously invisible features of the human body have been made visible and have been transformed into signs that interact with each other to create the analogy of the disease – the functional interaction.

2.1.4. The Functional Interaction

While the flow cytometry described above can produce numerical and calculable results not all is so simple. For example, we receive a count of white blood cells from a particular patient's blood but what can we say about it without a referent? How can we say that it is either normal (what constitutes a normal range?) or that it is either too low or too high? We need to first establish reference for what is a normal human range that perhaps furthermore differs by age, sex and weight (undoubtedly many other categories exist as well). Let's now complicate the problem; while blood cell levels attain importance as indicators through a numerical function (they can be counted and from that it becomes evident how one goes about establishing a referent through statistical means), how would one establish a referent for the swelling of the neck as occurs with

the swelling of the lymph glands during lymphadenopathy? Outside of the cases encountered and noted by doctors, does there exist a referent independent of the particular of each patient? Another example that many people have encountered at some point: you visit a doctor and she asks you to rate the pain you are feeling on a scale of 1 to 10 (a pain you may be feeling due to a broken finger or a herniated disk in the spine). What referent exists to actually assess that pain – what point does the interval scale of 1 to 10 actually convey? How can the particularities of pain even be conveyed from the particular experience of a person to a generalized or universalized aggregate that is available to the gaze of the clinician?

This ability of the clinician to order and organize these human experiences does not develop out of the procedure of taxonomy or ordering but of an analogical process. The ability of the clinician to recognize something as a symptom depends on the coexistence of that particular phenomenon within a network of other such 'symptoms'. What gives these 'symptoms as signs' an identity is not commonality or a comparison to some referent form but the difference between the signs themselves as "in a system of signs, there are only differences, [and] no substantial existence" (Ricoeur 1976, 5). Signs, in this clinical sense, do not signify a referential form as no such stability exists.[7] This referential form, the human body, has no ideal form as every human body is different. A referential form, however, did develop, but did so out of accumulation and not out of an assertion of a deduction of a universal form; as clinicians expanded their experience by encountering an ever increasing number of human bodies (patients), regularities could be established. Through this gradual process (superimposition in Kant's idea of reflection), clinicians could eventually comprehend what the different parts of the human body should look like and what they shouldn't look like.

This experience, to some extent, also began to include the interior of the human body and referential forms for the different organs could also be eventually established[8]; clinicians could eventually figure out what the different organs should look like, how large they should be, the

[7] The problem is as old as philosophy itself. Can the content ever fit the form without remainder? Can the universal ever completely account for the particular? A favourite debate topic for philosophers from Aristotle to the present.

[8] This process, described in the chapter titled "Opening up a few Corpses" in *The Birth of the Clinic*, was substantially more complex as the operation techniques were not as developed and in most cases the patient needed to have expired before the body could be 'opened up' – surgeries of the time often concluded with the patient

basic structure of their respective internals, their texture, their pigmentation, how squishy or dense they should be, etc. Accordingly, with the recognition of what an organ should be like, developed the recognition of what an organ shouldn't be like; the swelling of a particular external or internal body part could therefore be recognized as something irregular and problematic. Doctors of earlier times would have then proceeded to examine the swelling or inflammation itself – its size, its warmth, the humors that secreted from it, etc. With the advent of the clinic, however, came the recognition that (1) there was usually more than one descriptive quality (symptom) associated with any particular illness and (2) that these multiple symptoms did not operate in isolation from one another but formed a functional interaction which could more fruitfully be used to describe a disease. Similar to how symptoms operate as signs, the designation of the disease does not depend on this functional interaction in and of itself but on this interaction's relation to and difference from other such functional interactions of symptoms as signs; the process is analogical.

The ability to recognize specific diseases and account for the dispersion of differences encountered on a case by case basis was enabled by the recognition of the "combinative variety of… simple forms" (Foucault 2010, 121). It was no longer the case that a disease was recognized as an abstraction or deviation of a perfect form or essential figure of a disease, it was now the issue of recognizing the disease in itself; in recognizing "all the elements that compose it and the form of that composition" (Foucault 2010, 122). This is the very cornerstone and the beginning of being able to establish consistence between diseases; of being able to recognize between one and another.

> "The combinative study of elements revealed analogous forms of co-existence or succession that made it possible to identify symptoms and diseases… The analogies on which the clinical gaze rested in order to recognize, in different patients, signs and symptoms… 'consists in the relations that exist first between the constituent parts of the disease, and then between a known disease and a disease to be known'" (Foucault 2010, 122).

It is therefore a particular combination of symptoms which allows for the recognition of diseases; the recognition of the interaction of these systems in an intelligible formation. Therefore the analogy spoken of here is not that of the '4th species of metaphor' encountered in Aristotle's *Poetics* but functions off an "isomorphism of relations between elements" (Foucault 2010, 122).

Moreover, it the particular analogical transfer, that is observed from several repeating combinations, which allows for this 'fruitful' analogy to develop.

> "The fruitful analogy that identifies a symptom is in relation to other functions or other disorders… form[s] a constellation in which the coexistence of elements designates a functional interaction. It is the analogy of these relations that makes it possible to identify a disease in a series of diseases" (Foucault 2010, 123).

This is perhaps the most crucial part: the ability to analogise the particular functional interaction and transfer it to another such function. It is the ability to say that the interaction of one group of symptoms is analogous to another group previously encountered. Note the difference between this analogical transference and resemblance; the former designates and superimposes on existent forms to create an ideal or essential form. But the major shift that is brought about by this idea of fruitful analogy is the disappearance of the attempt to establish this essential or ideal. The recognition of disease shifts from the degree of dispersion and diversion from an essential form and begins to function on a principle of analogy: one combination of symptoms is analogous to another as that other is analogous to yet another and so on. With the eventual accumulation of cases, knowledge and scientific certainty can be "[extracted] from a mass of sufficient probability" (Foucault 2010, 124) – a probability which strengthens the certainty of the analogy.

However, the truth of the analogy and the way it becomes certain, is a topic that gets away from the purpose of this section. The importance of this fruitful analogy lies in the structure of the combination, in the functional interaction itself. Specifically, what is the logic of the combination? How does the function in the functional interaction develop? How is it structured and how does it fuse the combination of symptoms. Now, it is not the purpose of the archeological analysis to investigate such subtle details and so Foucault can be excused in not investigating such micrological details considering the monumental task of the overall book. What he does say is:

> "From one symptom to another, in the same pathological entity, a certain analogy could be found in their relations with 'the external and internal causes that produce them'" (Foucault 2010, 123)

First we must note that Foucault refers to these 'causes' as 'elements' interchangeably. But what are these causes? What distinguishes internal from external causes? The *Columbia*

Encyclopedia defines external factors as "infectious agents, include both microscopic organisms (bacteria, viruses, and protozoans) and macroscopic ones (fungi and various parasitic worms)" while defining internal factors as "hereditary abnormalities, congenital diseases, allergies, endocrine disorders, circulatory disorders, and neoplasms, or tumors" (*Columbia Encyclopedia*). With these definitions we should have a pretty clear idea of what constitutes these external and internal causes in the medical sense. What we have to expand on, however, is this idea of external causes. We will see later on that some 'internal' causes of the mystery illness were suggested. Some papers did provide hypotheses that suggested the possibility of genetic predisposition of those with HLA-DR5 (now HLA-DR11 and HLA-DR12) to at least some aspects of the mystery illness and it is still noted today that those with the particular serotypes who develop AIDS have a higher risk of developing Kaposi's sarcoma.

The researchers who first tackled the mystery of this new condition did not limit the external causes, which they used to create these first hypotheses, to microscopic and macroscopic infectious agents as we saw in the official encyclopedic definition above. While these infectious agents certainly did play a great part in the creation of these first hypotheses, the clinicians also reached out to a number of other elements such as drug use and to the particularities of the homosexual lifestyle (as it was then understood) to explain the new disorder. It was suggested in these early explanations that it was possible that amyl nitrite inhalers (poppers) depleted the immune system. Likewise, and most importantly, it was also suggested that the sexual promiscuity of homosexuals itself, separate from the transmission of infectious agents during sex, was responsible for the illness. Rarely in these early hypotheses and discussions was there a suggestion of a possible new infectious or etiologic agent. The outmost focus of the clinicians was to explain this 'new' with the use of that which was already known.

This new has to do with the peculiarity of the inventive instance of this study. How does one decide on the external causes of a disease when it is completely new for the clinical gaze? The viral etiology of AIDS was only established in 1986 when the respective findings of the Gallo and Montagnier research teams were, in a sense, combined to designate the human immunodeficiency virus (HIV). But what can one say in the absence of such information? How is knowledge created when neither side of the causation 'HIV causes AIDS' yet exists – when neither side has yet been designated or named. What we will see in the following chapters is an attempt to establish pathogenicity and etiology with what they had available and what existed

ready at hand. Indeed, we will see that the first reaction of the clinicians to with something new like this mystery disease is not to then similarly search for its cause in the new but to try to establish its cause in that which is already known. And yet, something new is still created; the inventive spark is still imparted unto the new functional interaction of elements. However, it would not be prudent for the investigation to stop there for this inventive spark itself must be investigated – such an important detail cannot be left to individual ingenuity. While popular culture likes to paint such breakthroughs as sudden moments of discovery followed by a proclamation of 'Eureka!' scientific progress is not exemplified by them. Deus ex machina is not an established plot device in the story of scientific progress.

A critical part that has not yet been covered is the question of the combinative logic of the functional interaction. Is there some guiding logic which regulates this combination? In the following empirical chapters I will trace the development of a rule of formation for the pathogenesis and etiology of the disease but before I get to that step, I must establish some understanding of how we can approach the investigation of this rule of formation. There is no standard structural blueprint to the logic which guides such rules of formation. Also, to further deviate from the structural analytic framework and propositional logic, we cannot even say that the logic of these rules of formation is necessarily logical.[9] It is, after all, an invention of human thought, of human intuition and human action.[10] This is an idea that is in many ways a definitive question of post-structuralist though; do humans actually think in logical terms? Is our civilization (unlike the principles of our high culture which is the transcendent vision of the ideal that could one day be achieved) guided by the rules set out in the language of propositional and analytical logic? If it is, why is there war, famine, strife and sorrow in this world of ours? These initial few points can be expanded into a far lengthier discussion of the differences between analytic philosophy and structural social theory on one side and more recent 'post-structural' ideas on the other. I don't intend to do this here. It will suffice to say that rules of formation are

[9] A great discussion of this, which in many ways defines Foucault's 'post-structuralist' side, can be found in his rebuttal to Derrida's critique of the *History of Madness* in "Reply to Derrida" which can be found as an appendix in the 2006 English translation (Murphy and Khalfa trans.) of the book published by Routledge.

[10] While the discursive formation is formed on the basis of the rules of formation of that discourse, it is often misunderstood and criticized as a determinist structure lacking human agency. But the rules of formation are themselves human creations, they are not transcendent and they are not received from above. They are created by humans and that is itself a sign of human agency.

not beholden to the rules of transcendental analytics and propositional logic. Now that we have discussed what they are not, let us cover what rules of formation are.

Foucault states that "[t]he rules of formation are conditions of existence (but also of coexistence, maintenance, modification, and disappearance) in a given discursive division" (Foucault 2011, 42). A rule of formation regulates the creation and existence of statements and the formation of statements. It is that by which we are able to understand the existence of objects and "the rules of formal construction" (Foucault 2011, 66) of those objects. This is where the archeological framework can be used to discern and show how the object of discourse, the mystery illness, was constructed from the convergence and combination of statements within a specific discourse and according to the rules of formation for statements within that discourse.

Further towards the end of Archeology of Knowledge, Foucault additionally clarifies that the rules of formation are not to be confused as being some essential character of the objects which were created under such rules. They are not to be confused with something like Aristotle's Forms. The rules of formation are "neither the determination of an object, nor the characterization of a type of enunciation, nor the form or content of a concept, but the principle of [an object's or statement's] multiplicity and dispersion" (Foucault 2011, 191). And so the rules of formation are not those that guide form the functional interaction and they are not at the heart of the logic within this complexity of combination. The rule of formation is not that logic which arranges external and internal causes into a particular functional interaction and analogy of a disease. The rule of formation is the element which makes such an arrangement, such complex combinations, positive and scientific knowledge – knowledge that is communicable and intelligible amongst and by people within a discourse. The rule of formation is that thing which allows for the dispersion of such a taxonomical method of categorizing, designating, and diagnosing diseases.

But if we want to employ the schema of the functional interaction for the study of the mystery illness and how different hypotheses of that illness developed, the notion of 'rules of formation' does not help us much as it only points to that which has been established and so lends us no insight into how those hypotheses were established, only of what they were once established. So to investigate the development of such hypotheses we need another theoretical instrument. For this purpose, the next section of this chapter will discuss Derrida's deconstruction of the idea of invention. After all, invention of new hypotheses is exactly what we are dealing

with in this case. Furthermore, in the following discussion I would also like to introduce the concept of tekhnē as a contrast to the rule of formation. That is, where the rule of formation is something set and static, tekhnē as I will introduce it and use it, is a form of human understanding (*Verstand*) which precedes the rule: it is the first murmur of a discourse, it anticipates the rule.

2.2. Invention

In this section of the chapter I will discuss Derrida's essay entitled "Psyche: Invention of the Other". What is most important about this essay is that it investigates the idea of invention and strives to present a deconstruction of invention. How this fits into the previous discussion of the functional interaction will become clear in due time but first we need to discuss and understand the invention and the inventive act. In his essay, Derrida discusses two 'types' of invention although neither can or should be thought of as being mutually exclusive or completely separated from the other. Similarly, I will right away caution the reader against understanding invention as a thing or material object. We are not here discussing the contraptions or gizmos of the inventor-entrepreneur scurrying off to the patent office after that much fabled 'Eureka!' moment. Our discussion revolves around the invention of thought and of new modes of understanding. In particular, this first 'type' of invention is one which recognizes what was already there all along. And this is the puzzle that Derrida introduces: for something to be invented it must already exist, invention is not invention on the first/ inaugural time, but only when it is recognized again.

What is a signature in the context of invention? What is it to put a signature onto something? Would it be correct to consider signatures in a purely linguistic and immediate sense of the author consciously writing a signature at the end of a letter or some other document? Or can we consider signatures as aspects which appear in the different toils of human existence. Ad absurdum, we could realize 'signature' in all particulars of human action. Here, we will talk of signatures as we would of inventions and discovery. It is that thing that the inventor/ maker imparts on an object or configuration which gives it that quality of being new, unique, or somehow better than what has previously existed. But we have to be more particular and begin with several examples which will distinguish particular instances of signature.

When we see a primary school level history book and read that Columbus 'discovered' the New World, how do we understand this? Would it make sense to say that Columbus 'invented' the New World? This would obviously not make that much sense but in further discussion we will see that the two terms are not too far apart from each other. Did Columbus put his signature on the 'New World' he discovered?[11] In what dimensions did this occur? Is it the actual act of discovering the 'New World'? Can his signature be described as the historical legacy he left – whether one celebrates Columbus Day or lambasts the atrocities that those explorers committed against the indigenous populations of the New World (and the atrocities of explorer conquistadores to further follow)? We can say without doubt that the New World existed before Columbus discovered it. Then what is at the basis of the claim that in fourteen hundred and ninety-two Columbus sailed the ocean blue and discovered this land anew? What constitutes the basis and essence of discovery in this case?

Besides contributing to the theories of the spherical nature of the world, Columbus undoubtedly contributed to the legacy of colonialism (positive or negative) which gave way to further exploration and colonization of this New World (and indeed, countries like Canada and the United States are indebted to Columbus for their existence to some degree). As European nations gradually grew in power and empires were formed, Columbus' initial voyage was elevated to the ranks of legend. As it became part of the locust of relations within the European consciousness, Columbus' voyage was immortalized. It was, not however made into anything special at the point of discovery – when Columbus or his crew first set foot onto the solid ground of the New World. Indeed, if we are to follow the accounts of his first voyages, we find that the 'would be legend' actually thought that he was in quite a different part of the world at that point.

