Reevaluating Viral Transmission: A Critical Examination of Virological Methods and Assumptions

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Date: 5 February 2025

Keywords: Virology, Viral Transmission, Infectious Diseases, Methodological Flaws, Person-to-Person Transmission, Inoculation Techniques, Transmission Pathways, Ecological Validity

Abstract. This paper critically evaluates the foundational principles of virology, focusing on viral transmission, which underpin the broader scientific claims about the existence and pathogenicity of viruses. A thorough examination reveals significant empirical gaps and methodological limitations. Historical and contemporary studies are reviewed to highlight the lack of consistent evidence supporting person-to-person viral transmission, often relying on proxies such as molecular detection rather than direct causality. By addressing these shortcomings, this paper challenges the conventional paradigms of virology and their implications for public health strategies, including vaccination and quarantine measures. This work aims to foster a critical reassessment of virological methods and assumptions, promoting a more robust scientific framework for understanding diseases.

1 - Introduction

The discipline of virology has long been a cornerstone of modern medical science, providing theoretical and practical frameworks for understanding diseases, vaccine development, and public health strategies. The field implements various techniques to identify viruses, and a breakdown of these techniques is shown in Figure 1. The sequence of steps in virology workflows is normally Isolation → Purification → Detection → Characterization. Central to virology is the foundational concept of viral transmission, the process by which viruses spread between hosts. This paper critically evaluates the foundational principles of virology by focusing on viral transmission.

A virus is defined as follows: A microscopic infectious (transmittable) agent that replicates only inside the living cells of an organism. It consists of genetic material (DNA or RNA) enclosed within a protein coat (capsid) and, in some cases, a lipid envelope. Viruses infect host cells, hijack their machinery to produce more viruses, and can cause various diseases in humans, animals, and plants.

Virologists frequently presume the transmissibility of viruses without empirically demonstrating this in natural settings, thereby bypassing the initial step, highlighted in red in Figure 1. This assumption serves as the foundational premise of the field, without which there is no basis for further virological investigation. If a virus is not demonstrably transmissible, it cannot propagate and infect additional hosts, raising questions about its existence.

Viral transmission is therefore not merely an academic construct; it forms the bedrock of policies and interventions designed to combat diseases attributed to viral agents. However, despite its ubiquity, the scientific and empirical basis of this concept has not been subjected to adequate critical scrutiny.

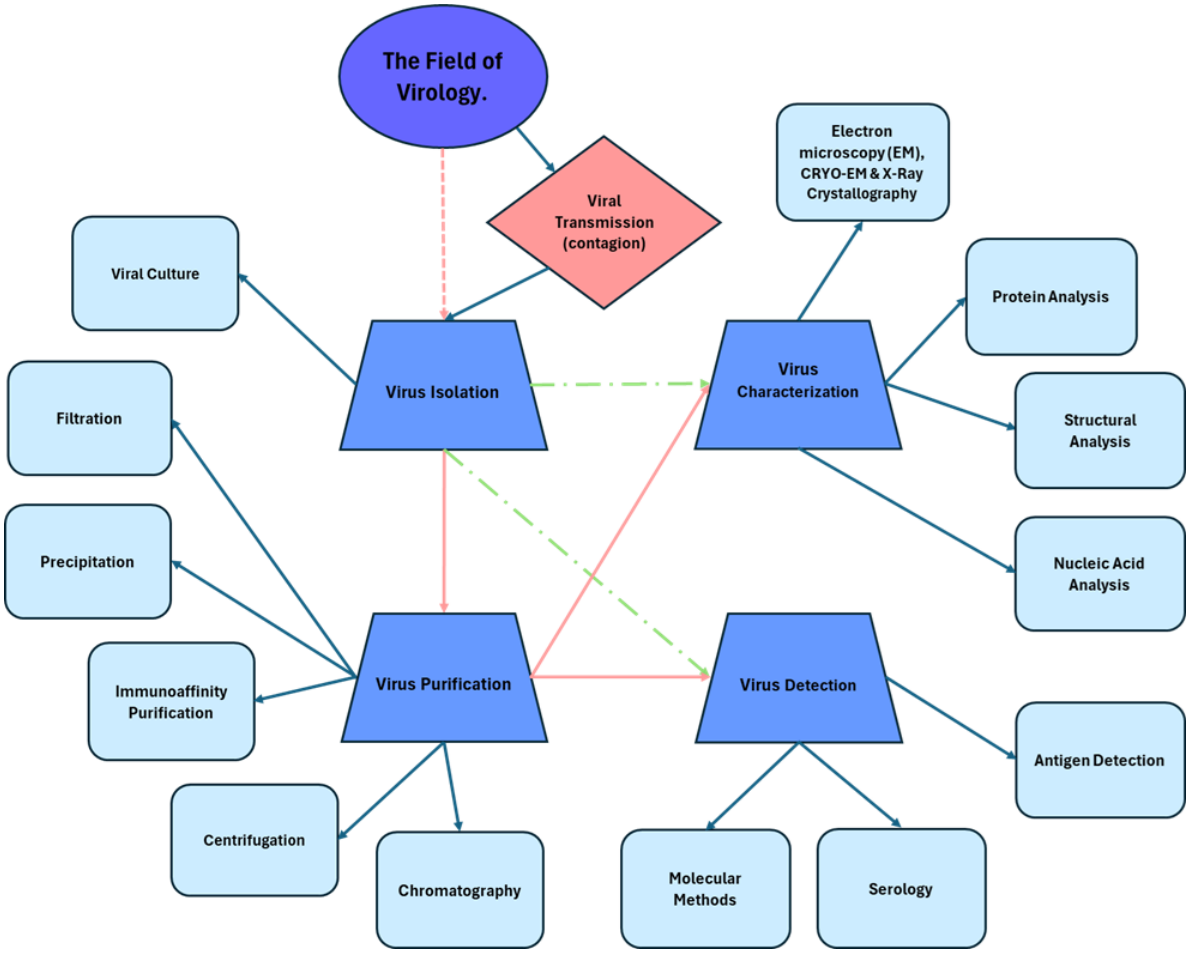

Figure 1: Overview of key methods in virology for virus identification.

Additionally, the use of artificial inoculation methods as well as viral transmission assessment protocols in virology research introduces further complications. These processes can inadvertently induce disease-like symptoms, independent of the virus under investigation. Such effects raise significant concerns about the reliability and ecological validity of experimental results, particularly when natural transmission pathways have not been conclusively demonstrated.

Natural means of transmission, such as airborne dissemination, direct contact, and vector-mediated transmission, have not been consistently demonstrated under controlled conditions, adding to the uncertainty. Historical attempts to demonstrate viral transmission often yielded inconclusive or contradictory results, challenging the assumption of direct person-to-person contagion as the primary mechanism of disease spread. Moreover, contemporary virology frequently relies on indirect methods, such as molecular detection of viral genetic material, which may indicate but not conclusively demonstrate causation. These gaps in evidence raise fundamental questions about the validity of virology's central tenets.

In this paper, we systematically review historical and contemporary studies on viral transmission, critically evaluate the methodologies employed, and explore their broader theoretical and practical implications. By highlighting the empirical gaps and methodological weaknesses, we aim to contribute to a necessary reevaluation of virological paradigms, fostering a more rigorous and transparent approach to the study of disease.

2 - Viral Transmission

Viral transmission is central to virology, shaping our understanding of how infectious diseases spread and persist. Transmission mechanisms encompass a wide array of biological processes, including direct contact, airborne dissemination, vector-mediated transmission, and fomite interactions, which is claimed to enable viruses to establish infection in new hosts. These mechanisms form the basis for defining viruses as infectious agents and for developing strategies to mitigate their spread.