And so discovery is not initially construed as discovery at the point of discovery. The full extent of the importance and purpose of such a discovery may only be made clear long after the fact of the discovery. It may be prudent now to ask, 'at what point is the signature of the maker placed onto the discovery?' However, what we need to do first is rephrase the question and so we need to ask, 'What is required for the signature of the maker to be placed on the discovery?'

[11] This is similar to the discussion of Columbus that Derrida has in his own essay but what I would like to do is simplify it a bit (from a discussion of Cicero and the philosophy of Paul de Man) before regressing back to the main point that Derrida is trying to make.

2.2.1. Communicability

At its basis, such a discovery can only be named ‘discovery’ if it is known by more than just the person responsible for the discovery. If a person makes a discovery but does not tell a single soul about it and takes its secrets to the grave can we rightly say that any discovery was made at all? Hypothetically, we may say that there was a discovery so let’s look at a hypothetical example. But let us imagine for a second a scenario which is to some degree plausible. Imagine a person who, due to the particular conditions of some specific place and time, has a brilliant idea – a revelation that would shatter our present understandings of science and nature as we know them. He does not write this idea down, however. Perhaps it is because the idea is so complex that it does not correspond to that person’s capacity to express it and so they keep it inside, never finding the right words or the right situation to say it out loud to another person for fear of seeming insane. Perhaps, it is an idea that, while it may be brilliant in hypothetical retrospect, does not seem all that special to the person thinking it and, therefore, they never mention it to anyone and gradually forget it. Maybe, the person was in a state of intoxication or narcotic influence when he had this idea and while he knows that it was something very important, he cannot, for the life of it, remember this discovery the next morning. Can this discovery be accurately labeled a discovery? Can it be seen as such if this one person is the only one to recognize it as a discovery or further yet, if this one person himself does not even count it to be an idea of any importance?

Concretely, a discovery is only a discovery when it is recognized as a discovery – it preconditions within itself the recognition of itself by more than one person. Schrodinger’s cat may be alive or dead but we can only know its fate if we open the box (provided the cat is not able to tell us itself from within the box by emitting some recognizable cat sound). Does a similar state of superposition exist when we talk of discovery and invention? In the case that we recognize something as a discovery, the possibility of it not being a discovery is nullified ipso facto. At this point we do not have the choice not to recognize a discovery as a discovery – it is

not up to any single person to pick and choose what will count as a discovery and what won't[12]. If we do not recognize it as a discovery and the cat turns out to be dead, no mention of the occurrence is made in the annals of history altogether. It is simply forgotten with innumerable different other events that have occurred throughout recorded human history. It is not that it is not mentioned consciously, the point is that it isn't noticed like the majority of human activity.

2.2.2. Example: Inventing the Curveball

When was the baseball curveball invented? Was it when the first pitcher to throw it that certain way managed to throw it that certain way for the first time? Was it invented when that first pitcher who managed to throw it that first time, similarly managed to systematize it and learn how to do it consecutively? Was it invented when the opposing team managed to discern this throwing technique and labeled that pitcher as a cheat and the technique as illegitimate? Was it when the sports journalist gave it a flashy new name? We can go on and on with such similar questions and propositions. What is common amongst them, is that in their current form these questions all assume and present a time point of invention and they cannot begin to establish any sort of analysis. Analyzing something from this purely historical and genealogical perspective leads into a trap of ad absurdum reducibility.

Trying to ascertain the genesis of the idea can only go so far before it leads us into obscurity and questions such as, how do we know that the first pitcher to reportedly throw the curveball was even the first pitcher? Maybe he learned the technique from someone else who never became famous? Maybe there were earlier unrelated and unaccounted-for events of a curveball being thrown. Of what importance is the first/inaugural event itself then if it is not at that point that invention becomes invention but only when that initial event is recognized and

[12] At this point, you may point my attention to the realm of discoveries within the hard sciences like physics where there are clearly delimited points at which something is deemed a 'discovery' like the 5-sigma rule. Here I would direct you not to the conclusion and confirmation of the experiment by a set of regulative statistical parameters but to the inception of the experiment, the point at which that one scientist had the brilliant idea which she shared with her colleagues who then formed a team which went on to be appropriately funded and carried out the experiment. That origin – that point of genesis – (and I use the term 'genesis' with caution here) is what is at stake in this discussion. That original idea, that new and innovative way to look at something is what is important. This is the discovery of which we are talking here – this is the process of invention that we are trying to understand.

counter-signed by another – something which may occur soon after the inaugural event or very long after it.

To designate this point at which the invention becomes an invention, Derrida prefers to use language more akin to the political and judicial sphere. For him an invention becomes such when it is accorded a patent – a legal standing and recognition. And yet, after a somewhat digressive discussion into this juridicopolitical sphere, the status of invention is clarified as "the public recognition of an origin, more precisely, of an originality" (Derrida 2007, 28). The original signature of the inventor is then, not something that is made, but something that is recognized and countersigned by a consensus. Invention then is not something that is the quality of the ingenious individual; it is a social practice and is something that is the outcome of the actions of the other(s) (consensus on recognition or recognition of consensus) as much as it is the outcome of the individual's inventiveness. Now that we have further pondered the broader philosophical problematics of invention and have brought it out of the sphere of the particular and into the sphere of the social we can begin to more closely look at the internal workings of the inventive act.

2.2.3. invention iunctura (Invention by Combination)

In his essay, Derrida establishes and distinguishes two different types of invention. He claims that the two are historically situated in different time periods. The first kind is the one that we have so far discussed – invention that is in many ways synonymous to discovery or to 'seeing for the first time'. It is also the older kind of invention and has been, in accordance with the advent of the modern era, replaced by the second type of invention; invention by combination. In opposition to the first type which "discovers, for the first time, [and] unveils what was already found there", the second type "produces what, as tekhnē, was not already found there but is still not created, in the strong sense of the word, is only put together, starting with the stock of existing and available elements, in a given configuration" (Derrida 2007, 25). It is not the point here to discuss if Derrida is correct – if there can be found a clear cut disappearance or devolution of the first kind of invention in favour of the second kind with the advent of scientific and technological tekhnē. I think that what we need to consider is that neither kind of invention has disappeared as such. As the preceding examples show, invention as 'seeing for the first time' is something that still occurs. It is something that happens at all levels of human experience, from

the personal and mundane to the public and important. Then what is it about this second type of invention – invention by combination – that makes it the primary form of recognized invention in our age? This question alone merits its own lengthy investigation but what we can leave off with here is that it is both a social and cultural recognition. In many ways, this second type has become the dominant one because it is the one that is most frequent within the sciences, within industry and within the sphere of consumerism – it is the type of invention that has given us a gradual change of the cell phone into a miniature personal computer with new functionalities added with each subsequent revision. Am I simply saying that this type of invention has found more purpose within the capitalist mode of production? I may be, and similar points have been raised by theorists such as Marcuse (technological rationality) and Lyotard (efficiency in *The Postmodern Condition*)

The clever reader will note the similarities between the functional interaction discussed earlier in the chapter and this invention by combination. Before I delve into a discussion of these similarities and a synthesis of the two ideas I would like to discuss briefly several important technical points of this type of invention; we need to understand what is meant by tekhnē and the 'double movement'.

What is meant by tekhnē? In terms of its Greek origin, the word stands for "the activities and skills of the craftsman, [and] also for the arts of the mind and the fine arts" (Heidegger 1977, 13). It is then related to all aspects of the human producing something – of creating, of making. As Heidegger notes in his famous essay *On the Question Concerning Technology*, technē[13] is also a revealing, it is the ability of the human to conceptualize the existent as something new. Heidegger notes that it 'belongs to' poiesis[14], to bringing-forth, to making. However, before an object is made it must be recognized, it must be made in the mind. For nature (physis) to be re-conceptualized and made (poiesis) into something new it must be reconfigured in accordance

[13] While the spelling of the words technē and tekhnē are different, Heidegger and Derrida are talking about the same thing.

[14] Before further questions appear in the mind of the reader, I would like to note that 'technē/ tekhnē' are not the same as 'rules of formation'. The 'rules of formation' are specifically the established rules for the formation of statements within a discourse (it is something that is used to create), while technē is the revealing that occurs within the mind (conceptualizing the rock as a tool of sorts) before the action takes place (picking up and using the rock as a tool). While there may be some similarities between the two terms, they are not interchangeable.

with a particular tekhnē. When a rock found on a beach is suddenly reconceptualized as a hammer/tool in the hands of a human, a particular and important process takes place. Technē is that way of seeing a thing as something else – invention of the first type. But technē is not the technical know-how of the act of hammering or hitting something; it is the realization which occurs within the human before he pics up that rock that it can in fact be used for the purpose of hitting something or as a projectile which can be launched into the tree in an attempt to knock down that bird or squirrel so that it can be eaten.

In many ways it is the movement of the thing's essence – an essence that is not contained a priori within the thing but which is itself socially accorded upon the thing. The change in its essence is a social movement, a changing relationship of the social to the thing. And this is key: technē is social – it is changing, it is dynamic, it is historical, but not necessarily entwined in historical materialist dialectics nor is it necessarily a logical construct of transcendental propositions. It is the methodology of a particular historical[15] epoch. It is the art of seeing something else in what already exists; the conceptualization of the existing into something new through tekhnē – the signature of the inventor. Just as importantly, it is the acceptance of the new way of seeing the invented as invention by another – the countersignature. This can be described as a double movement – the movement of a thing from one existence to another and the subversion of the established and existing – an "upsetting of…expectation and reception that [the invention] nevertheless needs" (Derrida 1997, 25). At the same time as the existing thing moves towards being reinvented as something new, the other must move towards it to recognize it as invention.

At this point I want to briefly attempt to bring the archeological framework and this deconstruction of invention a bit closer together. The reader may have noted that this concept of technē/ tekhnē and Foucault's functional interaction are in some ways similar to each other. We must be careful, while they have some common elements, they are each distinct. The rules of formation speak of those rules which are set and pronounced and by which statements are formed within a particular discursive formation. Technē is the ability of thought to see and to reveal – of poiesis. As Heidegger describes the technē of our time, it is the ability to look upon the forest and

[15] It would be foolish of me not to at least mention culture and geography here even if Derrida does not address them.

enframe it as a standing reserve of lumber, would be houses, and profits. In more recent times, we look upon the sun, and besides blinding ourselves, we see a limitless standing reserve of clean energy. Indeed, it would be interesting to investigate to what extent the environmental discourse has changed our relation to technology.

To complicate matters further, I also intend to use this idea of tekhnē/technē in a particular way. As the rules of formation speak of a set discursive formation and of a set system, the reference to tekhnē will be made to indicate something new; a spark of novelty, subversion, and contingency – to an inventive tekhnē. As such, due to the particular case study in question, tekhnē is something that comes before the rules of formation even if this tekhnē is destined to then become such a formative rule.

In this deconstruction of invention, if we look closely at the system that Derrida lays out, one problem arises. If a new form of tekhnē is required to conceptualize the existing into something new, that same form of tekhnē is needed by the other to recognize the invented as invention – to be able to similarly conceptualize the existing in that particular way as did the inventor. Does this mean that the particular tekhnē needs to already exist within the social milieu or is it something that is transferred onwards with the invented object?

If the tekhnē already exists and is part of the public consciousness – what is then invented? What would be then the upsetting and subversive element that upsets expectations and heralds invention in the mind of the other who must countersign the invention? That is, such a thing would not be an invention; it would be nothing new in terms of a revealing or poiesis. It may be something new but would conform to the existing order of things. It would not be an invention but a reproduction. In terms of human thought and knowledge, it would be something that conforms to the existing rules of formation. It would not be that inventive turn of thought that precedes the rule nor would it be that which then becomes the rule or the form of positivity within the particular discourse.

This is why I intend to argue that this tekhnē is something that is transferred with the invented object and this allows for the other to be able to comprehend the invented as invention. This however, still leaves the problem almost untouched as it still does not answer how (1) the tekhnē forms/combines/ configures the existing into the new and (2) how the invented object becomes available for comprehension to the other – how the tekhnē is coupled onto the invented

object. Must the other first comprehend the tekhnē which is being transferred with the object to understand the object or does this happen in some other way as the invention comes face to face with the understanding (Verstand) of the other.

This will be the focus of the subsequent analysis in the following two chapters: to better investigate how the first hypotheses to the etiology and pathogenesis of the mystery illness were invented and to investigate the structure of the tekhnē by which this occurred. I then further want to bridge the gap between tekhnē and the rule of formation by showing how the former is transformed into the latter.

I have for long now foreshadowed this 'tekhnē' and what exactly will be the theoretical instrument which we will look at to study these hypotheses. It is no coincidence that Foucault's description of the functional interaction utilized by 19th century clinicians to recognize, designate and categorize diseases is so similar to what Derrida outlines as the second type of invention, invention by configuration, in his deconstruction. Derrida writes that invention "discovers for the first time, it unveils what was already found there but is still not created, in the strong sense of the word, is only **put together, starting with a stock of existing and available elements, in a given configuration**" (Derrida 2007, 24) (my emphasis). In terms of the functional interaction, Foucault writes that "[t]he fruitful analogy…identifies a symptom is in relation to other functions or other disorders[;] [this] form[s] a constellation in which **the co-existence of elements designates a functional interaction**…From one symptom to another, in the same pathological entity, a certain analogy could be found in **their relations with 'the external and internal causes that produce them'**" (Foucault 2010, 123) (my emphasis).

Within these two quotations, what we can find is a reducible similarity. Note that both are talking of either configuration or combination of the existing into a new. What Foucault finds is that the clinicians of the classical era begin to recognize and categorize diseases based on the complexity of the combination of symptoms and signs. No longer is each individual symptom an indication of one or another imbalance within the humors of the body. Indeed it is only through the non-referential stability of this complex combination of symptoms that the designation and communicability of diseases can become possible and standardized within and between doctors or clinicians. I would argue that this process of combination can just as well be seen as a configuration. It is the taking of existing and available elements such as symptoms and signs and

their configuration (reordering, structuring) into a new object. It is indeed within the complex combination of the functional interaction that we can see the tekhnē of what Derrida calls 'configuration'. It is this 'functional interaction that will serve as a preliminary tekhnē for the case study at hand. It is within this complexity of combination that we see an inventive tekhnē that then became a rule. But no functional interaction is the same as another and it is my goal to trace in detail the particulars which gave rise to the mystery illness.

What I have so far outlined in this chapter is not complete and the theoretical labour will continue in the following two empirical chapters. It is also impossible and impractical to continue such an investigation solely through the theoretical real of abstraction or through some sort of philosophical thought experiment. In such a theoretical exegesis, what would happen is that after some time we would arrive at a dead end with no real conclusion or result. As we navigate this space between the particular of invention and the universal of the rule, we would wander ever more closely to that point where they meet – where the inventive tekhnē becomes a rule of formation for the creation of further statements. But as with the problem of the particular and the universal which numerous great philosophers have dedicated their entire life's work to resolving and as with a mathematical limit where we can infinitely approach a number without actually reaching it, there exists that puzzle piece that cannot be accounted for; that paradoxical space which neither belongs to the particular nor the universal.

The Greek philosophers called such a paradox and such a liminal space aporia – that space between the particular and the universal, between form and content, between the base and superstructure[16]. A similar aporia exists within the case study of the mystery illness; as we bring the inventive tekhnē and the structure of the rules of formation closer together we will find a puzzle piece that is either missing or will not fit into the order of things. However, I would argue that the method of a careful and extensive historical, textual analysis will bring us closer to grasping and understanding such aporiatic elements than a purely theoretical treatise ever could.

[16] Philosophers of the Frankfurt tradition, like Adorno and Marcuse, have pointed out an inconsistency within what they called 'automatic' Marxism where it is often taught that the base (mode of capitalist production) completely determines the superstructure (the ideology of the society). While many consistencies can be found in regards to such a dialectic, Marcuse famously pointed out that there are aspects of our civilization and our venture towards the virtues of 'culture' (like our value for the beauty of art) which cannot be accounted for completely by the base, hence, an aporiatic element within the base/ superstructure dialectic.

Now that we are armed with all the conceptual legwork that we need to further the analysis, I want to briefly cover one other matter: the interpretation of the medical journal article. Primarily written by clinicians and medical doctors for an audience of fellow professional colleagues, it has long been seen as a very closed off body of literature with many barriers for the layperson reader. So how does one deal with such an obstacle? Besides staring at the journal articles until things made sense, the following short section will detail several other important considerations to take into account when analyzing medical journal articles.

2.3. The Analysis of the Medical Journal Article

As I began to read medical journals in order to find source material for this project, I was immediately confronted with three questions: (1) How do I overcome the boundary of the technical medical language used in these journal articles? (2) How do these boundaries impede my understanding and ability for interpretation? (3) Ultimately, what am I looking at and looking for in these articles?

The highly-technical language used in these journals is a specification of the more general biomedical discourse. It is, nevertheless, a language and operates much in the same way as any other 'transfer of temporal properties of the voice to the special properties of inscribed marks' (Ricoeur 1976, 42). More precisely, the ultra-technical language of the biomedical discourse can be called a linguistic 'system for possible statements, a finite body of rules that authorizes an infinite number of performances' (Foucault 2011, 30). What precisely makes it a discourse is this 'finite body of rules'. The infinite nature of possible statements precludes a finite body of rules which make it possible to create new statements and create the infinite series. It is precisely the study of these rules which constitutes a discourse analysis. As such, the complex technical language in question is not so much at issue as the way in which the rules which govern this language operate. Specifically, by what formative rules are statements created within this discursive formation? The two approaches, one of a linguistic nature, the other of a discursive , are different and the technical aspect of biomedical language does not, to the same extent, hamper the discursive analysis as this analysis does not directly focus on the outmost technical details of immunology and medicine – it simply recognizes these as vital elements of this specific language as the reality of language is not contained exclusively within the different words/ phrases of a language as is evident by our ability to create new words and new meanings for existing words.

The decipherment of the technical language is not then an issue. With this issue resolved how is one to approach the medical journal article? When I began to study these journals, I was at first, and as expected, perplexed. However, after some time and after reading enough of these articles, I began to see the communicative property of these journals. At their core, the journal's main value is to create the denotative statement – a scientific one which says 'what is' and how that 'what is' is. However, at the heart of discourse – of what Ricoeur calls "the dialectic of the event and the propositional content" (Ricoeur 1976, 14) lies the interlocutionary act – as at the heart of discourse exists the idea that "it is addressed to someone" (Ricoeur 1976, 14). "This presence of the pair, speaker and hearer, constitutes language as communication." (Ricoeur 1976, 14)

Who is this hearer in the medical journal? It is not the patient, for the patient is not able to grasp the complexities of the technical language. It can only be other doctors – others who are able to communicate in the specific phrase universe.

Does communication exist within the logic of denotatives? Yes, but within the journal article, a separation occurs between this dissemination of denotative facts and what I will call tentatively, theory building. The separation between the two is quite distinct. If not, as sometimes happens, it is not stated by the authors, there is a clear separation to be seen/ interpreted by the separate sections of the article.

Of particular interest for this study are the 'Discussion' sections of these articles. This is where I have noted the clearest form of interlocution – the clearest and least technical form of 'things being said' in a way that is intended for others within the specific discursive formation and genre of discourse. It is these discussion sections that I intend to carefully analyze. It is in these sections that the ultra-technical jargon can be disseminated and depressed. Underneath it, what remains is theoretical and, as I will show, interpretable.

Chapter 3. The CMV Hypothesis

3.1. Section 1

For a disease to be defined and designated – turned into a form of positive knowledge – it must be recreated as a stable discursive function. That is, it must become communicable. It must be stabilized so that it can be spoken about; used in speech, exegeses, and arguments. Furthermore, this stabilization and designation – this transformation into positive knowledge – depends on its commensurability with the technical field of calculation and mathesis. Not only must it be spoken about but it must similarly be a thing open to probabilistic thought and to prediction. How then is the discursive function of the disease created? How is it that one comes to speak of it intelligibly?

Undoubtedly medicine has changed to a great extent from the vivid description which we have seen in *The Birth of the Clinic* to the ways in which modern medical institutions train doctors to designate functional interactions of signs as symptoms in either the classroom setting or in the practicum of the clinic. Still, disease remains a field of language where symptoms first have to be articulated vis-à-vis each other for the sign of a disease to appear within a *de jure* visibility of the medical gaze (with the conditions of discursivity and calculability) as, what Foucault terms, 'a fruitful analogy' with which a clinician is able to distinguish one disease from another. And while the technical/ technological means by which disease is articulated into such a 'fruitful analogy' have changed and have become arguably more complex (with biomedical language similarly becoming more technical), the goal of articulating a disease into discursive positivity has remained the same – to name it, to be able to distinguish between it and other diseases.

We must also ask and answer the question of whether, as Foucault suggests, the clinic disassociates itself from the task of seeing "the essential truth beneath the sensible individuality". To put simply, is the patient simply the space within which the clinician fights the disease, or is

the patient – that sensible individuality – still very much connected to the designation of the disease (perhaps in a way that differs today to that of the Classical period). In which way would it be connected? What connections exist between the patient and the disease?

Such questions can only be answered if we consider the medical gaze in relation to the object it invents and the process by which it does so. Indeed, the inventive process here has to begin with the existent. We shall look at how the process of observation crosses over into the process of diagnosis (designation and naming). We shall see how diagnosis depends on the way that observation of the patient is carried out. I will work through the process by which the different symptoms and diseases are transferred into the functional interaction and the thought gradation by which the new fruitful analogy is invented. The reader should keep in mind that what is meant by observation should not be thought of strictly as pertaining to the sense of sight. Observation in the clinical setting includes many different things such vital signs, the results of different tests such as blood tests or biopsies, and of course can include other information from the patient's history – their life, relatives, relations, travel, etc.

Ultimately, we will see that it is not the different elements/ internal/ external causes that are at stake. It is the function by which they are configured and combined. It is the function by which these different causes are transferred into the analogy of the disease. It is not the causes themselves that constitute the invention of the disease but the operative tekhnē – thought gradation – that orders them into a given configuration which constitutes the object of invention In other words, the representation of the object does not consist of the culmination of signs accorded to it but of the functional interaction which orders these signs in relation to each other. It is that functioning mechanism which is able to order and reconfigure the existent (signs/ external causes/ internal causes) and this is what constitutes the representation of the object.

How does this functioning mechanism/ tekhnē/ functional interaction develop? Is it a creation developed elsewhere which is then transferred over into the discursive formation/ genre of discourse? Or is it something that develops internally within the discursive formation/ genre of discourse alongside its initial murmurings? Is it a combination of both of these processes acting concurrently?

Let me now end this brief introduction with a quote from Paul Ricoeur on a principle of discourse and the discursive formation,

> "Now, no discourse can claim to be free of presuppositions, for the simple reason that the conceptual operation by which a region of thought is thematized brings operative concepts into play, which cannot themselves be thematized at the same time. No discourse can be radically stripped of presuppositions; nevertheless, no thinker is dispensed from clarifying his presuppositions as far as he is able." (Ricoeur 2003, 303)

And if we think about it, the above is true for the smallest of discourses – those between two friends – to the largest of discourses – those of biomedicine.

3.1.1. Observation

> "In our present state of ignorance, some frank speculation seems permissible"
>
> -Durack, D. (1981)

David Durack is the author of one of the articles we will look I will look at later. It is interesting that Durack uses the word 'ignorance' to describe the present situation. Ignorance precludes that the truth of the matter is available but hidden from view due to some blinding smoke and mirror. However, I would say that more accurately put, the truth of the disease as we know it now did not yet exist then. It has not yet been created as its possibility of existence. Thus, the discourse within which it can gain this possibility does not yet exist. What existed then was a different discourse and different possibilities of what could be developed into a hypothesis - what could be invented as the theory of the disease.

As Durack states, this frank speculation or theory building is a process which is necessary in this period of unknowing. But theory here is not something which is new and original. As we shall see, it is a combination and conflagration of existent things – existent processes and correlations which are combined with existing factors to invent an explanatory function.

The purpose of this section is to start bringing out those elements which were noted by the clinicians. What observations were initially made? What was, in a sense, 'accented' and what was not? Before long, we will find that what was accented was the presence of sexually transmitted diseases. We will see that venereal disease was taken as a starting point in the investigation of the mystery illness. As surely as homosexuality itself was thought to be anything but a coincidental part of the puzzle, so were existing sexually transmitted diseases thought to

contain the truth of the mystery. Through them, in them, in some previously un-thought of configuration was to be found the pathogenesis of the disease.

The major proponents of this theory were also the same ones who can be said to have begun the dialogue (but not yet discourse) on the mystery illness and therefore undoubtedly had profound effects on the direction and focus of the discourse. Three journal articles mark the beginning of a dialogue: "Pneumocystis Carinii Pneumonia and Mucosal Candidiasis in Previously Healthy Homosexual Men" by Michael S. Gottlieb and colleagues, "An Outbreak of Community- Acquired Pneumocystis Carinii Pneumonia" by Henry Masur and colleagues, and "Severe Acquired Immunodeficiency in Male Homosexuals, Manifested by Chronic Perianal Ulcerative Herpes Simplex Lesions" by Frederick P. Siegal and colleagues.

One of the first hypotheses to be presented as explanation for the leukopenia and the decreased function of the cellular immune system was cytomegalovirus (CMV). The Gottlieb paper was the one to most clearly argue this position. As abrupt prologue, the hypothesis can be briefly explained as follows: The decreased function of the cellular immune system in these patients was caused by frequent exposure and re-exposure to cytomegalovirus which gradually broke down the function of the cellular immune system and thereafter allowed opportunistic infections to develop.

There were, of course, problems with this theory: (1) was the sudden reactivation of CMV the cause of the decreased function of the cellular immune system or was it simply one of the symptoms caused by some other factor? (2) CMV being the cause behind the decrease in the function of the cellular immune system was mostly substantiated by a study where mice were exposed to near lethal doses of CMV pathogen which, as a result, decreased the function of their cellular immune systems. These problems were quickly raised by other clinicians who were in conversation with the authors of the CMV hypothesis. Ultimately, the hypothesis was dropped. However, what is of interest to this study is that this hypothesis was presented in the first place – it existed. But by factors did it exist? What outside elements created the space of possibility for this idea to be positive knowledge and to be presented as hypothesis?

3.2. Hypothesis

3.2.1. The Masur Paper and the Denotative Genre

While I will argue that the Gottlieb article is the one that needs to be looked at to understand the rule of formation for the creation of hypotheses as to the etiology and pathogenesis of the mystery illness, two other journal articles on the topic of immunocompromised homosexuals were published alongside the Gottlieb article in the same issue of NEJM: "An Outbreak of Community- Acquired Pneumocystis Carinii Pneumonia" by Henry Masur and colleagues, and "Severe Acquired Immunodeficiency in Male Homosexuals, Manifested by Chronic Perianal Ulcerative Herpes Simplex Lesions" by Frederick P. Siegal and colleagues. In this section I would like to briefly analyze these two articles and discuss why the ideas within them did not go on to constitute the inventive tekhnē and then 'rule' of the emerging clinical discourse. This is important to establish as it will strengthen the claim that the primary article responsible for establishing the rule of formation of hypothesis in this emerging discourse was the Gottlieb article.

First, I would like to begin with the Masur article "An Outbreak of Community-Acquired Pneumocystis Carinii Pneumonia" (1981). This article ascribes to pure denotation and does not go beyond this, what Lyotard and Wittgenstein would call a language game. It presents the facts of the 11 patients – their previous medical histories, symptomology, the immunologic tests performed, diagnosis, and treatment. By the standards of science that science instills on itself, this was the most 'truly' scientific article; doing nothing more than reporting observations and providing no claims and no hypothesis as to why eleven previously healthy men had suddenly developed immunosuppression, lymphadenopathy, and pneumocystis carinii pneumonia (PCP), a rare form of lung disease usually seen in those undergoing cancer treatment or other procedures where the patient's immune system is compromised in the clinical setting.

> "Eleven cases of community-acquired Pneumocystis carinii pneumonia occurred between 1979 and 1981 and prompted clinical and immunologic evaluation of the patients… Eight patients died. In the remaining three, no diagnosis of an immunosuppressive disease was established, despite persistence of immune defects. These cases of pneumocystosis suggest the importance of cell-mediated immune function in the defence of p. carinii." (Masur Figure 1)

> "Certain viral diseases common in the homosexual community, such as cytomegalovirus, can depress the immune response: yet, these viral processes have not previously been associated with opportunistic superinfection in this population. Moreover, in patients with evidence of cytomegalovirus infection, it is unclear whether the viral process was the precipitating cause of the immune depression or the result of reactivation subsequent to the initial immunosuppressive process." (Masur Figure 1)

> "Narcotic abuse has been reported to cause in vitro immune defects similar to those described here, yet it has not previously been associated with pneumocystosis." (Masur Figure 3)

> "Since homosexuals are suddenly contracting a variety of opportunistic fungal, viral, and mycobacterial infections, it seems unlikely that this outbreak has been due exclusively to a new virulent or resistant strain of pneumocystis. The high mortality rate also seems more likely to have been due to the immunologic lesion than virulence or resistance of a specific organism or to peculiarities of clinical management." (Masur Figure 4)

Right away what should be highlighted in comparison to the Gottlieb paper is the aim of the article. The Masur article can be said to be much more conservative in how much it is willing to speculate or theorize. For example, the concluding sentence of the above quote states what was already known, that cellular immunity is an important factor in defence of PCP (Masur Figure 1). There is something to be said of the genre of discourse here. It remains, to some extent, descriptive and denotative rather than explanatory. The article lists several possible scenarios but never goes as far as to favour one over another. For example, Masur does pay attention to cytomegalovirus as a cause but quickly dismisses it in saying that it might be just as likely that the CMV infection is secondary and caused by whatever primary immune dysfunction that occurred (Masur Figure 2).

Additionally, the team raises the possible association with drug use due to seven of the eleven patients having some history of drug use but similarly dismisses this too as being unlikely as no previous association between drug use and p. carinii had previously been observed (Masur Figure 3). Note that this dismissal of CMV as a cause is not done as a response to the Gottlieb article – that article is not cited. Neither is the 1981 Drew article on the Prevalence of Cytomegalovirus Infection in Homosexual Men cited or mentioned in the paper. However, even in the denial of the CMV cause, we find that silent interlocutor to whom Masur is responding – a pre-emptive step taken to silence that interlocutor before s/he is able to propose CMV infection as a cause. This points to the fact that awareness about the disproportionately high prevalence of CMV infection in the homosexual community was a very well-known topic in the medical

community. It seems to be a disease that was quickly raised in conversation when the topic of homosexual health was at hand.

The second thing to note is the argumentation style of the article. As soon as it proposes CMV as a possible cause, it swiftly rejects it. As soon as it proposes drug use as yet another possible cause, it swiftly rejects that too. In doing so, it positions itself within a particularly marginal space within the emerging discourse. It poses no larger question or argument to the audience who would be reading the article as it quickly rejects and neutralizes possible cause after cause.

What the article does conclude is that this outbreak of p. carinii pneumonia is more likely due to the abnormal immune response within this population and not caused by a change in the nature of the pneumonia itself (Masur Figure 4). The pathogen did not become more infectious or more resistant to available treatments; rather, something had occurred within the homosexual population to make it more susceptible to p. carinii. Without, outright stating it at this point, the article hints at the possibility of something new but is not able to say whether this new thing is in the form of a new pathogen or if this new development has something to do with a genetic irregularity within the homosexual population.

This is as far as this journal article is willing to push the findings – no possible etiology or pathogenesis is stated, only that further research and observation are needed to continue. Here I would like to briefly outline a proposition. What ideas and what hypotheses become those foundational and formational elements of an emerging discourse? Look at the Masur article, its very design and genre of discourse is one of denotation. It does not aim to create or modify. It does not aim to put into some relation several of the elements it outlines and it does not modify any existing relations. Because of this approach, it is arguably the most objective and scientific article, but at the same time, it has a marginal impact on the forming discourse. What is then the ability of ‘good science’ to modify discourse? Even in the somewhat dated writings of Thomas Kuhn on the shifts in scientific paradigms, it is those anomalous findings and those outliers that eventually create the necessary conditions for the shift of paradigms. Good science does nothing more that concretize and re-affirm existing modes of thought. This is an important point to consider within this whole topic of invention and the formation of new discourses – in other words, what gets through?