However, the scientific evidence supporting the transmission of many viruses remains inconsistent. Historical attempts to replicate natural infection pathways under controlled conditions frequently failed to demonstrate the anticipated outcomes. While modern virology has advanced significantly with tools such as molecular diagnostics, genetic sequencing, and cell culture systems, these methods often infer transmission rather than directly observing it. For instance, detecting viral RNA or DNA in a host or environment is often assumed to indicate infectivity, even without direct evidence of disease causation.

Furthermore, experimental studies designed to evaluate viral transmissibility often rely on artificial conditions that do not fully reflect the complexity of natural host interactions. Controlled human challenge trials, a key method in transmission research, have repeatedly failed to replicate the infection patterns observed in epidemiological studies. These challenges point to a fundamental gap in our understanding of the dynamics of viral spread.

This section critically examines the historical and contemporary methodologies employed to study viral transmission, with an emphasis on their limitations and the implications of their findings. This analysis evaluates the reproducibility and validity of experimental transmission models to determine whether current paradigms accurately reflect the complexities of viral infectivity and pathogenicity. Through this lens, we aim to provide a nuanced perspective on virology’s underlying assumptions and propose ways to enhance scientific rigor in studying diseases.

2.1 - Historical Context of Transmission Experiments

From the late 19th to the mid-20th century, numerous attempts to experimentally demonstrate viral transmission yielded inconclusive results or outright failures. For example, studies conducted on diseases such as measles, smallpox, and poliomyelitis often failed to produce infection in animal or human subjects despite direct inoculation with bodily fluids from infected individuals. Early experiments, such as those by Chapman (1801) and Willan (1809), repeatedly failed to induce measles through exposure to blood, mucus, or vesicular fluid from infected patients. Similarly, studies on smallpox by Rodermund (1901) involved deliberate exposure to infected material without resulting illness, raising questions about the mechanisms and validity of assumed transmission pathways.

2.2 - Modern Challenges in Demonstrating Viral Transmission

Despite advances in molecular biology and diagnostic tools, challenges persist in establishing the direct causal link between viral exposure and disease onset. Many contemporary studies rely on proxies, such as molecular detection of viral genetic material, rather than direct evidence of infection and transmissibility. For example, controlled studies on influenza, including experiments by Milton Rosenau (1918), exposed healthy individuals to symptomatic patients or their secretions but failed to induce illness in a significant proportion of cases. Similar findings have been reported in recent studies on respiratory viruses, where controlled human challenge trials often fail to replicate natural transmission dynamics.

2.3 – Inoculation Methodological Critiques

Experimental inoculation techniques are central to virology research, enabling the controlled study of infection dynamics, replication, and host responses. However, these methods often involve introducing foreign material directly into the host, by passing natural barriers. This process can inadvertently induce disease-like symptoms due to the toxic nature of the substance being administered or due to tissue damage, independent of the virus under investigation. Such effects raise significant concerns about the reliability and ecological validity of experimental results, particularly when natural transmission pathways have not been conclusively demonstrated. Below, we critically evaluate various inoculation methods, their inherent limitations, and their broader implications, incorporating evidence from literature.

2.3.1 Intracerebral Inoculation

Intracerebral inoculation involves the direct introduction of viral material into the brain of animal models. While this technique is useful for studying neurotropic viruses, it bypasses natural defenses such as the blood-brain barrier. The injection itself can induce inflammation, edema, and other neuropathological effects that mimic disease symptoms, even in the absence of viral activity. This complicates the interpretation of results and highlights the need for caution when extrapolating findings to natural infection scenarios.

2.3.2 Intranasal and Intratracheal Inoculation

These methods, commonly used for respiratory viruses, aim to replicate natural airborne transmission. However, the direct deposition of concentrated viral material on mucosal surfaces may provoke localized responses unrelated to the virus's pathogenicity. The absence of environmental cofactors, such as microbial flora and particulate matter, further reduces the ecological validity of these experiments [1]. Moreover, without prior evidence of natural respiratory transmission, these methods risk overestimating the virus’s infectious potential.

2.3.3 Intravenous Inoculation

Intravenous inoculation introduces viral material directly into the bloodstream, bypassing mucosal and epithelial barriers. While this technique facilitates systemic infection studies, it also circumvents critical early stages of natural infection. The introduction of foreign substances intravenously can trigger systemic inflammation and other adverse effects, independent of viral activity [2]. Such effects may confound experimental outcomes and inflate assessments of a virus’s pathogenic capabilities.

2.3.4 Intramuscular and Subcutaneous Inoculation

Intramuscular and subcutaneous inoculations are employed for studying vector-borne viruses and those requiring dermal entry. These techniques often involve unnaturally high viral doses, which can cause tissue damage or a reaction at the inoculation site [3]. The absence of vector-associated factors, such as saliva or enzymatic components, further limits the biological relevance of these experiments, particularly when natural transmission has not been validated.

2.3.5 Oral and Enteral Inoculation

Oral and enteral inoculations replicate gastrointestinal transmission routes but often require unnaturally high viral loads to overcome the host's natural defenses, such as gastric acid and bile. The disruption of gut homeostasis or the provocation of non-specific reactions due to foreign material introduction complicates result interpretation [4]. These challenges underscore the need to establish natural transmission pathways before employing these methods.

2.3.6 Dermal and Transdermal Inoculation

These techniques involve the application of viral material to breached skin or mucosal surfaces. While useful for studying localized infections, these methods bypass the natural integrity of skin and mucosa, introducing foreign matter that can provoke localized inflammation or a reaction unrelated to viral activity. Without evidence for natural transmission via these routes, such experiments risk producing results of limited translational value.

2.3.7 Disease Induction by Artificial Inoculation

A critical limitation of these artificial techniques is their potential to induce disease-like symptoms solely due to the introduction of foreign matter. Studies have demonstrated that injected substances, even in the absence of infectious agents, can trigger robust inflammatory responses, tissue damage, or systemic effects. For example, the "danger model" describes how the immune system reacts to signals from cellular damage or foreign substances, rather than pathogens alone [3]. Similarly, tissue damage from inoculation can elicit a reaction and mimic pathology [4], while non-specific inflammatory responses to injected materials have been observed across multiple studies [2, 5]. These findings underscore the necessity of validating natural transmission mechanisms before relying on artificial methods to study viral infectivity or pathogenicity.

2.3.8 Broader Implications for Virology

The applicability of artificial inoculation techniques is questionable when natural transmission pathways have not been conclusively demonstrated. Such methods risk producing results that are biologically irrelevant or misleading, undermining the broader understanding of viral behavior. To address these limitations, experimental designs must integrate physiologically relevant models and prioritize the validation of natural transmission routes. Studies using artificial inoculation techniques are often cited as definitive proof of viral transmission [5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24]. These studies and others like them can however not be seen as conclusive evidence for viral transmission without validating natural transmission mechanisms.

2.4 – Viral Transmission Assessment Protocols

Viral Transmission Assessment Protocols are essential tools in virology for studying the spread and impact of infectious diseases. These techniques often involve direct or indirect methods to detect and track viral presence in hosts and environments. However, many of these methods can inadvertently induce disease-like symptoms or physiological changes, complicating the interpretation of results. This section critically examines various transmission monitoring techniques, highlighting how they can contribute to disease symptoms and stress responses in both humans and animals.

2.4.1 Nasal and Oropharyngeal Swabs

Nasal and oropharyngeal swabs are commonly used to collect samples for molecular detection of viral genetic material, such as RNA or DNA. While these swabs are minimally invasive, they can cause localized irritation, inflammation, and other symptoms that mimic those of viral infections. Nasal congestion and rhinorrhea: The insertion of nasal swabs can irritate the nasal mucosa, leading to congestion and nasal discharge. Studies have shown that the mechanical action of swabbing can trigger sneezing, coughing, and even rhinorrhea (nasal discharge), which are often interpreted as symptoms of respiratory infections [25, 26, 27, 28]. Nasal Bleeding and Discomfort: In some cases, nasal swabs can cause minor bleeding or discomfort, particularly if the swab is inserted too forcefully or if the individual has sensitive nasal passages. These effects can be mistaken for

symptoms of an underlying infection. Stress Response: The discomfort and anxiety associated with repeated swabbing can induce a stress response, which may exacerbate symptoms unrelated to viral infection.

2.4.2 Blood Sampling

Blood sampling is a routine procedure for detecting viral antibodies, antigens, or genetic material. However, the process of drawing blood can induce physiological changes that may be misinterpreted as disease symptoms. The insertion of needles for blood sampling can cause bruising, pain, and localized inflammation. These effects are typically minor but can contribute to a general sense of malaise or discomfort. Some individuals experience vasovagal reactions (e.g., fainting, dizziness, or nausea) during or after blood draws. These reactions are stress-induced and can be mistaken for symptoms of systemic illness. The stress associated with blood sampling, particularly in individuals with needle phobia, can potentially mimic or exacerbate disease symptoms.

2.4.3 Handling and Restraint of Animals

In animal studies, handling and restraint are often necessary for sample collection, inoculation, or observation. However, these procedures can induce significant stress and physiological changes in animals, which may mimic or exacerbate disease symptoms. The stress of handling and restraint can lead to the release of stress hormones such as cortisol which can potentially mimic or exacerbate disease symptoms. Animals subjected to frequent handling or restraint may exhibit behavioral changes such as reduced activity, aggression, or altered feeding patterns. These changes can be misinterpreted as signs of illness. Improper handling or restraint can cause physical injuries, such as abrasions, bruising, or even fractures. These injuries can lead to localized inflammation or systemic stress responses, complicating the interpretation of experimental results.

2.4.4 Environmental Monitoring

Environmental monitoring techniques, such as air sampling or surface swabbing, are used to detect viral particles in the environment. While these methods are non-invasive for the host, they can still contribute to stress and discomfort in experimental settings. In studies involving air sampling, animals may be exposed to noise or mechanical disturbances from sampling equipment. These disturbances can induce stress responses, potentially altering behavior. Surface swabbing in animal enclosures can disrupt the animals' environment, leading to stress or anxiety. Additionally, the presence of researchers in the animals' space during sampling can cause behavioral changes that may be misinterpreted as disease symptoms.

2.4.5 Invasive Monitoring Techniques

Invasive monitoring techniques, such as intubation or implantation of monitoring devices, are sometimes used in virology research to study disease progression or transmission dynamics. These techniques can induce significant physiological changes and stress responses. Intubation for sample collection or monitoring can cause trauma to the respiratory tract, leading to inflammation, coughing, or difficulty breathing. These symptoms can be mistaken for those of a respiratory infection. The implantation of monitoring devices, such as telemetry sensors, can cause localized inflammation, pain, or infection at the implantation site. These effects can complicate the interpretation of disease symptoms. Invasive procedures often require anesthesia, which can have

systemic effects on the host, including altered physiological responses. The recovery period from anesthesia can also induce stress and behavioral changes.

2.4.6 Behavioral Monitoring

Behavioral monitoring is used to assess disease progression or the impact of viral infections on host behavior. However, the methods used to monitor behavior can induce stress or physiological changes. Continuous video monitoring can cause stress in animals, particularly if they are not accustomed to the presence of cameras or lighting. This stress can lead to altered behavior. Behavioral tests often require handling or manipulation of animals, which can induce stress and alter their behavior. For example, forced swimming tests or maze experiments can cause anxiety and physiological stress, potentially confounding the results.

2.4.7 Implications for Virology Research

The use of pathogen surveillance and diagnostic methodologies in virology research is essential for understanding disease dynamics, but it is crucial to recognize that these techniques can themselves induce symptoms or physiological changes that may be misinterpreted as evidence of viral infection. The stress and discomfort associated with these methods can alter behavior, and overall health, complicating the interpretation of experimental results.

However, before these monitoring techniques are implemented to "detect" disease, it is imperative to establish proof of natural transmission in the most natural setting possible. Without clear evidence that a virus can spread naturally from host to host under real-world conditions, the foundation of virology—that viruses are transmissible infectious agents—becomes tenuous. Relying on artificial monitoring techniques—such as nasal swabs, blood sampling, or invasive procedures—without first proving natural transmission may lead to mistaking experimental artifacts for genuine disease processes.

For example, if a virus cannot be shown to spread naturally through airborne transmission, direct contact, or other ecological pathways, the use of molecular detection methods (e.g., PCR) or cytopathic effects (CPE) in cell cultures becomes scientifically questionable. These methods, while useful for identifying cellular changes, do not inherently prove that a virus is capable of causing disease through natural transmission. Without this foundational proof, virology faces a significant challenge in defending the existence of a virus as a causative agent of disease.

Moreover, the absence of natural transmission evidence raises ethical and practical concerns about the implementation of public health measures, such as vaccination campaigns, quarantine protocols, and antiviral treatments. If the assumption of natural transmission is unproven, the scientific justification for these interventions becomes uncertain, potentially leading to misallocation of resources and unnecessary societal disruption.

To address these challenges, virology must prioritize studies that validate natural transmission mechanisms in ecologically relevant settings. This includes conducting controlled experiments in environments that closely mimic real-world conditions, such as observing disease spread in communities or animal populations without artificial interventions. Only after natural transmission is conclusively demonstrated should diagnostic and monitoring techniques be employed to study the virus further.

By establishing a clear chain of evidence—from natural transmission to laboratory detection—virology can strengthen its scientific rigor and credibility. This approach ensures that the observed symptoms and disease processes are genuinely attributable to the virus in question, rather than being artifacts of experimental procedures or monitoring techniques. Without this foundational proof, virology risks undermining its own claims and facing significant skepticism from both the scientific community and the public.

2.5 - Reevaluating Evidence for Viral Transmission

Building upon the inoculation methodological critiques discussed in Section 2.3 and the viral transmission assessment protocols discussed in Section 2.4, this section further examines the limitations of experimental designs that underpin claims of viral transmission. In Section 2.3, it was highlighted that commonly employed inoculation methods—such as intracerebral, intranasal, and intravenous techniques—bypass natural barriers, often inducing symptoms unrelated to the virus under investigation. Section 2.4 also discusses commonly employed assessment protocols that can mimic or exacerbate disease symptoms. These findings call into question the ecological validity of such methods and emphasize the need for a rigorous reevaluation of how viral infectivity and transmission are studied.

To critically evaluate the validity of existing paradigms, this section analyzes an extensive body of historical and contemporary studies attempting to demonstrate person-to-person viral transmission. While these studies employ diverse methodologies, a consistent pattern emerges: the inability to replicate natural transmission dynamics under controlled conditions. This raises important questions about the assumptions underlying virology's foundational claims.

The discussion begins by exploring early experiments, which sought to transmit diseases such as measles, smallpox, and polio through bodily fluids, yet repeatedly failed to produce the expected outcomes. By reviewing these studies and their implications, we aim to uncover the empirical and methodological gaps that persist in the field. The studies that were reviewed include the following:

1. **J. R. Paul** - "The earliest clinical descriptions of [infantile paralysis] came from several diverse areas: England, 1795; Italy, 1813; India, 1823; and U.S.A., 1830 … There was no mention of contagion, or of epidemics, nor was the condition regarded as a medical problem of any magnitude."
2. **The Journal of Infectious Diseases, Vol. 2, No. 2 (Mar. 1, 1905)**:

   - **Chapman, 1801**: Tried to transmit measles using the blood, tears, the mucus of the nostrils and bronchia, and the eruptive matter in the cuticle without any success.
   - **Willan, 1809**: Inoculated three children with vesicle fluids of measles but without success.
   - **Albers, 1834**: Attempted to infect four children with measles without success. He quoted Alexander Monro, Bourgois, and Spray as also having made unsuccessful inoculations with saliva, tears, and cutaneous scales.
   - **Themmen, 1817**: Tried to infect 5 children with measles. 0/5 children became sick.
3. **Charles Creighton, 1837** - (A history of epidemics in Britain). "No proof of the existence of any contagious principles by which it was propagated from one individual to another."
4. **EH Ackernecht, writing about Anticontagionism between 1821 and 1867** - "That the anticontagionists were usually honest men and in deadly earnest is shown, among other things, by the numerous self-experiments to which they submitted themselves to prove their contentions." also see "Famous are the plague self-experiments of Clot-Bey, the offers for plague self-experiment by Chervin, Lassis, Costa, Lapis, and Lasserre, and the cholera self-

experiments of Fay, Scipio Pinel, Wayrot, and J.L. Guyon. The amazing thing is that almost all of these experiments failed to produce the disease."