3.2.2. The Siegal Paper

One of the many difficulties of mapping succession and the temporal vectors of derivation of discursive formations within this project is of course due to the length of time in question. Rather than mapping synchronicity and temporal direction across epochs (Classical period to modernity), this project, and the inventive instance it maps, concentrates on a timeframe of about eleven months. Within this timeframe, a complete tracing of ideas is difficult and next to impossible. What is at issue is not even the ideas themselves but the more practical level of the publication system from which the data of this project originates. At one level, the presented hypothesis depends on this mapping of temporal derivations, on another, the publication system makes the task impossible. A good example of this comes with one of the three major articles published in the 305th NEJM volume. I argue that the concept of the CMV hypothesis found in the Gottlieb paper is the one that goes on to be concretized as the rule for the formation of further pathogenesis and etiology hypotheses within the emerging discourse of the mystery illness. At first glance, the Siegal article, "Severe Acquired Immunodeficiency in Male Homosexuals, Manifested by Chronic Perianal Ulcerative Herpes Simplex Lesions," contradicts the hypothesis of this claim by presenting a pathogenesis of the mystery illness quite similar in concept to the Gottlieb paper. How am I to defend this then? Does my claim fall apart at this point?

This seeming contradiction can be explained and averted due to the paradox which emerges if we look at the respective reference lists of the two articles. While the Siegal article is published in the same issue and number of NEJM as the Gottlieb article, the Siegal article also cites the Gottlieb article as one of its sources[17]. How can this be if they are published in the same issue? This points to several of probable scenarios. It is possible that the two teams of clinicians had communication networks set up prior to their respective publications and knew of each other's work and dilemmas. However, while the Siegal article notes the Gottlieb article among its sources, no such reciprocity can be noted in the cited works of the Gottlieb paper. The relationship is then unidirectional; the Siegal paper is already one that is exposed to the CMV hypothesis – its drive is reactionary. Its ideas and its line of argument is already something that is informed by the Gottlieb paper.

[17] Note that the article cited is the one from that same issue of NEJM and not Gottlieb's earlier article contained within *Morbidity and Mortality Weekly* (Volume 30, Issue 20, 1981) which similarly outlines a stripped down version of the CMV hypothesis.

> "Reports of Kaposi's sarcoma and opportunistic infections similar to those that we observed (e.g. P. carinii, Cryptococcus neoformans, and cytomegalovirus suggest that our findings are part of a nationwide epidemic of immunodeficiency among male homosexuals." (Siegal Figure 1) (Referencing the Gottlieb paper)
>
> "A series of four previously healthy homosexual men with active cytomegalovirus infections complicated by P. carinii pneumonia has been reported." (Siegal Figure 2) (Referring to the Gottlieb paper)
>
> "Cytomegalovirus must be considered a candidate initiator of the immune defects observed." (Siegal Figure 3)

In the above, we see that the Siegal's team was well aware of Gottlieb's work. Rather than perceiving the Siegal paper as belonging to the same instance of time as the Gottlieb paper, we have to remember that the flow of thought does not conform to the publication regiments of academic and medical journals. Ideas may pre-exist their written form. The Gottlieb and Siegal papers may have been published within the same issue of the medical journal but the histories of the creation of each article are not necessarily temporally synchronous. Indeed, if we look at the *Morbidity and Mortality Weekly Reports,* the first mention of community acquired pneumocystis carinii pneumonia amongst homosexuals is made by Gottlieb and colleagues in the June 5th, 1981 edition of the reports (Volume 30, Issue 20). A larger report of similar cases from hospitals and doctors from all across the country was published a month after that on July 3rd, 1981 (Volume 30, Issue 25). This July 3rd edition is the one that includes a submission of data by the Siegal team. At the very least, we know that the Gottlieb team was the first, we know that they had, theoretically, more time before the publication in the 305th (24) volume of NEJM, and that a preliminary version of the Gottlieb piece was already available in the June 5th, 1981 edition of the MMWR where Gottlieb and colleagues lay out a preliminary version of their CMV hypothesis – a version that is spare on the detail but which already outlines the direction they will take and the final publication in NEJM.

What is mere concept within the Gottlieb paper becomes rule within the Siegal paper. Rather than thinking of the Siegal paper as the Gottlieb paper's contemporary, we must conceptualize the Siegal paper as perhaps the first one to utilize the rule of formation in question. There is more to say about the Siegal paper but at this point, to say more, we need to begin the discussion of the Gottlieb paper. A discussion at the end of which I will point out several more aspects of the Siegal paper which will allow me to show how the Siegal paper was already one

that used the new rule of formation created on the basis of the CMV hypothesis presented in the Gottlieb paper.

3.2.3. The Gottlieb Paper

The Gottlieb study was the first to be reported nationwide in the *Morbidity and Mortality Weekly Reports* (MMWR 1981, 30:20)[18] with a more complete version presented in the *New England Journal of Medicine* in the same year, a version also entitled "Pneumocystis Carinii Pneumonia and Mucosal Candidiasis in Previously Healthy Homosexual Men". What I first want to do in the analysis of this article is investigate those building blocks from which a discourse is founded. Remember that a discourse differs from language: a discourse constitutes the series or set of rules by which statements can form within the discursive formation. Let us then look at those rules which governed clinical observation and hypothesis creation. Similarly, we should also pay attention to what is said and what is kept silent - that reason which had to be clarified and that sensibility which need not be stated, which can remain silent as it is common (dead[19]) thought.

> "The fact that this illness was first observed in homosexual men is probably not due to coincidence. It suggests that a sexually transmitted infectious agent or exposure to a common environment has a critical role in the pathogenesis of the immunodeficient state. Sexually transmitted infections, including cytomegalovirus, are highly prevalent in the male homosexual community." (Gottlieb Figure 1)

What do these conclusions made in such a rapid succession state? The first two sentences alone claim that the illness was first reported among homosexual men because homosexual men

[18] It is interesting to note that the data published in MMWR 30(21) June 5th, 1981 looked at 5 patients while the paper later published in *The New England Journal of Medicine* (NEJM) on December 10th, 1981 presented the data of only 4 patients. It is hard to say why this 5th patient, described as "a previously healthy 36-year-old man,' is excluded from the NEJM publication. The only assumption I can make is that Patient 5 is not included in the NEJM article because he began treatment after the journal article was submitted for publication but could still be included in the MMW report due to the latter's much shorter and much more rapid publication cycle. Thereby, the 5th patient appears in the June 5th, 1981 MMWR and does not appear in the December 10th NEJM publication.

[19] Here I am referring to the distinction that Ricoeur makes between living and dead metaphor. Dead metaphor is metaphor that is buried deeply within speculative/ philosophical discourse – it is in a way normalized and detached from poetic discourse and made into objective structure. Dialectical structure and all of its numerous reincarnations throughout the centuries (even millennia) of philosophy and religion is a prime example of this.

are more likely to catch diseases – which we know because of the high prevalence of past or present CMV and hepatitis B infection amongst the homosexual population. Since this illness was first reported among homosexual men, it is most likely a result of sexual transmission. This idea of homosexual sexuality and its connection to disease becomes a vital element of the clinical investigation and in the attempt of the clinicians to make sense, in whatever capacity is available to them, of the immunodeficient state. We must also understand this process as an attempt to create a narrative for the disease. As I will show further, but as is also evident from the quotation above, evidence and results do not contain within themselves the necessary logic of connexion. Two pieces of evidence do not necessarily contain within themselves, by the very logic of their denotative and ostensive function, the *de facto* linkages that would make it evident how they connect. Such a logic is created post hoc by the clinicians who interpret the evidence they have with the aid of existing theories, hypotheses and already present knowledge which circulates within their respective field or even within the general social milieu of the time where knowledge of specific groups and subcultures exists at the level of stereotype or anecdote.

As these few sentences within the quotation create connexion and causation between the different elements immediately available, they also work to delimit the discursive field[20] - the rules which will govern the creation of statements within the discursive formation. And it is important for the reader to understand that it is not this one journal article, nor this one group of authors which created these rules. The rules pre-exist, they are simply being transferred into this discursive formation from elsewhere and are being invented/ reinvented here as the rules of this discourse – reconfigured and reconstituted. But this pre-existence of the rules must be clarified. As I will show, the rule which is transferred may not be originally a rule but simply a concept, element or some sort of inventive tekhnē – the concept or concept-as-element only becomes a rule vis-à-vis its connexions within the functional interaction.

The specific rules that I speak of are those that delimit the investigation of this illness as a homosexual illness and therefore as one of the sexually transmitted kind. Furthermore, those rules set out that the truth of the disease is to be found in the homosexual character. And that rule, by

[20] Lyotard would call this (in his reading of Kant) the faculty of the milieu: "this is the faculty which enables the territories and realms to be delimited, which has established the authority of each genre on its island" (Lyotard 1988, 131). 'Island' refers to the archipelago analogy which he uses in this section.

the very character of invention, delimits the scope of investigation to that which already exists; those existing elements like CMV and homosexuality which need to be combined in some new configuration to reveal the truth of the mystery illness.

> "It is known that after infection with cytomegalovirus, very high titers of the virus may be shed in the semen of asymptomatic subjects for more than a year." (Gottlieb Figure 2)

> "It is therefore likely that sexually active, young homosexual men are frequently reinfected through exposure to semen and urine of sexual partners. Such reinfection with large inoculums of virus before recovery from the cellular immune dysfunction induced by previous cytomegalovirus infection could conceivably lead to overwhelming chronic infection and immunodeficiency or Kaposi's Sarcoma." (Gottlieb Figure 3)

> "… [I]ncreased prevalence [of cytomegalovirus] may reflect the greater degree of sexual promiscuity among the homosexual than among heterosexual men." (Drew Figure 1)

Note the focus that is parted on the presumed sexually transmitted nature of the illness (Gotlieb Figure 2, Gottlieb Figure 3). Gottlieb and colleagues make the presupposition that the pathogenesis of the illness must be sexual because we are dealing with the homosexual population. Herein lies the core of the CMV hypothesis, the idea that especially promiscuous homosexual men are the ones who become ill with the mystery illness. They are infected with CMV and then are constantly re-infected through further sexual activity which prevents the body from reacting appropriately to the initial infection. High inoculums of the virus can 'conceivably' cause prolonged immune dysfunction. Note that the word 'conceivably' is an important marker within the quotation. It denotes that this idea is within the realm of possibility and is something accessible to human understanding. More importantly, it is something that is reasonable enough of a relationship to be understood by the audience of this journal article, other clinicians and doctors – a necessary process in the double movement of the signature and the counter-signature, communicability.

What the clinicians theorize is that cellular immunity can be compromised if the exposure to the virus (even a low grade pathogen like CMV) if sufficiently heavy (Gottlieb Figure 2). Literally, if exposed to (however, Gottlieb does not make it clear if the route is either oral or anal) a heavy enough viral load for long enough periods of time, the immune system breaks down – gradually weakened like castle walls under the steady and persistent bombardment of siege weapons. According to this hypothesis, the immune system of the homosexual gradually withers away from over usage.

As we can see, a key element emerges within this hypothesis, an element which allows the authors to bridge the linkage between the compromise of the cellular immune system and cytomegalovirus – the idea of homosexual promiscuity. The authors were fully aware that according to the 1981 Drew et al. study, "Prevalence of Cytomegalovirus Infection in Homosexual Men", an estimated 90 percent of all homosexuals had evidence of prior or current CMV infection (Drew Figure 1), so why were only some men immunocompromised instead of nearly all homosexual men? The reasoning was quite simple, the extent of one's promiscuity and the frequency of sexual activity. Those with the most promiscuous lifestyles were more likely to gradually destroy or wear out their cellular immune system with repeated exposure to CMV after the initial infection which caused immune dysfunction.

It is hard to answer the question of how the idea of homosexual promiscuity entered the formulation of this hypothesis. Following the archeological method, Foucault would argue that such searches for the geneses or the Original are futile. I suspect that part of it came from empirical findings of the time, the other part from popular knowledge of homosexuality of the time. And yet this is what is interesting about such positivity of knowledge – the state in which such modalities of thinking become common sense, when one need not reveal the propositional logic behind these conclusions because of the lack of opposition to them. Here, we can see the presence of what I had described in the previous section as an 'aporiatic element'. It is this transference from the outside of a fragment, of a puzzle piece, that cannot be accounted for by the discourse in question. We often accredit such things to the genius of individuals and yet we have to remember that ideas do not appear out of empty space in the human mind and similarly, that person must have consciously or subconsciously picked up that knowledge or thought elsewhere. Similarly, we as researchers are not able to grasp such an element as we are similarly confined within the boundaries and rules of the discourse we are studying. To attempt to better understand this aporia would require a separate study of a bordering discourse or even several of such discursive formations. At that point, the project grows to enormous proportions and begins to straddle the line between discourse analysis and an archeological description of the entire episteme of an epoch. It is would therefore be fruitless to figure out from within this discursive formation the twist or reconfiguration of thought which allowed the clinicians to bridge the divide between promiscuity as a binary indicator between heterosexuals and homosexuals and the transformation of that binary into a relative measure of promiscuity within the homosexual population.

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The Drew article on the prevalence of CMV in the homosexual population does note that the prevalence of CMV in the homosexual population might be due to the higher sexual promiscuity within the male homosexual population compared to the heterosexual counterpart but all it does is use this to account for the higher rate of CMV in the homosexual population. A particular and quite striking reconfiguration of thought must be made to position promiscuity, not as a simple binary indicator of homosexuality versus heterosexuality, but as an indicator of relative sexual promiscuity within the homosexual population signified by a presupposed higher CMV viruria in some men (who then became ill). These two usages of promiscuity are quite different in logic: the first states that homosexuals are more promiscuous, the second states that the more promiscuous homosexuals are more readily exposed to CMV – it becomes a function

which designates the degree of promiscuity. This transference of promiscuity from a binary indicator to something that functions as the internal logic of a hypothesis goes beyond resemblance, metaphor (analogy), or trope. As the concept of promiscuity enters the space of invention which forms this hypothesis, its role is transformed from that of 'element' or 'external cause' to that of the internal logic of the inventive tekhnē. Promiscuity becomes the function of the functional interaction being formed. At once as the whole CMV hypothesis hinges on the idea of perceived homosexual promiscuity, the idea of homosexual promiscuity hinges on its connexion to perceived CMV exposure.

Above all, what this new relation of promiscuity allows for is a re-configuration of the pathogenesis of a CMV infection. Much like today, it was already then known that CMV isn't particularly harmful. Perhaps the population estimates of prior and current CMV exposure were lower back then as Drew et al. only notes a rate of 47% for heterosexual males and 94 for homosexual males while more recent estimates from 2008 peg the figure at 80% for all adults in the United States but it was still known to be very common. Therefore, the idea of promiscuity in this CMV hypothesis allows the clinicians to transform a previously harmless and mostly asymptomic disease into one that is very harmful and potentially deadly.

What we see being done by the Gottlieb article, then, is the constitution of a functional interaction consisting of existing elements which are combined via functions that have been transferred into this discourse and invented again under the genre of discourse particular to this discursive formation (denotative *to* explanatory) – promiscuity ceases to be a simple binary element constituting the homosexual identity and becomes the crux of the explanation for the immunocompromise. At the same time as the elements are combined into a functional interaction via some function, one of the elements is made into a function and is reconfigured through the elements which it is combining. A story must be told of the things that exist and in doing so the explanatory function within this functional interaction must be cut, spliced and put back together again so as to fit the other elements within the interaction.

The first point that should be noted in the above is that the transferred function may simply be an element before it enters the inventive space of the functional interaction. The second point to be noted is that the function (or concept that becomes function) may not yet be a rule: rules can only become rules when they are further dispersed within the discursive formation. And

yet, this dispersal, this usage and re-usage, itself depends on the function acquiring recognition as positive thought. But if the CMV hypothesis was quickly discarded, how can this usage and re-usage occur? Do the relationships created by the functional interaction not need to be, in a sense, concretized to become this positive knowledge?