5. **Note on Hospitals by Florence Nightingale, 1858** - "Suffice it to say, that in the ordinary sense of the word, there is no proof, such as would be admitted in any scientific inquiry, that there is any such thing as 'contagion." also see "Just as there is no such thing as 'contagion,' there is no such thing as inevitable 'infection."

6. **A. C. Bull, 1868** - "It does not seem apparent in this small [polio] epidemic that contagion played any role, because the disease occurred here and there in the different places of the district without the possibility of establishing any relation between the various cases or the families of the same."

7. **K. O. Medin, 1887** - "Medin did not consider polio to be a contagious disease."

8. **C. Caverly, 1894** - "There was a general absence of infectious disease as an etiologic factor in this [polio] epidemic. The element of contagium does not enter into the etiology either. I find but a single instance in which more than one member of a family had the disease, and as it usually occurred in families of more than one child, and as no efforts were made at isolation, it is very certain that it was non-contagious."

9. **C. B. Leegaard, 1899** - "Infantile paralysis is of an infectious, but not of a contagious nature. As a matter of fact, no indisputable instance of contagion could be proved."

10. **Journal of American Medical Association, Volume 72, Number 3, 1919** (or additional link **here**)**:**

    - **Warschawsky, 1895** - Injected small pigs and rabbits with blood taken in the eruptive stage. All results were negative.
    - **Belila, 1896** - Placed warm nasal mucus and saliva from measles patients on the nasal and oral mucous membrane of rabbits, guinea-pigs, cats, mice, dogs and lambs, but without any positive results.
    - **Josias, 1898** - Rubbed measles secretions over the throat, nose and eyes of several young pigs, but without any effects.
    - **Geissler, 1903** - Inoculated sheep, swine, goats, dogs and cats in various ways with the bodily fluids from patients with measles; including smearing, spraying, rubbing. All results were negative.
    - **Pomjalowsky, 1914** - Injected measles infected blood into guineapigs, rabbits and small pigs. All results were negative.
    - **Jurgelunas, 1914** - Inoculated blood from patients with measles into suckling pigs and rabbits, but without effect.

11. **Dr. Rodermund, 1901** - From his diary of Smallpox experiments. For 15 years he smeared the pus of smallpox patients on his face and used to go home with his family, play cards at the gentleman's club and treat other patients and never got sick or saw a single other person get sick.

12. **Walter Reed, 1902** - "Without entering into details, I may say that, in the first place, the Commission saw, with some surprise, what had so often been noted in the literature, that patients in all stages of yellow fever could be cared for by non-immune nurses without danger of contracting the disease. The non-contagious character of yellow fever was, therefore, hardly to be questioned."

13. **I. Wickman, 1905** - “Pediatrician Ivar Wickman investigated the 1905 [polio] epidemic and visited over 300 cases. The epidemiological picture showed that polio cases often occurred miles apart from each other with no obvious connections.”

14. **The New York Neurological Society, 1907** - “A Collective Investigation Committee of the New York Neurological Society…was appointed to investigate this [polio] epidemic of 1907 … The committee concluded from the returned blanks that poliomyelitis was infectious but not a contagious disease.”

15. **H. C. Emerson, 1908** - “A large number [244] of children were in intimate contact with those that were sick [polio], and of these children an insignificant minority developed the disease.”

16. **J. Zappert, 1908** - “Zappert collected 137 [polio] cases in North Austria ... he failed to prove contagion in this epidemic.”

17. **A. S. Hamilton, 1908** - “Hamilton reported three epidemics of poliomyelitis, all more or less extensive, occurring in Minnesota in 1908 … There was no evidence found that the disease was infectious or contagious.”

18. **L. E. Holt & F. E. Bartlett, 1908** - “We have collected reports of 35 epidemics of poliomyelitis prior to the year 1907 … The comparatively small number and wide distribution of the cases in most of the epidemics is very striking, and seems to indicate that the different cases had no relation to one another or to a common cause … We have taken especial care to secure the data regarding the occurrence of more than one case in a family or household. On this subject we have included not only facts derived from a study of the epidemics here collected, but also other single instances which have been scattered through literature. In all we have collected a total of 40 instances, comprising 96 cases, in which more than 1 case occurred in a family or household … Whether we can go farther and state that the disease is communicable is an open question.”

19. **Massachusetts State Board of Health, 1909** - “Poliomyelitis prevailed in epidemic form in Kansas during the summer of 1909 … No method of contagion could be found, and the author does not consider the disease contagious.”

20. **K. Landsteiner & E. Popper, 1909** - “Attempts to transmit the disease [polio] to the usual laboratory animals, such as rabbits, guinea pigs, or mice, failed.”

21. **E. W. Martin, 1909** - “I do not believe poliomyelitis is contagious.”

22. **Landsteiner & Popper, 1909** - "Attempts to transmit the disease [polio] to the usual laboratory animals, such as rabbits, guinea pigs, or mice, failed."

23. **F.E. Batten, 1909** - “Against the infectivity of the disease may be urged, first, the absence of spread of infection in hospital. The cases of poliomyelitis admitted to hospital freely mixed with other cases in the ward without any isolation or disinfection, some 70 children came in contact, but no infection took place.”

24. **The Boston medical and surgical journal, 1909** - An inquiry a 1908 polio outbreak found the following: “A large number of children were in intimate contact with those that were sick, and of these children an insignificant minority developed the disease.” 244 children were in intimate contact with those who were afflicted with polio. Of those 244 children, an "insignificant minority" developed the disease.

25. **Flexner & Lewis, 1910** - Multiple unsuccessful polio transmission attempts. "Many guinea-pigs and rabbits, one horse, two calves, three goats, three pigs, three sheep, six rats, six

mice, six dogs, and four cats have had active virus introduced in the brain but without causing any appreciable effect whatever. These animals have been under observation for many weeks."

26. **S. Flexner, 1910** - "No instance of the spontaneous transfer of the virus from a paralyzed to a normal monkey arose, although many opportunities for contagion in the course of our many experiments occurred."

27. **F. E. Batten, 1911** - "Against the infectivity of the disease may be urged, first, the absence of spread of infection in hospital. The cases of poliomyelitis admitted to hospital freely mixed with other cases in the ward without any isolation or disinfection, some 70 children came in contact, but no infection took place. On these grounds it is probable that the paralytic stage of the disease is not contagious. Secondly, the striking absence of infection when contact has been most close. In November 1909, H. E. was taken ill with poliomyelitis; all five brothers and sisters, although in closest contact, remained unaffected. In October 1909, M. K., aged 2½, was taken ill; two sisters, aged 6 and 11 respectively, slept with and were in close contact with the child and remained unaffected. Twin sisters, aged 2½, one was affected, the other unaffected."

28. **M. J. Rosenau et al., 1911** - Injected 18 monkeys with the nasal and buccal secretions obtained from 18 persons who were suffering with polio. These results were negative.

29. **R. W. Lowett & M. W Richardson, 1911** - "No instances as yet have been reported in which one monkey has taken the disease [polio] from another, although long continued and intimate contact has been maintained."

30. **I. Strauss, 1911** - "The material consisted of ten [polio] cases … The mucus was obtained by passing dry cotton swabs into the nasopharynx through the mouth … The filtrate was then centrifuged and either 2 or 4 c.c. were injected intracerebrally into [10] rhesus monkeys… No monkey became ill as a result of these inoculations."

31. **Dr. Acker, 1911** - "I have not seen any cases of [polio] contagion. We put the patients on one side and typhoid cases on the other, and no nurse or mother was infected. If the disease was so contagious, I don't see why the nurses and mothers would not have been infected."

32. **L. M. Beeman, 1911** - "Two [polio] epidemics in Connecticut studied by one of our ablest and most careful investigators point to the conclusion that in these epidemics contagion was not present."

33. **H. D. Chapin, 1911** - "There is one aspect of this disease [polio] of considerable interest. Years ago we never thought of contagion with reference to it."

34. **S. S. Adams, 1911** - "One very large institution in New York reported that it did not take any means whatever to isolate and did not consider the disease [polio] a contagious one. As to the disease originating in the hospital, the invariable reply was, no. That was our experience in the hospital."

35. **A. B. Soltau, 1911** - "Is poliomyelitis infectious or contagious? That it has been labelled "infective poliomyelitis" is no proof." […] "It is uncommon to find more than one case in a family, and in none of the Plymouth cases was more than one affected in the same household. Nor…was there any evidence of contagion. Further, it is practically unknown for the disease to spread in hospitals, though no isolation precautions are usually taken." […] "The proofs, however, of infection by direct contagion, or through the intermediary of "contacts" are scanty."

36. **C. Levaditi & V. Danulesco, 1912** - "As early as 1912, Levaditi and Danulesco reported that normal Rhesus monkeys housed with infected monkeys did not develop poliomyelitis."

37. **Scientific American Supplement, 1912** - "Dr. Manning further points out that the disease is probably not transmitted by ordinary contagion for the following reasons:

1. The experimental production of the disease in monkeys is by inoculation.
3. Poliomyelitis artificially induced in monkeys has never been spontaneously transmitted to animals confined in the same cage or room.
4. The comparative rarity of multiple cases in families.
5. Acute cases of poliomyelitis introduced in wards of hospitals not followed by a secondary case."

"It appears, then, that … ordinary contagion, that is to say, transmission by mere contact or proximity of two persons does not occur."

38. **J.J. Moren, 1912** - "Monkeys suffering from polio in the same cage with healthy monkeys, do not infect others. Also, in the majority of cases occurring in an epidemic only one member of a family is affected. In proportion to the number exposed, very few suffer."

39. **E. M. Mason, 1912** - "The question of [polio] contagion, in the usual sense of the word, is not settled … Healthy monkeys have been kept in cages with others in various stages of the disease, yet no infection has been reported."

40. **R. M. Hewitt, 1912** - "I submit that the evidence in favour of contact transmission [of polio] is slight. It is not common to find more than one case in a family. It has not been found to spread in hospitals, although isolation precautions were not taken."

41. **R. Farrar, 1912** - "As regards the epidemiologic evidence of [polio] contagiousness, it has usually been impossible, even in epidemics, to trace lines of contact from case to case … it is characteristic that the cases are scattered, occurring in persons who have never been in any sort of direct or known indirect contact with a previous recognized case. Not infrequently the patient is a child living far away from the nearest known previous case, and who has certainly not been away from home within a period of several weeks before the attack. Still more striking evidence of the non-contagiousness of poliomyelitis is afforded by the fact that one rarely finds more than a single case in a family, and still more rarely finds multiple cases in a family separated by such an interval as would suggest the infection of one from the other. Numerous instances can be cited where large numbers of children have been exposed in schools or institutions, to acute cases of poliomyelitis without the development of any secondary cases."

42. **M. J. Rosenau, 1913** - "Careful and masterly epidemiological investigations of poliomyelitis have been conducted by the Massachusetts State Board of Health extending over a period of five years. The results of these studies were summarized by Dr. Mark W. Richardson, who plainly brought out the fact that the disease, as observed in Massachusetts, does not have the earmarks of a contagious disease. The disease prevails in rural rather than under urban conditions. In fact it shows little tendency to invade cities, and when it does enter the city it does not strike the crowded, congested portions of the city." […] "Cases of infantile paralysis in all stages of the disease have been taken into the hospitals, orphan asylums, children's homes, reformatory schools, and other institutions in the Commonwealth, but in no instance during the five years in which the disease has been studied has it ever spread under these circumstances."

43. **P. H. Römer, 1913** - "No proofs of the contagiousness of the disease [polio] could be obtained in the great epidemic in New York in 1907, nor in the epidemic in the Steiermark (Furntratt, Potpeschnigg) nor in Pomerania (Peiper)."

44. **The Lancet, 1913** - “In the Deddington [polio] epidemic…it was apparently impossible to trace any direct contagion, even by the interposition of poliomyelitis carriers.”

45. **W. H. Frost, 1913** - “The statistics presented in this report show that of 2,070 persons exposed to poliomyelitis by residence in the same houses and same families as poliomyelitis patients, only 14 (0.6 per cent) developed the disease.”

46. **A. H. Jennings & W. V. King, 1913**, - “Poliomyelitis, moreover, is marked by the occurrence of sporadic cases, not to be explained by contact infection and there is a lack of evidence of direct contagion, two facts which are true of pellagra.”

47. **H. W. Frauenthal et al, 1914** - "Advocates of the contagion theory were at a loss to account for the fact that spontaneous [polio] transmission among laboratory monkeys was never known to occur ... There is no proof that spontaneous transmission of acute poliomyelitis, without an inoculation wound, can take place. There is no proof that contact contagion takes place. Spontaneous development of the disease among laboratory animals is unknown."

48. **M. J. Rosenau, 1914** - “Rosenau, Sheppard and Amoss therefore injected 18 monkeys with the nasal and buccal secretions obtained from 18 persons who were suffering with the disease [polio] at the time, or in the stage of convalescence, or from persons suspected of acting as carriers. These results were negative. At the same time Straus of New York had a series of negative results, and other American workers were also unable to find the virus where we assumed it should be. These negative results seemed to us to have positive significance and was the first definite indication that we were upon the wrong trail. That poliomyelitis is not a “contagious” disease was clearly brought out by Dr. Richardson and other observers who have spoken this morning, all of whom have emphasized the point that the disease shows little or no tendency to spread in crowded districts, in schools, in institutions, in asylums, in camps and in other places where one would expect a disease spread by contact through secretions of the mouth and nose to spread most readily. We have in mind the fact that many cases of the disease have been brought into asylums and hospitals throughout the State of Massachusetts, in all stages of the infection; yet secondary cases have not occurred under such circumstances. On the contrary the disease prevailed in Massachusetts more particularly in rural and country districts sparsely settled.”

49. **M. W. Richardson, 1914** - “The experience of Massachusetts has not been such as to support the theory that infantile paralysis is spread from person to person by direct or indirect contact. The rural preponderance of the disease, the comparative immunity of children confined in institutions and hospitals, the summer incidence, the failure of the disease to find its greatest incidence in cities and localities where density of population and overcrowding are most marked, and the irregular distribution have all militated against the acceptance of such a theory.”

50. **W.H. Frost, 1916** - “As regards the epidemiologic evidence of [polio] contagiousness, it has usually been impossible, even in epidemics, to trace lines of contact from case to case … it is characteristic that the cases are scattered, occurring in persons who have never been in any sort of direct or known indirect contact with a previous recognized case. Not infrequently the patient is a child living far away from the nearest known previous case, and who has certainly not been away from home within a period of several weeks before the attack. Still more striking evidence of the non-contagiousness of poliomyelitis is afforded by the fact that one rarely finds more than a single case in a family, and still more rarely finds multiple cases in a family separated by such an interval as would suggest the infection of one from the other. Numerous instances can be cited where large numbers of children have been exposed in schools or institutions, to acute cases of poliomyelitis without the development of any secondary cases.”