3.2.4. Stasis

We see now how this CMV model was invented as the cause of the mystery illness: through a process of combination and transference elements were combined and reinvented to make presuppositions, opinions, and judgement into positive clinical knowledge. However, this positive knowledge was discarded by the medical community in a matter of months. And yet, as I will show, the specific model for the functional interaction that characterized the CMV hypothesis carried over to further hypothesis and theory building. So how can a thing which existed for such a short instance be so profound in its effect?

Does the concept (but not yet 'rule') of a functional interaction have to be static/ concretized/ established before it can be available for transfer? This question is not very easy to answer considering I am analyzing this moment of invention. Can any thought process be concretized in a period of just about thirteen months (The period between the first few cases appearing and the name AIDS being designated) to be available for transference as a rule – especially when it is discarded as a possible explanation at the end of those thirteen months?

Derrida states that "invention is invented only if repetition, generality, common availability, and thus publicity are introduced or promised in the structure of the first time [inventive instance] (Derrida, 34)." Here is where I would like to explicate a bit of Paul Ricoeur's work on *The Rule of Metaphor*. In it, working from *The Figures of Discourse* of Fontanier, Ricoeur states that "the 'static' of relationships provides the foundation for the 'dynamic' of transferences (Ricoeur 1976, 63)." Indeed, a relationship has to be static/ concretized/ recognizable to be available for the outward transference of tropes/ metaphors/ analogies – out from it. It has to be 'static' to allow the transference of external and internal causes and to allow for reasonable substitution (reason of substitution). What does this mean for a conceptual framework that doesn't deal with metaphors as a whole but, specifically, with analogy? What

remains static? What is dynamic? First let us more closely look at what is meant by ‘static’ and then I will revert to the definition of analogy for further analysis.

What is to be said of stasis in the inventive instance which itself presupposes dynamism? Here, we have to again think of the scale of what is being studied. What Ricoeur means by stasis is of a different scale – it presupposes long lasting relationships which make repeatable transference possible. The tropes and metaphors which Ricoeur has in mind are much more an element of standardized speech than the metaphors and analogies I am dealing with. Indeed, the “shield of Dionysus” analogy is as old as ancient Greece, but it is one that is constantly recreated and re-visited in classrooms around the world as students read Aristotle – therefore it exists, therefore it is understood. In contrast, the metaphors and analogies that I will be looking at, are much shorter lived, much smaller and localized.

I would argue that the relationship between homosexuality, promiscuity, and cytomegalovirus within the CMV hypothesis disappeared quite quickly under pressure from arguments against it and alternative theories created by other doctors and clinicians – there were points of stasis between the dynamic of transference within the functional interaction of this hypothesis (indeed, this is what allowed the statement of this hypothesis to exist). And yet, the CMV hypothesis was short lived and quickly refuted by the medical community at large. The points of stasis created within it, dissipated as the hypothesis was refuted. However, I would argue that while the ‘empirical’ side of the CMV hypothesis was refuted, the conceptual part carried on and was transferred to other potential pathogeneses of the mystery illness; no longer as a concept that was reconfigured and invented once more but as sensible intuition –as a rule for the creation of further statements. The transference was analogical.

So the question of stasis remains. If the analogical transference of the inventive tekhnē to rule requires the static of relationships, where does that stasis exist in the inventive instance I am studying? Is this stasis that is required for transference contained within the functional interaction? This conclusion would be problematic. The functional interaction is only accessible to the perception of general relations only insofar as separate representations are accessible to understanding (Verstand). It is therefore impossible to have the general rule develop solely out of the inventive tekhnē in the functional interaction. There must be other referents, other points of stasis which allow for the transference to occur.

We have to remember that the ideas about homosexual promiscuity and about the higher prevalence of sexually transmitted disease within the homosexual population did not cease to exist after being used in the Gottlieb paper. These ideas/ concepts may have been reconfigured within the functional interaction of the CMV hypothesis but the original idea still remains intact after the fact of the hypothesis. We have to understand separately the existence of these concepts: how Gottlieb uses them and how they remain as positive or common knowledge. Even within Gottlieb's usage, we have to distinguish between the original idea which still remains accessible to Gottlieb and colleagues even after the invention of the CMV hypothesis, and the (re)invented form of the idea as used within the CMV hypothesis.

Two ideas can then coexist within a discursive formation of which the second is an invention of the first (transferred/ reconfigured). Archeologically speaking, discontinuities do not operate at the level of events presented here. Discontinuities, as Foucault writes, operate at the epistemic level where one discursive formation or type of positive knowledge is completely replaced by another (Foucault 2011, 189).

These concepts, then, remain active and positive within the forming discourse of the mystery illness. They remain as a vital cornerstone to the possibility of statements and hypotheses which can be presented within the yet to be discursive formation. But to be a discourse, a discourse must have rules for the creation of statements within it. The CMV hypothesis, as contained within the Gottlieb publication, was not yet a rule and only a concept. To be a rule, it must enter that level of positive knowledge- to be repeated and commonly available. It is these notions of homosexual promiscuity and sexually transmitted disease proportionality which act as the necessary stability for the transference of the invented CMV hypothesis concept to the level of rule. It is these relationships as positive knowledge which existed among the clinicians that provided the necessary conditions of possibility for the concept of the CMV hypothesis to become a rule of formation for statements within (what then became) the discursive formation of the mystery illness.

To summarize, what I have said up to this point is that the ability of the function of the CMV hypothesis to become the rule by which further hypotheses functioned cannot exist solely within the functional interaction of the CMV hypothesis – it must rely on existing positive knowledge as a point of stability for this to occur. In the next section I will argue that the

functional concept of the CMV hypothesis does become the rule for the pathogenesis of the disease. I will also show in more detail how existing elements/ presuppositions – categorical representations – acted as the static of relationships which allowed for the dynamic of analogical transference of inventive tekhnē to rule.

3.2.5. The Siegal Paper (continued)

What I want to do here is complete the discussion of the Siegal paper which I left off unfinished earlier. Now that we understand the CMV hypothesis I can make further claims to show how the Siegal paper was in fact one which already utilized the rule of formation invented by the CMV hypothesis. At the same time as it modified the existing CMV hypothesis and added a new element to the functional interaction, it maintained the internal logic of combination that we see in the CMV hypothesis.

> "The cause of the immunodeficiency disorder that we observed is undoubtedly complex. Viral infection, specifically in unusually heavy inoculum transmitted by enteric routes, may be an important initiating factor." (Siegal Figure 4)

> "A group may be specifically hyporesposive to HSV, perhaps because of their genetic background – e.g., HLA-D-linked immune-response genes. Heavy exposure to HSV could lead to chronic infection, and secondary immunodeficiency could then result." (Sigal Figure 5)

> "Still another possibility is that among men who are homosexual, some have a latent, broad-based cellular immunodeficiency that becomes clinically manifest only because of heavy exposure to certain pathogens in particular combinations." (Siegal Figure 6)

The hypothesis for the pathogenesis of the disease, at least a partial one, is thus stated. Let us briefly look at this model of the hypothesis. What is particular to it? What is transferred from the concept to the rule? Specifically, note the concept of promiscuity, how does it remain in the statement? For one, there is still the emphasis on higher than average viral loads and the assumption of the unusual, irregular, especially promiscuous homosexual who is exposed to heavy inoculum rather than normal or average rates of inoculum.

What is missing? The idea of CMV's role in the immune defects is made smaller, marginalized. Siegal states that it is still important to consider it as a candidate initiator but his

own study actually concentrates on herpes simplex virus (HSV)[21]. So, while he acknowledges the findings and the CMV hypothesis in the Gottlieb publication, he tries to produce a similar functional interaction with the substitution of herpes simplex virus – the generalized and ubiquitous 'viral infection' which, while devoid of information as to specifically which virus or combination of viruses, encompasses the idea of the homosexual body as a space of sexually transmitted disease. What is important from this is to understand the conceptual leap that is made by the author. What we see develop is the possibility of multiple viruses, in some functional interaction, being responsible for the immunodeficient state; hence, the ambiguity with the idea of the abstract, nonspecific 'viral infection'. It opens up a possibility of thought about the mystery illness as a combination of factors and not simply the work of one specific antigen; whether it is cytomegalovirus or herpes simplex virus.

What is new, what is added? The Gottlieb paper was somewhat unclear in the path of infection – it alluded to the sexual transmission and that "very high titers of the virus may be shed in semen of asymptomatic subjects for more than a year" (Gottlieb, 1982). Here, the Siegal paper makes it very clear that the route of infection is enteric (intestinal and, hence, through anal sex) (Siegal Figure 4). It is perhaps because the Siegal study concentrates on herpes simplex virus recovered from perianal ulcerative herpes lesions that this connection to anal sex can be 'objectively' stated as part of the hypothesis. But these perianal lesions are one side of the coin – by themselves they do not immediately indicate anal sex itself and can be an indicator of many things. So the idea of anal sex as a preferred sexual act among the homosexual community had to exist within the consciousness of the clinicians[22] as something that homosexual men do.

Another important addition that the Siegal article proposes, and which will be further pursued in other papers, is a genetic angle to the equation of the disease (Siegal Figure 5). Being careful not to state 'homosexuals', Siegal argues that 'a group' may have some genetic peculiarities which would make them more sensitive to the herpes simplex virus and more likely to have severe symptoms if exposed to a sufficiently heavy inoculum of the virus. In its structure,

[21] Note that cytomegalovirus (human herpesvirus–5) and herpes simplex virus (human herpesvirus – 1 or 2) are two different viruses of the same family; herpesviridae.

[22] While this may not seem so radical these days, Cindy Patton argues that anal sex was only popularized among gay men (due to the production and popularity of hard-core gay pornography) in the late 60's and early 70's (Patton 2014).

this hypothesis maintains, and above that, is constrained by, the CMV hypothesis by reaffirming the idea of the particularly sexually active homosexual man because it is only through 'heavy exposure' to HSV inoculum and a state of constant infection and re-infection due to it, that cellular immunity can be compromised to the extent that rare opportunistic diseases such as p. carinii pneumonia and Kaposi's sarcoma begin to develop. HSV replaces CMV as the combinative logic within the functional interaction and an additional element of genetic predisposition is introduced to overcome one of the problems with the CMV hypothesis – the relatively high rates of current and previous cytomegalovirus infection in the population as a whole even if the homosexual proportion of the population had markedly higher rates. The original hypothesis did not make it clear why specifically particularly sexually active gay men were becoming ill and not, let's say, particularly sexually active straight women who could similarly be exposed to high inoculums of the virus from the semen of many sexual partners.

The Siegal paper then makes one final hypothesis (Siegal Figure 6) which in many ways is an abstraction of the semi-genetic hypothesis. Almost as if understanding that the differences observed can be summed up to the particular patients that each clinical team observed, and that the idea of recurring HSV infection causing this immunodeficient state is just as likely as Gottlieb's version of cytomegalovirus infection doing the same thing, Siegal obfuscates the idea of any one virus such as CMV or HSV being solely responsible by stating that "certain *pathogens* in particular combinations" (note the plural form, my emphasis) are responsible for the immunodeficiency. Other parts of the hypothesis, like the necessity of 'heavy exposure' for the eventual dismantlement of the cellular immunity remain the same. Similarly, the idea that the immunodeficiency is caused by an existing and known pathogen remains intact in this hypothesis. What is added is the idea of a genetic predisposition which makes the particular 'group' have a weaker or delayed response to infections such as HSV (and presumably CMV due to the abstract phrasing of this final hypothesis). Note, that at this point, there is no conceptual space existing which could house the possibility of something brand new – of a completely unseen before virus. It cannot be proposed as rules of this discursive formation do not allow for it. And so to return to the short quotation by David Durack that I have in the introduction, it is not that the clinicians were in a state of ignorance which hid the truth, it is that they were in a state of discourse which did not allow for the possibility of the truth.

And so we begin to see a model emerge which uses the internal logic of the inventive tekhnē in the CMV hypothesis as a rule of formation and a rule of combination which allows different clinical teams to reconfigure the elements available to them in particular ways. As we shall see in further articles, the CMV hypothesis is outright refuted by some publications, but even in this refutation, these other articles continue to use the concept provided by the Gottlieb paper as a rule for creating their own hypotheses. CMV, as an element of this hypothesis model, is discarded, but this does not nullify the hypothesis model it was used to create. The hypothesis model, hence, becomes a thing around which further statements can be created; a skeleton which can house and connect different organs – a rule of formation for further hypotheses. This in itself poses interesting questions to the idea of science. On which basis is scientific knowledge generated? I have, so far, raised two important points:

(1) Just as the Masur article, purely denotative (hence, purely scientific) work is that which is not able to 'invent' or facilitate the possibility of a new discursive formation. To invent something like a new clinical discourse, that first inventive work must be able to go outside the purely denotative and present an aspect of speculation and explanation.

(2) At the same time as this first inventive work must invent something new, it does not invent anything new in the common usage of the word. As I have argued and as I have shown, the CMV hypothesis combined an~~d~~ existing and available elements in a new configuration; in a new functional interaction as per an inventive tekhnē – this inventive tekhnē perhaps being the newest thing presented and at the same time being something that has already existed. The idea of the especially promiscuous homosexual was not new and entered the inventive space from the outside, transferred into this developing clinical discourse from some other part of the social milieu.

But we must also understand the rule of formation within a localized discourse as a fragile thing. The static of relationships allows for the dynamic of transference, but as we shall see this static of relationships does not necessarily remain static and stable for very long.

Chapter 4. The Rule of Formation

4.1. Dispersion

I think it is at this point that we must begin to understand what it is we are dealing with. In an earlier stage of the thesis I claimed that I am not doing what would be considered an archeology. For the most part, this is true but it is also true that what I am doing can be considered to be part of an archeological analysis. It is interesting to note that in *The Archeology of Knowledge*, Foucault outlines multiple, non- hierarchical, levels of the existence of events within a discourse (2011, 189). At the epistemic level there is the substitution of one discourse for another or the abandonment of one form of positivity for another – this can be said to be a 'true' archeology: an analysis describing the plays of positivity within an entire epoch, describing entire structures/ institutions – their appearance and disappearance of entire forms of thought and thinking over the flow of centuries.

We have to remember that it wasn't the same series of homogeneous events that at every level of the discourse brought about change, discontinuity, or transformation in the discourse. Indeed, the events that occurred, the changes in the possibility of statements, their rules of formation, and the changes in types of enunciation may not conform to any coherent or

homogeneous structure[23] nor to the tenants of propositional logic. So theoretical stability exists, but the same theoretical stability does not exist for all levels of a discourse. Similarly, the rule for forming hypotheses which will be described in the next section cannot be thought of as belonging in principle and in concept to any overarching discontinuity or transformation that occurred within the overarching medical discourse. What this work tries to do then is describe a statement group within a localized branch of the medical discourse and just one rule of formation for that group of statements while understanding that a multiple rules of formation at different levels of a discourse pertaining to this mystery illness.

Another related point has to do with the 'tracing' described above. What does one trace? How does one trace? We try to trace thought and ideas and we usually structure this in some form of a temporal succession. Indeed, the time of the Idea has passed and we, social theorists, no longer find the idea itself as exciting or interesting – the object itself is no longer the center of attention, only its process. We are no longer shocked that something is, rather, we are more interested in how it came to be and what preceded it – how it developed into what it is.