51. **Official Reports of the Bureaus of the Department of Health, 1916** - "No attendant, physician, nurse or domestic, and no patient admitted to any of the hospitals throughout the city, for other cause than poliomyelitis, during the [1916 polio] epidemic, contracted poliomyelitis. This has been the almost universal experience in the past, and has often been brought forward as a proof of the non-communicable character of the disease."

52. **W. H. Frost, 1916** - "Only a small proportion of cases can be ascribed to known contact with previous definite cases of poliomyelitis. Even including association with merely suspicious cases of illness, the majority of cases of poliomyelitis can not be traced to known contact, either direct or indirect, with any previous case. It is this apparent lack of relation between cases which has led so many investigators to seriously doubt or even deny the transmissibility of the disease." […] "The disease develops in such a small proportion of persons known to be intimately associated with acute cases. It also seems well established that the recognized cases of the disease must be relatively unimportant sources of infection. This follows necessarily, because a large proportion of the cases studied have been in persons not associated in any known way with previous recognized cases—often under circumstances which precluded the possibility of even indirect contact."

53. **W. L. Holt, 1916** - "I investigated [a polio epidemic] the best I could and was much surprised that I could trace hardly any cases to personal contact with others, there rarely being successive cases."

54. **C. K. Mills, 1916** - "During the present [polio] epidemic I have not been able to find any evidence of transmission of the disease to a healthy child or adult by a nurse, attendant or doctor in contact with cases of poliomyelitis."

55. **I. D. Rawlings, 1916** - "Anyone who has had much experience with poliomyelitis is struck by the infrequency, relatively, of the secondary cases among direct contacts ... there were approximately 1,500 direct contacts, and yet but one possible case occurred among them. Also, among the large number of people that came from New York and other infected areas not a single case occurred. One is constantly struck with the fact that there are relatively few contact cases."

56. **M. W. Richardson, 1916** - "Facts Against its [polio's] Transfer by Direct or Indirect Human Contact.

2. Summer incidence of the disease: The vast majority of cases occur during the spring, summer, and fall, when personal contact is least intimate. With the onset of winter, when the population becomes more and more congested in houses, schools, etc., the disease becomes reduced almost to nothing. […]
3. Maximum prevalence of the disease in country districts where personal contact is least intimate at all times.
4. Failure to spread in general hospitals for children in which, up to recent times, cases have been received without let or hindrance.
5. Failure to spread in schools and institutions for children in which single cases have occurred and where personal contact with large numbers of children has been intimate.
6. Extreme rarity of the disease in doctors nurses, and other attendants upon persons sick with infantile paralysis.
7. Entire absence of infection in laboratory workers with the virus of infantile paralysis. Of course, in these two latter instances, the effect of age is important, but occasional infection would certainly be noted if the disease were markedly contagious.
8. Comparatively rare occurrence of more than one case of the disease in large families of children even under the markedly congested conditions of tenement life.

9. Epidemies cease oftentimes in mid-career, so to speak, when the human material has been by no means exhausted and the opportunities for direct or indirect contact are at their maximum.
10. Long continued immunity of cities and towns in close commercial relations with infected centers even though interchange of population with possibilities of indirect contact is marked. […]
11. The Colrain epidemic in 1908 was perhaps the most severe on record, thirteen per thousand of the population being affected, yet according to Emerson, who investigated the epidemic in this and neighboring towns, evidence of contagion was practically lacking. […] "The human contact theory cannot be made to fit these facts except by efforts so extraordinary that the value of the theory is thereby practically destroyed."

57. **T. H. Weisenburg, 1916** –"I started out with the idea, after having read Wickman's and Römer's articles and from the New York reports, that the disease [polio] was personally contagious, but the more experience I had the more I got away from that impression." […] "There is no instance of any nurse or physician who either acquired the disease or who carried the disease elsewhere. A number of the attending physicians with young children in their families went home daily and did not carry the infection with them. I have no doubt that many more such examples occurred in New York. It was the impression of all the nurses and physicians that poliomyelitis was not spread by personal contact."

58. **C. T. Brues, 1916** - "On account of several peculiar facts connected with previous epidemics of poliomyelitis, it has appeared possible that the disease may not be spread directly from one person to another" […] "The disease has always been regarded as more abundant under rural conditions." […] "Cases of this disease do not usually appear in such a way that they can be positively traced to contact." […] "Where large numbers of persons are crowded in congested dwellings, there is no tendency toward a rise in the incidence of poliomyelitis. This is abundantly shown by the details which have been cited in the preceding pages and offers poor support to the view that these cases have been contracted as a result of contact with children suffering from the disease, or as a result of contact with healthy carriers of the poliomyelitis virus."

59. **C. F. Bolduan, 1916** - "Personally I believe we ought to abandon the idea that this disease [polio] is spread by direct contact of person to person by the respiratory passages (coughing, sneezing, spitting, etc.)."

60. **H. L. Abramson, 1917** - Attempts to induce polio in a monkey by injecting the spinal fluid of 40 polio patients into the brain failed.

61. **Dold et al. 1917** (Original paper in German from **Muenchener Medizinische Wochenschrift 64 (1917), bottom of p 143**) - Injected healthy people with the nasal secretions taken from one ill person, 1/40 healthy people became ill.

62. **J. C. Geiger, 1917** - 66 kids came into intimate contact with a child afflicted with polio. 0/66 became ill.

63. **W. H. Frost, 1917** - "Extensive epidemiologic observations are consistent in their testimony that definite lines of contact between [polio] cases can seldom be traced, and that the disease shows other features which we are not accustomed to expect in a directly transmitted infection." […] "Almost without exception poliomyelitis reaches its highest prevalence, both endemic and epidemic, during the summer and autumn months, declining markedly with the advent of winter." […] "Even in the most intense epidemics of poliomyelitis only a small proportion, usually not more than one to five per thousand of the total population in the epidemic area is affected with recognizable symptoms of infection; yet the epidemics are invariably self-limited, declining rather sharply, often in mid-season, after only this small proportion of the population has been attacked." […] "The most intensive study of

numerous outbreaks has consistently failed to show precisely the sources and routes of infection." […] "Direct contact between patients cannot be traced in the majority of cases … a very considerable proportion of cases occur under conditions which absolutely preclude all probability of the patient's having been in recent contact with any previously recognized frank case of poliomyelitis, or even any case of febrile illness." […] "A large proportion, often a majority of cases, have certainly not been in contact with previous frank cases of poliomyelitis, either directly or indirectly, through distinctly traceable channels" […] "A large proportion even of children intimately exposed to acute cases escape the disease." […] "It is noteworthy that epidemics have characteristically reached a higher degree of prevalence in the population of rural communities and small towns than in large, densely populated cities."

64. **H. Emerson, 1917** - "Contact between recognized [polio] cases can seldom be traced."

65. **W. A. Evans, 1917** - "Every effort to control infantile paralysis is based upon the theory that it is spread by contact and carriers; and yet it is with exceeding difficulty that we recognize the facts in relation to this theory. For instance, in New York City in this epidemic, in the report made about a month ago, it was stated that in 97% of the cases there was but one case to the family." […] "The disease tends to end with the opening of schools, so that there is much reason for believing that it is not a disease that is due to contact infection."

66. **A review of the investigations concerning the etiology of measles, A. W. Sellards Harvard Medical School. Boston, Massachusetts as seen below:**
- **Jurgelunas, 1914**: Tried to produce measles in monkeys using inoculations of the blood and mucus secretions from measles patients as well as by exposing the animals to patients in measles wards. All results were negative.
- **Sellards, 1918**: Tried to transmit measles to 8 healthy volunteers without a prior history of measles exposure. 0/8 men became sick after multiple failed attempts.
- **Sellards and Wenworth, 1918**: Inoculated 3 monkeys in various ways, including intensive injections of blood from measles patients. The animals remained well.
- **Sellards and Wenworth, 1918**: Blood from measles patients was injected simultaneously into 2 men and 2 monkeys. Both men remained symptom-free. One of the two monkeys developed symptoms that were not suggestive of measles.

67. **Milton Rosenau, 1918** - "Monkeys have so far never been known to contract the disease [polio] spontaneously, even though they are kept in intimate association with infected monkeys."

68. **Hess & Unger, 1918** - "In three instances the nasal secretion of varicella patients was applied to the nostrils; in three others the tonsillar secretion to the tonsils, and in six, the tonsillar and pharyngeal secretions were transferred to the nose, the pharynx, and the tonsils. In none of these twelve cases was there any reaction whatsoever, either local or systemic."

69. **Hess & Unger, 1918** - The vesicle fluids from people with chickenpox was injected intravenously into 38 children. 0/38 became sick.

70. **M. W. Richardson, 1918** - "The fact that the hospital personnel in infantile paralysis does not acquire infection is an experience so nearly universal that the rare exceptions serve only to prove the rule." […] "No case has come to my notice in all the literature in which a laboratory worker has acquired infantile paralysis in the course of his investigations, even though, as in one instance, the syringe broke, and virus was sprayed into the face of the investigator." […] "The epidemiological facts are strongly against the theory that infantile paralysis is spread from person to person by direct or indirect contact."

71. **C. H. Lavinder et al., 1918** - “In individual cases contact, either direct or indirect, with a previous case of poliomyelitis could but rarely be established, and in many instances the possibility of such contact could be satisfactorily excluded.”

72. **Published in the Journal - American Medical Association, 1919** - Need of Further Research on The Transmissibility Of Measles And Varicella. “Evidently in our experiments we do not, as we believe, pursue nature's mode of transmission; either we fail to carry over the virus, or the path of infection is quite different from what it is commonly thought to be.”

73. **Milton J. Rosenau, March 1919** - Conducted 9 separate experiments in a group of 49 healthy men, to prove contagion. In all 9 experiments, 0/49 men became sick after being exposed to sick people or the bodily fluids of sick people.

74. **Wahl et al, 1919** - Conducted 3 separate trials on six men attempting to infect them with different strains of Influenza. Not a single person got sick.

75. **Schmidt et al, 1920** (Original paper in German **here**) - Conducted two controlled experiments, exposing healthy people to the bodily fluids of sick people. Of 196 people exposed to the mucous secretions of sick people, 21 (10.7%) developed colds and three developed grippe (1.5%). In the second group of the 84 healthy people exposed to mucous secretions of sick people, five developed grippe (5.9%) and four colds (4.7%). Of forty-three controls who had been inoculated with sterile physiological salt solutions eight (18.6%) developed colds. A higher percentage of people got sick after being exposed to saline compared to those being exposed to the “virus”.

76. **Williams et al, 1921** - Tried to experimentally infect 45 healthy men with the common cold and influenza, by exposing them to mucous secretions from sick people. 0/45 became ill**.**

77. **Mahatma Gandhi, 1921** - "and the poison that accumulates in the system is expelled in the form of small-pox. If this view is correct, then there is absolutely no need to be afraid of small-pox" also see "This has given rise to the superstition that it is a contagious disease, and hence to the attempt to mislead the people into the belief that vaccination is an effective means of preventing it."

78. **Blanc and Caminopetros, 1922** (original paper in French **here**) - Material from nine cases of shingles was inoculated into the eyes, cornea, conjunctiva, skin, brain, and spinal cord of a series of animals, including rabbits, mice, sheep, pigeons, monkeys, and a dog. All results were negative.

79. **R. B. Osgood et al., 1922** - “In poliomyelitis, the evidence of human contact contagion is so doubtful and rare that the burden of proof seems to be on those who maintain that the human carrier is the common source of infection.”

80. **C. T. Brues, 1923** - “The seasonal prevalence of the disease [polio] lends no support to the theory of contact contagion.” […] “The geographical … relations of poliomyelitis present several features which … do not lend themselves to explanation on the basis of contact infection.” […] “Rural communities are almost invariably more severely affected than urban ones. This has been observed everywhere that the disease has become an epidemic. Even in New York City during 1916, the incidence of poliomyelitis in the several boroughs of that city was almost exactly in inverse proportion to the density of population. It was highest in Queensboro and Richmond (Staten Island), less in Brooklyn and still far less on the thickly populated island of Manhattan, which includes one of the most densely crowded areas in the world.” […] “The epidemic severity, even in the parts of a large city, does not vary with the density of the human population … This is the lower East Side, the most crowded part of the city, where any disease spread by contact should become rampant, but its incidence rate was

lower than that of any entire borough in the city … The conditions here, which I have been able only briefly to outline, directly contradict any theory of personal contact." […] "It is impossible to establish the probability of direct contact with a previous case in more than a small proportion of cases." […] "The failure of the disease to spread in hospitals to nurses, attendants or other patients has been noted incessantly by various observer." […] "The several points which have been so briefly outlined show the many difficulties and contradictions which make it extremely difficult and to my mind impossible to understand the epidemiology of poliomyelitis on the assumption that it is a disease spread by personal contact." […] "The failure of the personal contact theory to meet the requirements has led to the assumption that poliomyelitis is spread mainly by healthy carriers, or third persons harboring the virus, who may distribute it in a more infectious condition than those actually in the prodromal or acute phases of the disease. This accounts for the fact that contact with a severe case involves little chance of infection, and explains to some extent, although very imperfectly, the spatial spread of epidemics. Many features, however, as we have seen, show it to be inadequate" […] "No portal of entry has been found in laboratory experiments which could function under natural conditions without some medium for inoculation." […] "Experimental poliomyelitis has so far been produced only by the injection of virus through what are, in most cases at least, wholly unnatural channels." […] "Our present explanation of the spread of poliomyelitis through contact partakes of the same vague uncertainty that…fails to explain several important and well-authenticated epidemiological characteristics of the disease, and we must regard it at best as a weak working hypothesis."

81. **Robertson & Groves, 1924** - Exposed 100 healthy individuals to the bodily secretions from 16 different people suffering from influenza. 0 people of 100 whom they deliberately tried to infect with Influenza got sick.

82. **Bauguess, 1924** - "A careful search of the literature does not reveal a case in which the blood from a patient having measles was injected into the blood stream of another person and produced measles."

83. **The problem of the etiology of herpes zoster, 1925** - "Many other authors report entirely negative results following the inoculation of herpes zoster material into the scarified corneas of rabbits: Kraupa (18); Baum (19); LSwenstein (8), Teissier, Gastinel, and Reilly (20) ; Kooy (21) ; Netter and Urbain (22); Bloch and Terris (23); Simon and Scott (24); and Doerr (25). It is evident, therefore, that the results of attempts to inoculate animals with material from cases of herpes zoster must be considered at present to be inconclusive."

84. **W. L. Aycock, 1926** - "However, epidemiologic evidence of direct contact is scant … the proportion of direct contact cases [polio] is reduced to an extremely small figure."

85. **A. C. Nickel, 1926** - "Last summer, Dr. E. C. Rosenow and I saw about fifty-five cases of poliomyelitis within a radius of 75 miles of Rochester, and frequently we would see a case in a very secluded spot where contact infection was quite unlikely."

86. **Volney S and Chney M.D., 1928** - A study where it is clearly stated that cold is not infectious.

87. **E. B. McKinley, 1929** - "Poliomyelitis is essentially a warm-weather disease, yet cases are exceedingly rare in tropical countries. It does occur in the Tropics, however, but never in epidemic proportions … Epidemiologists state that infantile paralysis is usually more prevalent in sparsely settled communities than in the large cities."