4.1.1. The Durack Editorial

When I first embarked on this whole project and investigation, the Durak piece was one of the first that caught my eye. Its style and form is drastically different from the other medical journal articles I looked at. The best I can do is describing it as being much freer from the format and constraints of the 'normal' medical journal article format. In this, it is much more 'readable'. This will be most notable in just how much of the article I quote in this section and try as I might

[23] This is perhaps the most common/ popular and ill-informed claim thrown at what has been labeled post-structural or post-modern thought. Claims like "post-modernists believe in nothing and don't believe in any theories" are all too common but quite frankly belong closer to structural and ideological thought of the early 20th century (the Fascist motto of "nothing is real, everything is permitted" comes to mind) than to post-structural thought of the 60's and onwards. Indeed, we think theoretically, and indeed we conceptualize but we do so with the precaution that there exist multiple levels of phenomenon and one single model reduced ad absurdum cannot account for or explain society in its totality at all levels of its existence. However, we accept this as a positive quality and understand that even this seeming chaos of differences can be tracked and that these different levels with their different systems/ theories/ concepts/ ideas/ thoughts interplay to effect and change the world realistically/ profoundly/ materially. Therefore, it is absolutely not necessary to try to commensurate all of these differences along a common model that can simultaneously account for every level of life and thought. Let the differences be, it is precisely in the culmination of their difference that we can see societal and epistemic changes take form.

to cut down on this, there is just too much valuable information here to ignore. While still being a 'medical' article it manages to be so without a heavy reliance on highly technical biomedical language. In this article we also see perhaps the clearest example of the author refuting the CMV hypothesis while at the same time using the concept of the CMV hypothesis to create a new hypothesis. What we also see is the new hypothesis utilizing a number of new elements including Kaposi's sarcoma, 'recreational' drugs, and biological predisposition.

Let us first look at a series of quotations that lead to the refutation of the CMV hypothesis:

> "Male homosexuals are at an increased risk for the acquisition of common viral infections, including those caused by hepatitis B virus, cytomegalovirus, and Epstein-Barr virus, and viruses can cause immunosuppression." (Durak Figure 1)

> "A search for the origins of this unusual neoplasm (KS) brings us back to cytomegalovirus, which has been implicated as a possible etiologic factor on the basis of serologic studies. The final answer on whether cytomegalovirus is a cause or an effect in this setting will carry important implications for tumor biology." (Durack Figure 3)

> "The cytomegalovirus hypothesis suffers from an obvious problem: It does not explain why this syndrome is apparently new. Homosexuality is at least as old as history, and cytomegalovirus is presumably not a new pathogen. Were the homosexual contemporaries of Plato, Michelangelo, and Oscar Wilde subject to the risk of dying from opportunistic infections?" (Durack Figure 4).

> "Present indicators are that we are seeing a truly new syndrome, not explainable simply by failure to diagnose earlier cases. Therefore, we must suspect that some new factor may have distorted the host-parasite relation. So-called, "recreational" drugs are one possibility." (Durack Figure 5)

> "Users of amyl nitrite are more likely than non-users to have had hundreds of sexual partners and to contract venereal diseases. Preliminary data indicate that this "liberated" subgroup may be at highest risk for immunosuppression." (Durack Figure 6)

Durack's article begins with much of the same data that we have seen earlier. It establishes that the homosexual is much more prone to different infections than the heterosexual. However, much like the Siegal paper does at the very end, Durack, already recognizes that it would be foolhardy to name just one 'common virus' as the sole cause of the immunosuppression – a multitude of options (and possible interactions) exist as the medical gaze, long ago, has long

ago identified the homosexual body as a space of numerous diseases and has even created terminology such as ‘gay bowel syndrome’ to group and categorize some of them.

In a move that both rejects and recognizes it, Durack points out the obvious flaw with the CMV hypothesis (Durack Figure 2, Durack Figure 4). Cytomegalovirus may not be the cause of the immunosuppression but simply one of the infections which is reactivated by the initial immunosuppression. Considering that over 90 percent of homosexuals are or have been infected with cytomegalovirus, why are only some getting sick? Additionally, the virus is quite common amongst the general population which further complicates the CMV hypothesis. There is also the added complexity of the role of Kaposi’s sarcoma (KS) (Durack Figure 3).

I have not talked much about this other issue of Kaposi’s sarcoma up to this point but it was another major factor in the mystery of the new illness. Proponents of the CMV hypothesis argued that it was possible that cytomegalovirus was connected to the neoplasia – that cytomegalovirus could, in fact, cause the growth of the tumor. This was due to the fact that clinicians were able to find CMV titers within KS tumors. Due to the connections between homosexuality and cytomegalovirus which we discussed in the previous chapter, the directionality of this etiology seemed at the very least plausible at the time. As with many things of this time, it is not the case. The Kaposi’s sarcoma is an opportunistic neoplasia that develops because of the immunosuppression caused by HIV and not due to cytomegalovirus even if CMV can be detected within the KS tumors. For the clinicians, the role of Kaposi’s sarcoma in this mystery illness was a puzzle onto itself.

To fill the void created by the refutation of CMV as the primary etiological factor, Durack proposes a new element which may be factored into the equation of this disease; drug use amongst gay men (Durack Figure 5). The issue of the ‘new’ is a topic onto itself and will be discussed later. Here, consider what is new? Is this element of drug use ‘new’? Can the medical enterprise by the very limit of not being able to step outside the horizon (der Horizont for Husserl) of experience, hypothesize a new? What is the process by which it can do so? What is the stage at which it can reach out and include within an explanatory statement something which does not yet exist? What we find here is not a ‘new’ factor in the sense of the totally new, but a ‘new’ factor in the sense of combination and reconfiguration. The cytomegalovirus element of the CMV hypothesis is discarded by Durack and yet the framework exists – something ‘new’ must be

added. Note that the 'recreational drug' that Durack is referencing here is amyl nitrites or as they were also known 'poppers' (Durack Figure 6). This drug is primarily a muscle relaxant. It causes a several minute long head rush (or period of euphoria) and was also used to enhance sexual intercourse. We now know that due to its properties as a muscle relaxant it was used during anal sex as it could relax the sphincter muscles. However, we also need to remember that it is unclear in what capacity Durack and his team imagined the use of 'poppers'[24] – for what purpose and in what situations.

Remember that the element of CMV had a complex role in the CMV framework. Within the functional interaction of the CMV hypothesis, promiscuity was transformed from a simple indicator of homosexuality to an indicator of especially promiscuous homosexuality. A total reconfiguration of promiscuity as binary indicator to promiscuity as the function of the interaction had to occur – from a binary indicator to an indicator of relative promiscuity within the homosexual population. Now that CMV is absent from the functional interaction of this hypothesis, another element takes its place. Note how this new element is connected to the function of promiscuity.

Durack was not the first to suggest drug use amongst homosexuals as a possible etiologic factor; this suggestion can be seen in the Masur paper. Even the Siegal paper points out the heavy rates of drug use amongst some of the patients they looked at. However, this is the first instance where drug use is included in a hypothesis and placed in relation to other etiologic factors. It becomes an element which indicates relative promiscuity. It is reasoned that those homosexual men who are the most sexually active are consequently also the ones who use the largest amounts of 'poppers'. Because of this, these particular men are the ones whose immune systems are gradually compromised by the usage of the drug. At the same time, they are also the most sexually active homosexuals, and are consequently exposed to a large number of low grade pathogens. At this point, Durack doesn't make it clear if it is only the high usage of the 'poppers'

[24] The Special Report published by the CDC (1981) states that "the interest in a causal role for inhalants containing amyl nitrite or isobutyl nitrite or both ("popper", as they are commonly called) stems from the hypothesis that they are used as sexual stimulants or recreational drugs by some homosexual men." Note that his report was a synthesis of all existing information on the issue and even they could not detail the specific of amyl nitrite use or were to prude to go into such details. Also note that poppers were not something specific to the gay club scene. They were widespread all across the club scene of the day and were actually popularized during the 70's disco phase.

that are responsible for the weakening of the cellular immunity or whether it is both the high usage of drugs and a high exposure to low grade pathogens which is the cause. However, by this connection of poppers to promiscuity, the original function of promiscuity is maintained. In this new hypothesis, promiscuity still designates the degree of promiscuity in the homosexual community. This degree of promiscuity is now combined with adjuvant drug use and … (I'm not sure what the idea is), via adjuvant drug use, within the homosexual community where the presence of immune dysfunction and more exposure to low grade pathogens. Again, the function of promiscuity is maintained via its connection to drug use just as it was maintained via its connexion to cytomegalovirus in the CMV hypothesis. CMV takes a lesser role in the functional interaction alongside other low grade pathogens. What happens next is the statement of Durack's hypothesis in a more complete form. In the following, he combines what he stipulates himself on the idea of 'poppers' with already existing thought on the role of viruses in the immunosuppression.

> "In our present state of ignorance, some frank speculation seems permissible. Let us speculate that the combined effects of persistent viral infection plus an adjuvant drug cause immunosuppression in some genetically predisposed men. During the early stages, patients may have only a nonspecific illness and minor infections such as thrush. Then Kaposi's sarcoma may develop as an opportunistic tumor (perhaps cytomegalovirus-induced), which is set free by the failure of immune surveillance. Finally, as the defect in cellular immunity becomes progressively more severe, serious opportunistic infections develop. According to this hypothesis, several factors may interact to form a final common pathway of immunosuppression, which then leads to both neoplasia and infection." (Durack Figure 7)

In this complete rendition of his hypothesis, Durack clarifies the earlier ambiguity I noted. It is indeed a combination of both 'poppers' and of constant viral (re)infection which gradually tears down the immune system. Additionally, there is also this idea of 'genetic predisposition' amongst certain homosexual men which Durack introduces here to his own article. We saw something similar in the Siegal paper where that team proposed the possibility of 'a group' having particular genetic abnormalities that cause their defences to not respond the same way to herpes simplex virus infection. Here, the specifics of this genetic predisposition are not stated. It is not clear whether the genetic predisposition leaves the immune system of this group of men more vulnerable to the effects of the drug, or whether it is a genetic predisposition is something that results in a weaker immune system overall.

Durack notes that the immunosuppression may develop in 'some' genetically predisposed men. This tells us that Durack isn't arguing that all gay men have such a genetic predisposition. The addition of this genetic angle can specify that within the group of homosexual men, some have a genetic predisposition which gives them a weaker immune system. Some of these men with the genetic irregularities will also be those who are very sexually active and therefore at a higher risk of developing the immune dysfunction. This somewhat modifies the CMV hypothesis in terms of who is getting infected. Instead of singling out all highly sexually active gay men as the CMV hypothesis does, Durack's hypothesis further specifies who within this group is at risk. To some extent, Durack's hypothesis imagines a large group of very promiscuous gay men and only those who have the genetic predisposition become ill.

The idea of constant exposure and of the gradually withering homosexual body is still present in Durack's hypothesis. The idea of the especially promiscuous or sexually active homosexual is also there; it is in fact the presupposition that gives legitimacy to Durack's hypothesis (as well as Gottlieb's and Siegal's respective hypothesis). In other words, this idea of the especially promiscuous homosexual continues to maintain its role as a rule of formation for the hypothesis – it is the presupposition that gives legitimacy (makes them make sense) to this hypothesis and to others like Gottlieb's and Siegal's. The idea of the especially promiscuous homosexual, genetic predisposition to either HSV (in Siegal's paper) or some combination of pathogens and drug use (in the Durack paper) is that which gives some reason or logic to why these clinical teams have these patients with these strange and mysterious immune deficiencies in front of them. Beyond letting these clinicians bring some form of structure to why these symptoms are being seen in homosexual men, it is giving them a way to conceptualize why this is being seen in only some men and not in all homosexual men.

The biggest reconfiguration within the Durack hypothesis occurs in the idea of the etiological factors directly responsible within the body for the compromise of the immune system. Note that Durack is using broad terms and does not specify which drug is in question, even though he exclusively concentrates on 'poppers' within his article. Similarly, he is unsatisfied with an explanation that either that or this single virus causes the immunocompromised. Instead, he states the broad term of 'viral infection' which could entail a single viral cause or a multi-viral cause. By using both of these elements as the etiological factors, he is aiming to create a more

admissible and valid model of the disease by covering as much ground in terms of what is plausible.

Rhetorically, this aggregation of all possible factors to form an increasingly more 'robust' model of etiology and pathogenesis it is something that is done in the medical sphere quite frequently. The different ways in which the different aspects of human life are seen as increasing the risk of cancer is a perfect example of this: smoking, drinking alcohol, eating fatty foods, too much sugar, too little exercise, unhealthy weight, environmental factors such as the exposure to sunlight, radon gas and exposure to polluted air are just some of the things that increase a person's chance of becoming ill with one or another type of cancer. And this list does not even begin to include different genetic markers which might increase a person's risk. As such lists are slowly generated through decades of research, they slowly begin to encompass an ever-increasing proportion of what it means to be human. Where current biomedical discourse on cancer situates the human life as a risk for cancer, what we see occurring within the discourse of the mystery illness is the attempt to situate all possible markers, known to the clinicians, of the homosexual lifestyle (and even something about homosexual genetics) as a risk for this condition. The clinicians are certain that the truth of the disease must be found within the truth of homosexuality. As such, they are trying to reconstruct, *ex post facto*, a sort of 'essential' or complete homosexuality through the continuous combination and reconfiguration of more and more different factors that they consider to be essentially homosexual. The etiology and pathogenesis was already known to the doctors from the moment they observed the first few patients, now the task for the clinicians was to describe the disease scientifically. The next set of papers I would like to look at continues this process of combining different elements into the functional interaction. These two papers approach this from a very particular angle adding on aspects which actually modify the combined etiological factors of drug use and of heavy exposure to viral pathogens by providing it with, as Lukács would say, a historical character.

4.2. Extension

4.2.1. The Morris Paper

I'm including this particular piece in the discussion because it transforms and invents the CMV hypothesis in a very interesting temporal way. Additionally, it also combines and

configures into its hypothesis the element of drug use in a similarly interesting fashion – a fashion that is starkly different than that of the Durack hypothesis but which, in this comparison, shows the potentiality of the inventive process and the scope of possible hypotheses – their direction and the available presuppositions.

> "Homosexual patients are continuously exposed to viral antigens via the oral-anal-semen route. Indeed, cytomegalovirus, and probably Epstein-Barr virus, can be transmitted via semen." (Morris Figure 1)
>
> "Cytomegalovirus interaction has been shown to be associated with an impaired cellular immune response secondary to the presence of suppressor cells. It is conceivable that continued exposure to these antigens may lead to a cellular immune paralysis." (Morris Figure 2)
>
> "Why are sexually-active homosexual men developing these disorders of immune regulation? The lifestyle of these persons has changed in the last 10 years leading to greater promiscuity and use of drugs. A long latent period of perhaps 5 to 10 years may occur before they develop impairment of immune regulation with consequent disease complications, possibly secondary to known or mutant viruses, or latent viruses." (Morris Figure 3)

The first thing we will notice is the presence of the CMV hypothesis. Not only is the concept of the CMV hypothesis transferred as rule here, but the whole of the hypothesis as well – but this will be addressed a bit farther below. First let us look at the other additions that this hypothesis invents and the additional elements it combines into the functional interaction. One will first note that the route of transmission is much more clearly laid out here. Where the Siegal paper talked of transmission via 'enteric routes' (which itself was more clear than what is to be found in the Gottlieb paper), the Morris paper more clearly spells out that the transmission occurs through oral and anal sex.

This idea of the withered homosexual body is, while employed, used a bit differently from the other hypotheses that we have looked at. But this hypothesis does not stop here and introduces a particularly interesting element into the fold – the extension of the pathogenesis model beyond the narrow scope of the temporally immediate.

Notice how the idea of time is used here. Time becomes the function of the interaction, modifying the withered body analogy. The time required for the development of symptoms becomes as long as a decade in this hypothesis. It is no longer about the infection and re-infection

within a number of weeks or months but becomes the issue of re-infection within years, and many years at that. The authors link the appearance of the disease, not just to promiscuity, but to promiscuity that has been developing and that has existed since the early 70's, a decade associated with the hippie movement, the sexual revolution, and, of course, free love. I hope it is clear to the reader that the presupposition of a temporal extension being transferred into the hypothesis of the withering body is of a grand scale. Indeed, it engages with the historical character of homosexuality and tries to articulate the disease as an outcome of the far reaching social changes of that time and of the changes in overall lifestyle.

Time, in this hypothesis therefore, slows down. The hypothesis proposes that it isn't the issue of the male homosexual having a lot of sex in a very short period of time, but becomes an issue of a long term lifestyle by which the very gradual summation of particularly gay sexual encounters destroys the immune system. The sex and promiscuity are not the only things to be reconfigured, however. This hypothesis also proposes that the gradual promiscuity be combined with the usage of drugs amongst homosexuals within this past decade to produce the immune dysfunction. Notice, that this idea of drug use as an initiating factor existed in other hypotheses such as Masur's and Durack's but here it is transformed and attributed the same temporal qualities as sex; it becomes a part of the process which sees the homosexual body as a part of a withering which began a decade ago.

Note that the concept of the CMV hypothesis is central here. The model of the withered homosexual body (being gradually depleted by a pathogen like CMV or some combination of pathogens) is still the crux of the argument. It is not rejected but simply modified – invented again, in a different way. As in the Siegal paper, there exists an option that more than one antigen is the cause of the disease. In the Siegal paper, it was the option of the herpes simplex virus (human herpesvirus 1 and 2), which the team focused on, combined with their admission that cytomegalovirus must also remain "a candidate initiator of the immune defects". This new hypothesis seen in the Morris paper supposes another member of the herpesviridae family – the Epstein-Barr virus (herpesvirus – 4). It is somewhat interesting that Morris notes this here. Already at that time, tumor virologists had established a link between previous Epstein-Barr infection and an increased risk of developing several different forms of cancer such as Hodgkin's lymphoma. Perhaps, without outright stating it, Morris is trying to hint towards some sort of

possible linkage to cancer[25]. As many of the patients also developed Kaposi's sarcoma it was perhaps hard for a clinician to suggest that these men were developing two primary forms of cancer simultaneously. A person becoming ill with two concurrent but different forms of cancer is known to happen but in extremely rare cases. Morris, however, does not expand on any of this, and only points towards the possibility of such linkages.

What is important is the non-specificity deployed by the term "viral antigens". It is the same ambiguity that is found in the Siegal paper but notice that it is not based upon the rejection of the CMV hypothesis like the one found in the Durack paper. The concept of the CMV hypothesis does not then only become the rule for forming further hypotheses through its rejection or nullification but through a process of modification which can include both rejection and additive modification. It is not exclusively, as it were, a synthesis of only the good parts from each body of work as is often touted in evidence based practice. The process of statement creation can either modify the existing hypothesis through the rejection of its lesser parts and the synthesis of its greater parts or through the acceptance of the whole hypothesis which is then modified and added onto.

Remember, that at this point we are not looking at finding the hypothesis which was the closest to predicting the 'truth' of the mystery illness as we understand it presently. We are not here to debate that some hypotheses discussed here were in fact closer to that truth than others. The very idea of 'closeness' is alien here because one cannot be close to something that does not exist yet. The understanding of HIV/AIDS that we have today did not yet exist at that time as the very conditions for the possibility of its existence did not yet appear. Therefore, we should not, at this point, try to evaluate this inventive process as progressive or regressive, as good or bad, as 'good science' or 'bad science'. It was science and that is it. In our age, our expectations of what science is and what it should be, is molded by popular media to a fantastic (read: fantasy) extent which does not conform to the existing state of science. Note that we are not looking at ancient

[25] While outside the delimitations of this research, it should be noted that later work did focus on the 'cancer' angle. Specifically, the HIV virus was discovered while clinicians searched for a possible new form of leukemia or lymphoma causing virus. On the American side, when the Gallo team discovered the HIV virus, they actually first named it the 'human T-cell lymphotropic virus – 3' or HTLV-3. HTLV- 3 had the same naming scheme as the two other viruses discovered by Gallo: HTLV-1 and HTLV-2.

conceptions of science, what we have here is a very recent development, even a contemporary one for some.

4.2.2. The Friedman-Kien Paper

> "It is possible that a single type of infection, a particular combination of infections, or the antigenic load presented by multiple infectious agents may represent an immunologic insult that could overload or overstimulate the immune system." (Friedman-Kien Figure 1)

> "The recent appearance of this disease may be associated with the changes that have occurred over the last 15 years in the lifestyle of homosexual men living in large urban centers." (Friedman-Kien Figure 2)

> "Use of multiple recreational drugs, especially the inhalation of amyl and butyl nitrite, available through noprescription sources, is also an important aspect of this changing lifestyle." (Friedman-Kien Figure 3)

Much for the same reason as we have seen in other articles, the central role of CMV infection is rejected here. Again, the authors point out that it is more likely that re-infection with CMV seen in many of the patients is secondary to some other primary dysfunction in cellular immunity. And yet, the basic idea of the disease has not changed with the authors noting a probable 'single type of infection', like CMV (but not CMV), or a 'combination of infections' through an 'antigenic load' which might be the possible pathogenesis of this disease.

I think what is interesting to note is a slight modification of the withered body analogy that takes place in this article. What was established in Gottlieb's work was the idea that persistent exposure to these low grade pathogens will gradually destroy the immune system and make it vulnerable to infection by a number of opportunistic infections. The Friedman-Kien paper proposes something that is almost an inverse of the withered body analogy. Here, persistent exposure 'overloads' and 'overstimulates' the immune system instead of gradually destroying it and wearing it out. This produces an idea of the immune system that is analogous to something like a car engine that can be 'blown' if run close to or at maximum rounds per minute (rpm) for too long or an electric capacitor that bursts if the surge of wattage is too strong. Effectively, the two forms of thought, the withered body analogy and its inverse, produce the same effect – an explanation for the shutdown of cellular immunity. It is interesting to note, however, that the functional interaction can be modified in this way but still remains intact conceptually.

Similar to how the Morris paper goes beyond the immediate and creates a narrative that extends the space of the disease historically to the time of Woodstock, the sexual revolution, and to the time of hippie 'free love', the Friedman-Kien paper also puts a lot of weight on the idea that this is an issue of overarching lifestyle and not just of recent or immediate circumstances.

In this Friedman-Kien piece, the timeline is extended an arbitrary 15 years (Friedman-Kien Figure 2) instead of Morris' arbitrary 10 years to denote, what they seem to think was (and they were not the only ones as the early 80's saw a strong resurgence of social conservatism) that controversial period in history where traditional American values were abandoned by the youth of that generation in favour of sex, drugs and rock and roll. It is evident here that this assumption or presupposition does not belong to this discourse but is transferred into it from the outside social sphere. In what form this knowledge operates outside the social sphere and how it was made available to transfer is hard to say. It may have been an unspoken, but at the same time understood by everyone, type of prejudice or it may have been something that was propagated quite publically in certain sections of the population by certain political and religious figures – more socially conservative sections of the population to which some of these clinicians may have belonged. I would just like to point out that even if it is the latter case, such a presupposition, even if not avidly practiced by all the clinicians, must have at least been understandable and comprehensible by all the clinicians to even be proposed as part of the hypothesis by Morris and Friedman-Kien.

Aside from the idea of promiscuous sex with multiple partners that is at the core of this hypothesis model in the form of constant and heavy exposure that, according to this addition of a historical character, has been occurring since the late 1960s, Friedman-Kien also notes that drugs are another important possible factor responsible in the deregulation of the immune system (Friedman-Kien Figure 3).

As with the other papers that we have looked at, this hypothesis tries to add on and transfer into itself a variety of elements in an attempt to produce a more robust or complete explanation of the mystery illness. At its core, the logic of the hypothesis model has not changed from that of the CMV hypothesis. I have tried to argue that this is because adding on and combining an ever larger amount of elements is the only thing that is permissible under the rule of formation governing the creation of statements within this discursive formation. This goes

back to a fundamental difference between discourse and language. Language allows for an infinite series of statements and combinations of words and syntax which we see in the unceasing production of new words the usage of which quickly spreads throughout society. On the other hand, discourse is a delimited space with a finite and set number of rules which regulate the production of this theoretically infinite language.

This means that, theoretically, this hypothesis model could house any number of unique functional interactions of different elements in particular combinations. However, following the theoretical framework that I have set in Chapter 2, this does not mean that any of these combinations invent anything new.

Knowing what we know now about the existence of the human immunodeficiency virus (HIV), it becomes quite clear that this early discursive formation is not capable of even imagining a wholly 'new virus' and as much as the clinicians working within it may try – as many existing and known elements that they may combine to try to explain the etiology and pathogenesis of this mystery illness – they would not be able to arrive at the idea that would point to 'a new virus' or for the need to research and investigate a completely new virus. The hypotheses would necessarily stay what they are; attempts to rationalize the disease from what is known about the homosexual – of combining the known factors and diseases that have already been established by the clinic as having an association with the homosexual lifestyle.

And yet, the discovery of that new virus did occur only a few years later. This means that somewhere between this early discourse and that almost simultaneous discovery made by the Gallo and Montagnier teams, a new line of reasoning and therefore a new discursive formation with a new rule of formation for new hypotheses was formed; the possibility of this acquired immunodeficiency syndrome being caused by a virus had to develop as well.

But first, what had to occur, is for this discourse and this rule of formation to run its course and disappear. Such a discontinuity or cessation of a discourse is just as nearly impossible to trace completely as a beginning or genesis of a discourse but I will now try to discuss the possibility of being able to anticipate the new (of something like a completely new virus rather than combing over known pathogens and diseases that the clinicians associated with homosexuality), of that blinking and faint murmur from which this present discourse began and that we can sense flickering in anticipation (non-Heideggerian) of a new discourse. We will look

at the particular turn of phrase and though that occurred in a paper by Anthony S. Fauci in 1982. Why do I want to look at this particular paper?

It is undeniable that figures with a certain gravitas can go a longer way in establishing or beginning a discourse than someone who is unknown or even just somewhat known. A good example of this is the environmental discourse: while scientists had clamoured for years that there were definite climate problems facing the world, it took former vice-president and presidential candidate Al Gore and his documentary *An Inconvenient Truth*, to popularize that topic and bring the discussion to the living room of a household. I would argue that with this new mystery illness, there too could have been a larger influence from people who have more 'weight' in the field. Anthony Fauci was one of these people with a bit more weight in the field. For example, just two years after the publication of the journal article I will be looking at, Fauci became the Director of the National institute of Allergy and Infectious Diseases (NIAID) – a prestigious posting indeed.

4.2.3. The Fauci Paper

> "Cytomegalovirus has been thought to be the primary causal agent in the induction of the immunosuppressed state…[t]he likelihood of frequent re-exposure and reinfections with cytomegalovirus within a confined group could conceivably lead to a state of profound and apparently permanent immunosuppression directly related to recurrent viral infection, as opposed to the clinically insignificant degree and duration of immunosuppression usually seen in hosts with a single exposure." (Fauci Figure 1)

> "However, a counter-argument can be made that patients who are immunosuppressed for other reasons, such as iatrogenesis, also have a high incidence of cytomegalovirus infections. Therefore, it is possible that the immunosuppressed state is caused by other factors and cytomegalovirus infection is merely a consequence of this immunosuppression." (Fauci Figure 2)

> "[W]hy homosexual men and why occurrence or recognition only as recently as 1979? With regard to the latter point, it now appears that beyond question this syndrome is truly a new disease." (Fauci Figure 3)

> "Some recent change, therefore, seems to have occurred within the unique epidemiological confines of the male homosexual population that has been expressing itself only over the past 2 to 3 years. Is there a new virus or other infectious agent that has expressed itself first among the male homosexual community because of the unusual exposure potential within this group?" (Fauci Figure 4)

> "Furthermore, because we do not know the cause of this syndrome, any assumption that this syndrome will remain restricted to a particular segment of our society is truly an assumption without scientific basis." (Fauci Figure 5)

> "Is this immunosuppressed state due to chronic exposure to a recognized virus or viruses? Is this illness due to a synergy among various factors such as infectious agents, recreational drugs, therapeutic agents administered for diseases that are peculiar to this population such as the "gay bowel syndrome," or is this illness due to a combination of all of these factors?" (Fauci Figure 6)

The paper starts out much like many of the other ones we have looked at: it refutes the foundational CMV hypothesis proposed by Gottlieb and maintains that it is likely that CMV re-infection in these patients is itself an outcome of some antecedent cause (Fauci Figure 1, Fauci Figure 2). After going over several other studies (all of which I have covered), Fauci proposes something quite pivotal (Fauci Figure 3): that all these cases can be identified as having distinctly similar combinations of symptoms and signs and therefore can be designated as a wholly new disease. Although it does not yet have a proper name, the combination of symptoms and signs has reached a point of oscillation within this discourse where it is visible and communicable enough among doctors to be designated as a unique object of discourse and receive its own identity and classification.

After establishing the claim that this was indeed a new disease, Fauci also begins to phrase the pathogenesis of this disease differently from how other authors have done this (Fauci Figure 4). We see him step away from the idea that the disease is essentially homosexual and has its beginning within and because of homosexuality. This disease has simply 'expressed' itself within the homosexual community first but exists and has existed outside it. Not only this, but Fauci further distances this new disease from the assumption that it is in and of itself an essentially homosexual disease by stating that there is no scientific basis to believe that it will not appear in the wider population (Fauci Figure 5) – that heterosexuals are somehow immune to it.

Noting this I want to draw back the reader from assuming that this is somehow a watershed piece that completely silences and mothballs the discourse we have been studying. Indeed, Fauci reiterates the combinative logic established by the CMV hypothesis and still presents it as a form of positive knowledge and insight (Fauci Figure 6). We cannot assume, therefore, that this piece is something completely new that transcends the existing discursive formation; it is still very much within it. But again, an aporiatic element appears that does not belong to the rules of this discourse. He does begin a dialogue that situates this illness as not something born out of the homosexual character which is strikingly different from the path of inquiry established in all the previous articles on the topic and he does present the possibility of there being "a new virus' which is behind this new disease. Again, as with the case of the CMV hypothesis and the transformation of the notion of promiscuity, it is impossible to discern the logic or origin of this aporiatic element from within this discourse itself as it is something that has been transferred from the outside.

However, notice what this notion, that the disease can be found outside the 'homosexual lifestyle', does within this discourse as it enters it. First, it allows a complete change in where and how the disease is situated. The connexion/ enchainment between this new notion and homosexuality is distorted. Rephrasing the disease as first being 'expressed' within the homosexual community had the potential (and only the potential for other authors as nothing of the sort actually happens in this paper) to enable the idea that a search for the etiology and pathogenesis of this disease can be launched outside the confines of the 'homosexual lifestyle' while all the previous papers have exclusively focused on figuring out the mystery illness within the confines of homosexuality.

This change in the positioning of the disease within the discourse is also something that may have allowed Fauci to propose the idea of a new virus. However, even such an idea already reaches out of the self-imposed constraints of this project.

Chapter 5. Conclusion

The goal of this project has been to investigate the early hypotheses created to explain the etiology and pathogenesis of the early 1980s mystery illness that we eventually came to know by 1982 as "AIDS." My goal was to understand the conceptual framework employed by clinicians of that time to create those hypotheses, and in particular, how their creations reflected their knowledge and attitudes towards homosexuality. I wanted to look, in detail, at the way their efforts to frame hypotheses that might generate 'objective' scientific knowledge was affected by different ideas of, and prejudice towards, homosexuality.

I chose a fine-grained approach to identifying the gradations of thought expressed in the public representation of their hypotheses. For this task, I developed a theoretical framework which combined elements of Foucault's archaeology with ideas from Derrida's deconstruction of invention; this approach allowed me to understand how the logic for forming these hypotheses was first invented and how this logic then developed into a rule of formation for creating further hypotheses striving to explain this mystery illness.

5.1.1. The Inventive Tekhnē

I would like to conclude now by revisiting the shift from my discussion of the rules of formation and of Derrida's notion of invention in Chapter 2 and my application and modification of these ideas in Chapter 3.

According to Foucault's schema of the functional interaction of the disease and according to Derrida's model of invention, several existing and known elements interact in some particular configuration that repurposes those elements and accords to them a new being and thereby invents a new thing – whether it is a new disease as is the case here or a new type of consumer of electronics. This 'reconfiguration', however, can be argued to belong to poiesis (the ability of the mind to imagine something new when it sees something that exists – one of the three parts of Dasein) and constitutes some particular inventive tekhnē by which these elements are

reconfigured. The question that this framework leaves us with is where does this inventive tekhnē come from? As we look at our human civilization and at all its achievements and inventions we can understand that this inventive tekhnē is not a thing that can be one and the same from reconfiguration to reconfiguration. While the particular of the inventive tekhnē may be unreachable as it stays to close to experience, this tekhnē may still have some sort of structure.

The CMV hypothesis at first resembles that structure of invention that is described within Chapter 2. We see that the symptoms and conditions which were first observed by Gottlieb and colleagues in the four (but at first 5) cases are used as a starting point for the creation of the hypothesis: the presence of cytomegalovirus infection, the leukopenia (signaling some disruption in the cellular immunity), and the resulting pneumocystis carinii pneumonia. These conditions and symptoms are then combined with other external elements like homosexuality and promiscuity. However, we can then see that this idea of a higher promiscuity within the homosexual male population as compared to the heterosexual male population is then transformed to signal a relative promiscuity within the homosexual population thereby allowing for the idea that those whose cellular immunity is being lowered and those who are consequently becoming ill with PCP are those who are the most promiscuous and sexually active. Here, the element of promiscuity is transformed into the inventive tekhnē which allows for the specific configuration of these elements.

Although I believe it is critical to the transfer of invention to rule of formation, I was not able to further describe this inventive tekhnē as I outlined invention via combination in Chapter 2. Derrida does not go much farther than simply describing this second form of invention as an ordering structure that appeared to reconfigure existing elements into some new invented thing. However, I make it clear through my analysis in Chapter 3 that this inventive tekhnē comes from no place other than from the group of elements which comprise the configuration/ functional interaction.

However, while I move closer to understanding the inventive turn of thought which allows for this combination, I do not completely forego the aporia that I have noted as existing within the logic of the inventive tekhnē. Part of inventive tekhnē I have been able to address: we can now understand that its structure develops out of the contents of existing and available elements by using a transformed version of one of these elements (“promiscuity”, in my case) as

the logic for combining all the other elements. However, here I encounter a limitation as it becomes simply impossible to further trace the turn of thought which transforms this relation of promiscuity and gives it that new form within this discursive formation. I have noted that the reason of this transformation in the logic of promiscuity is largely unattainable (and reason can be unattainable as it is not transcendental, as in philosophy, but is learned) from within the confines of this discourse. It is crucial to understand the reason of this limitation: further understanding this aporiatic element within the logic of the inventive tekhnē requires an inquiry into some neighboring discursive formation that existed during that time. It would be speculative to guess which discursive formation, but perhaps some linkage can be found in the issue surrounding clinical trials of a hepatitis B vaccine that occurred in the late 1970's and solicited gay men as participants. All that this project is able to do is further establish some structure of this tekhnē and show how this tekhnē then became a rule of formation.

We can see that the tekhnē created by the relation of promiscuity to the other elements remains within subsequent hypotheses. Cytomegalovirus may be replaced by other diseases or by the added element of drug use and 'poppers' but the whole hypothesis (functional interaction, configuration) still hinges on the relation of these different elements to promiscuity. With this internal logic of hypothesis building based on the idea of the withering homosexual body (gradually being destroyed by a pathogen or pathogens), further hypotheses are then able to add on or replace different elements while still maintaining this key relation to promiscuity and are still then able to produce some variation of either the withering body analogy or an immune overload hypothesis.

Importantly, and perhaps paradoxically, since the original hypothesis was almost immediately discredited, it is only through the further use of this inventive tekhnē in subsequent hypotheses that the invention stabilized. Here we see what I have described as a double movement of the signature towards the countersignature. I have tried to argue that only when the invention is recognized by another (in this case, other clinicians and doctors) is it truly invented. But since what we have seen is the outright rejection of the CMV hypothesis by other authors, what can I say about the meaning of 'recognition' and of 'countersignature'?

Does recognition only exist in a positive sense within a discourse? Can the CMV hypothesis only be said to have been recognized if everyone agreed with it or is rejection yet

another form of recognition? I have shown that some articles did agree with the CMV hypothesis and tried to improve upon it by adding other dimensions such as genetics or by adding a longer history to the idea of the homosexual lifestyle. Other articles, however, completely rejected the CMV hypothesis and the very premise that only one pathogen was involved in creating the mystery condition. But what we must realize here is that two different levels of a discourse are in question here. At the level of statements – the content of the hypothesis – the CMV hypothesis is rejected by other authors. At the level of derivation – the logic of the first hypothesis is maintained by the other authors. The countersignature, then, is not as simple as mimicking the original signature but the 'recognition' can operate at the level of human thought. That is, the countersignature can be evident by a change in human thought and thinking.

5.1.2. The Rule of Formation

The first theoretical goal of my project was to trace this inventive movement that occurred to produce the CMV hypothesis and then allow for the further formation of other hypotheses. The second goal was to establish the existence of a rule of formation for these hypotheses. The two goals, while connected, are different. What does the rule of formation regulate? What are its functions within a discourse? What is a rule of formation itself?

Note how the little and short lived discourse I describe operates. Once this principle of promiscuity is established, it does not necessarily put up many walls or barriers for the other authors. It creates a field, but it does not dictate to a great degree what enters this field. After the formulation of the CMV hypothesis, other hypotheses were by no means determined. Indeed, CMV was replaced by a variety of other possibilities, such as another single pathogen like HSV, a multiplicity of different pathogens, drugs, poppers, and some combination of poppers and pathogens. Similarly, we saw the addition of genetic notions which ranged from homosexuals being genetically predisposed to just some homosexuals being predisposed to being much more affected by these low grade pathogens than the general population. After that we also saw the addition of a historical element which put the whole disease in the context of the past 10-15 years as it tried to connect to the general 'tearing of the moral fabric' during the 'hippie' years – Woodstock 1969, drugs, and free love. This transformed idea of promiscuity did not even impede the formation of conclusions of etiology to either the withering homosexual body analogy or the immune overload analogy. As I have argued, the field of possible content here is of an essentialist

nature where more and more elements that were thought to belong to the homosexual character could have been endlessly added.

This means that the theoretical possibility of statement-hypotheses was endless within this regime. If this discursive formation was not silenced and if it continued to exist it would have produced even more hypotheses. The principle of homosexual promiscuity was not then an overarching and controlling thing in that it dictated how each hypothesis was to be formed. Neither did it set out a limit on the number or the content of hypotheses. Similarly, it did not say what kinds of elements and what number of elements were to be in each hypothesis. What this principle did allow for was the existence of these hypotheses and the structure of this principle allowed for there to be more than one hypothesis. In other words, this principle of promiscuity was the principle of the multiplicity of these hypotheses. It was the principle which allowed for the dispersion and oscillation of these hypotheses within the discourse – the ability for doctors to comprehend and to respond to each other's ideas. By this, the principle of promiscuity acted as the rule of formation for these hypotheses.

5.1.3. The Homosexual Character and the Subjugated Knowledge of Biomedicine

Early in Chapter 2, I referred to one of Foucault's lecture series books titled *Society Must Be Defended* and described what he means by subjugated knowledge. This concept is related to the whole discussion of power as subjugated knowledge has power, but at the same time, such knowledge can be argued to exist outside power. I mean this in the sense that subjugated knowledge is placed into a space where it is no longer beholden to even the power that placed it there. Subjugated knowledge is that knowledge which becomes a deep buried root of the positivity of some other group of knowledge. It is not questioned, it is assumed, and it is taken as a 'given'. At this same time, it acts as a cornerstone or a structural strut for other forms of knowledge. In this it is subsumed or, indeed, subjugated to form the positivity of these other groups of knowledge. As such, these other groups of knowledge are not able to reach back and uproot this subjugated knowledge or change it. Without it, these groups of knowledge would lose all positivity and validity as knowledge. Symbiotically, this subjugated knowledge would cease to have power once the groups of knowledge which it supports have been transformed or silenced.

Switching back to the case at hand, why did the clinicians and doctors first focus on finding the cause and nature of the illness in the homosexual character or lifestyle? Even Epstein notes it as strange that instead of following conventional procedures and beginning the search at the level of the microbe or virus, the search went straight to assuming that the cause is to be found within the homosexual lifestyle. In her new book, *Disease of Unknown Origin*, Patton (forthcoming) argues that there was already a close but not publicly know relationship between gay doctors, nascent gay health clinics, and the Center for Disease Control, a collaboration which was, until about 1978, developing national standards for STD care for homosexuals. In addition, the Hep B vaccine research had involved gay clinics and gay community members in the research venues knew of the project and believed the government and researchers were acting on their behalf. Because of this work on STDs by gay CDC doctors and because the two major health threats that the CDC had left to deal with at this point were STDs and tuberculosis, Patton argues that the biomedical enterprise was already accidentally positioned to focus on the issue of STDs and the homosexual lifestyle when those first cases of p. carinii started to appear – the conditions for the possibility to think and reason in alternative ways simply did not exist because of this pre-existing network of research and knowledge.

This is where we can see what sort of strength and power that instilled and ingrained knowledge can have. It was not questioned why the search should be conducted within the homosexual lifestyle. It certainly was not asked if the search should be started there. It was simply done. It was taken as a given. The nature of the disease would be found in what homosexual men do in their everyday lives. To say that this focus developed because the first 5 men to have the illness were gay and that was all that was needed to establish some sort of presumed association is a faulty argument. It assumes that the medical enterprise has not encountered cases of infectious disease before this. This case was very unique to be sure, but it did not strip away all the wisdom that modern medicine had collected up to that point. Following this logic, it would have been something in the character of the legionnaire or of the elderly veteran that would have caused legionnaire's disease. Medicine of that time had mostly been able to get past such haphazard associations of diseases with the different groups based on characteristics that were seen to define that group. Thereby, the cause of the Great Pox was not to be found in the despicable character of the French (or in the despicable character of Italians, from the viewpoint of the French), and the Black Death was not spread by Jews and Roma. All of these old ways of associating disease with a particular ethnicity, creed, religion, occupation, and so on,

had been abandoned in favour of explanations that relied on microbes, bacteria, viruses, or in the case of conditions like scurvy, vitamin deficiency. Even if a disease was strongly associated with a particular occupation, like cowpox was with milkmaids, the Germ Theory of disease made the notion that it was something found in the nature of the milkmaid (something like a notion of a wholesome and clean life led by these women) that made her impervious to smallpox, obsolete and impossible.

And yet, when confronted with the case of this mystery illness, all of this was forgotten in favour of a form of knowledge, of subjugated knowledge, which saw the homosexual body as a conflagration of disease and which, furthermore, saw it as something unstable and on the brink of disaster. It is perhaps in the medical community's anticipation of such a disaster, that this subjugated knowledge – taken for granted, assumed – allowed for the positivity of these early hypotheses; it allowed for them to be taken seriously and to be thought of as likely explanations for the etiology and pathogenesis of the mystery illness.

I would like to end on a remark about this framework of analyzing inventive instances that I have developed in this thesis. To what end can such an analysis completely explain an inventive instance? Is it even possible to do this using any other method? Using this framework we can show how a discursive practice was formed and the gradation of thought that it had to follow to arrive at its resting place as a rule of formation. We can see clearly the point at which it was countersigned as invention. But invention is both private and public, both individual and social, both particular and universal – it is both form and content. And while the content may be social, it is only through the 'clever turn of thought' of the individual that something new is invented. Can that 'clever turn of thought' be shown to be yet another social aspect? Perhaps. But just as likely, in showing its existence as yet another social aspect, yet another aporia will be discovered – another puzzle piece that does not belong there and that has been transferred from another place. Such a tracing would then continue its search and reduction endlessly, ad absurdum; continually adding to the social character of the invention. To completely explain that clever turn of thought as yet another part of the social milieu would be to explain away the inventive instance of the invention. It would mean to erase the signature of the inventor and to only leave suspended in place the countersignature of social recognition, and in doing so, further establish a totalizing and determining structure. This is not the point, nor should it be the point, of this analysis. Indeed, such an analysis "does not claim to efface itself in the ambiguous modesty

of a reading that would bring back, in all its purity, the distant, precarious, almost effaced light of the origin. It is nothing more than a rewriting: that is, in the preserved form of exteriority, a regulated transformation of what has been written. It is not a return to the innermost secret of the origin; it is the systematic description of the discourse-object" (Foucault 2011, 156).

References

Center for Disease Control Task Force on Kaposi's Sarcoma and Opprotunistic Infections. 1982. "Special Report: Epidemologic Aspects of the Current Outbreak of Kaposi's Sarcoma and Opprotunistic Infections." *New England Journal of Medicine* 306 (4): 248-252.

Columbia Encyclopedia, 6th ed., s.v. “disease.”

Derrida, Jacques. 2007. *Psyche: inventions of the Other, Volume 1.* Stanford, CA: Stanford University Press.

Dorland Illustrated Medical Encyclopedia, 32th ed., s.v. “disease.”

Drew, Lawrence, et al. 1981. "Prevalence of Cytomegalovirus Infections in Homosexual Men." *The Journal of Infectious Disease* 143 (2): 188-192.

Durack, David T. 1981. "Opprotunistic Infections and Kaposi's Sarcoma in Homosexual Men." *New England Journal of Medicine* 305 (24): 1465-1567.

Epstein, Steven. 1996. *Impure Science: AIDS, Activism, and the Politics of Knowledge.* Berkeley: University of California Press.

Erni, John Nguyet. 1994. *Unstable Frontiers: Technomedicine and the Politics of Curing AIDS.* Minneapolis: University of Minnesota Press.

Fauci, Anthony. 1982. "The Syndrome of Kaposi's Sarcoma and Opprotunistic Infections: An Epidemiologically Restricted Disorder of Immunoregulation." *Annals of Internal Medicine* 96 (6): 777-779.

Foucault, Michel. 2010. *Birth of a Clinic.* New York: Routledge.

—. 2003. *Society Must Be Defended: Lectures ad College De France.* New York: Picador.

—. 2011. *The Archaeology of Knowledge.* New York: Routledge.

Friedman-Kien, Alvin E., et al. 1982. "Disseminated Kaposi's Sarcoma in Homosexual Men." *Annals of Internal Medicine* 96 (6): 693-700.

Gottlieb, Michael S., et al. 1981. "Pneumocystis Carinii Pneumonia and Mucosal Candidiasis in Previous Healthy Homosexual Men." *New England of Journal of Medicine* 305 (24): 1425-1431.

Heidegger, Martin. 1977. *The Question Concerning Technology and Other Essays.* Toronto: Harper Perennial.

Latour, Bruno, and Steve Woolgar. 1986. *Laboratory Life: The Construction of Scientific Facts.* Princeton, NJ: Princeton University Press.

Lyotard, Jean-Francois. 1988. *The Differend: Phrases in Dispute.* Manchester: Manchester University Press.

—. *The Postmodern Condition: A Report on Knowledge.* 1984. Minneapolis: University of Minnesota Press.

Masur, Henry, et al. 1981. "An Outbreak of Community-Acquired Pneumocystis Carinii Pneumonia." *New England Journal of Medicine* 305 (24): 1431-1438.

Morris, Lynn, et al. 1982. "Autoimmune Thrombocytopenic Purpura in Homosexual Men." *Annals of Internal Medicine* 96 (1): 714-717.

Patton, Cindy. Forthcoming. *Disease of Unknown Original.* New York: Farrar Strauss and Giroux.

—. 1990. *Inventing AIDS.* New York: Routledge.

—. 2014. *L.A. Plays Itself/ Boys in the Sand: A Queer Film Classic.* Vancouver, BC: Arsenal Pulp Press.

Ricoeur, Paul. 1976. *Interpretation Theory: Discourse and the Surplus of Meaning*, Fort Worth: Texas Christian University Press.

—. *The Rule of Metaphor. 2003.* New York: Routledge.

Siegal, Frederick P., et al. 1981. "Sever Acquired Immunodeficiency in Male Homosexuals Manifested By Chronic Perianal Ulcerative Herpes Simplex Lesions." *New England Journal of Medicine* 305 (24): 1439-1444.

Treichler, Paula A. 1999. *How to Have Theory in an Epidemic: Cultural Chronicles of AIDS.* Durham, NC: Duke University Press.

Woolgar, Steve. 1993. *Science: the very idea.* London: Routledge.