88. **The Journal of the American Medical Association, 1930** - "The etiology [of polio] has not yet been determined. With regard to epidemiology, opinions vary. Direct contagion from the patients or from carriers has been accepted as the form of propagation of the disease, but the many cases in which the contagion is not in this form contraindicate this theory. Besides that, many research workers have reported cases of poliomyelitis in which patients did not transmit the contagion to their brothers, in spite of the intimacy and promiscuity in which they lived. Family epidemics are uncommon."

89. **Dochez et al, 1930** - Attempted to infect 11 men with intranasal influenza. Not a single person got sick. Most strikingly, one person got very sick when he accidentally found out that is what they were trying to do. His symptoms disappeared when they told him he was misinformed.

90. **W. L. Aycock, 1931** - "The epidemiologist encounters almost unparalleled difficulties in the study of the disease [polio]. It is only in the exceptional case that any relationship can be established with other cases. No practical tests have been available for the verification, for example, of suspected abortive cases or healthy carriers, and in the more general epidemiological features he is confronted with many seeming inconsistencies and paradoxes. Earlier students of the disease had little upon which to construct a conception of its epidemiology besides such observations as could be made in attempting to trace the infection from one case to the next occurring in the vicinity. One of the theories advanced was that the disease is transmitted by contact—a theory originating not so much in the observation of frequent contact itself but more in the suspicion that mild illnesses coincident with frank cases, not definitely diagnosable but suspected as abortive forms of the disease, aided in the dissemination of the infection. Failing even to find these in sufficient numbers to account for the spread of the disease, there was added the supposed transmission of the virus through healthy people. The incompleteness of the early evidence for contact and perhaps the lack of laboratory procedures for its verification did not place the contact theory on such a firm footing that it could not readily be thrown aside for any newly proposed theory, of which there have been many." […] "Some of the features of poliomyelitis which have seemed not to fulfill the criteria for contact transmission and which have been held as arguments against transmission in this manner are the infrequency of contact between cases, the infrequency of multiple cases in families or in institutions, the infrequency of transmission of the disease to nurses and attendants of cases and, in a more general way, the tendency to rural preponderance and the seasonal prevalence of the disease."

91. **International Committee for the Study of Infantile Paralysis, 1932** - "The natural insusceptibility of monkeys is also evidenced by the fact that [polio] contagion from one animal to another has never been demonstrated." […] "Secondary cases among nurses and attendants, as well as among patients in hospitals, are so rare as to surround the few that have been reported with the suggestion of coincidental infection rather than true secondary cases. This is to be expected at the present time when precautions are taken against the spread of poliomyelitis as against other contagious diseases. On the other hand, promiscuous spread was not noted before such precautions were taken. Batten, in the Lumleian Lectures, 1916 (B 7), says that after 30 years' experience at the Great Ormond Street Hospital for Sick Children, London, where cases of poliomyelitis in the acute stage were frequently admitted to the general wards, he vouches for the fact that no secondary cases had ever occurred there. Browning (B 35) likewise reports that at Kings County Hospital, Brooklyn, prior to the 1916 epidemic, cases were scattered through a general ward and cared for in the same way as the other children, no new case of infection developed. The personal laundry and

bedding of the patients were not specially handled. Collier (W 2) in recounting a similar experience at St. George's Hospital, London, where in spite of the lack of any precautions, secondary cases had never occurred." […] "The lack of obvious connection between cases of poliomyelitis is one of the striking and constant features of the epidemiology of the disease. In only a small percentage of cases is it usually possible to obtain a history of exposure to a case, and while connected cases do occur, infection seems never to proceed regularly from cases to contacts in a well-defined series … From the preceding, therefore, it seems that in only a small percentage of cases of poliomyelitis can direct exposure to other cases be traced." […] "It is, however, a fact that natural contagion from monkey to monkey has never been noted, although well monkeys have been caged with poliomyelitic monkeys at all stages of the disease … it is also true that no instance has ever been recorded where a worker in a laboratory whose duty was to handle infected monkeys, has been infected." […] "Later epidemiologists have not, however, found the evidence as to the contagiousness of poliomyelitis so clear-cut and conclusive, as that reported by Wickman. Certainly poliomyelitis, as we ordinarily encounter it in the United States, does not behave epidemiologically in accordance with the concepts that have become crystallized as to how a contagious disease should behave." […] "It has been characteristic of outbreaks of poliomyelitis that only a small percentage of cases can be traced to previous cases and that the increase in incidence among those known to have been exposed, whether in families, institutions, or hospitals, has been low. These features ... have been difficult to reconcile on the basis of contagion"

92. **K. F. Meyer, 1934** - "The extreme rarity of sister infections, even in the vicinity of definite cases in a family, is so striking that the epidemiologist reluctantly accepts the concept of contagion for the disease poliomyelitis." […] "Well monkeys caged with poliomyelitic animals, or laboratory workers exposed to these apes, do not contract the disease." […] "Lack of connection between cases of this disease is a constant epidemiologic feature; it is very difficult—usually impossible—to establish well-defined chain transmissions." […] "Seasonal incidence, lack of tendency to spread in congested centers, schools, etc., and the behavior of poliomyelitis in the tropics and in rural areas, are phenomena which do not harmonize with the concept of contact transfer."

93. **L. L. Lumsden, 1935** - "Painstaking efforts were made throughout the studies to obtain all traces of transmission of the disease through personal contact, but it appears that in this outbreak in Louisville evidence of personal association between the cases of poliomyelitis, suggestive of cause and effect, was no more common than that which might have been found if histories had been taken of personal association between cases of broken bones occurring in the city in the same period."

94. **O. Dahl, 1935** - "Poliomyelitis, is not contagious. Contrary to the generally accepted belief, one cannot catch infantile paralysis, you have to build it. No one has ever proven it to be a fact that the condition known as poliomyelitis is an entity, a specific something, that can be transmitted to another. No one ever caught this condition simply by being in the vicinity of those who may be suffering with this condition." […] "It would be just as reasonable for you to believe that drunkenness is contagious and that your children would become drunk by being in the vicinity of a group of people that were paralyzed drunk." […] "We do have some ill-informed M.D.'s who will tell you that Poliomyelitis is contagious for a period of about three weeks. It is hard to believe a reasoning mind can actually, and honestly believe such nonsense, for thousands of times children have slept together when one of them had infantile paralysis, and the others did not catch it. It is a rare incident that more than one or

two children, in the same family, are stricken; although they are in daily contact." […] "How is any thinking person to believe that there is a virus that in some way causes inflammation in the gray matter of the spinal cord of infants when this supposed to be virus has never been isolated? It is but a hypothetical something. A reasoning mind could better believe that the moon is made of cheese, for the moon at least has the shape and color of cheese. No one has ever seen, smelt nor felt, nor in any other way isolated this supposed to be poliomyelitis virus."

95. **B. Sachs, 1935** - "For many years I was in charge of a neurologic ward, and before there was much concern about poliomyelitis as an epidemic disease all the patients with neurologic conditions were kept in the general wards of the hospital. I cannot recall a single case of poliomyelitis that appeared to arise as a result of direct contagion from another patient in the ward. Those are very important facts. If the nasal orifice is the only portal of entrance it seems to me remarkable that there were not more cases in which the contagion was carried from one person to another, even allowing for the immunity of many persons."

96. **Thomas Francis Jr et al, 1936** - Gave 23 people influenza via 3 different methods. 0 people got sick.. They gave 2 people already "suffering from colds" the influenza who also did not get sick

97. **Burnet and Lush, 1937** - 200 people given "Melbourne type" Influenza . 0 people showed any symptoms of disease. 200/0.

98. **R. D. Defries, 1937** - "The fact that the disease [polio] occurs in epidemic form during the summer months … renders it difficult to explain fully the spread of the disease by contact."

99. **T. J. Meyers, 1937** - "There are some rather interesting characteristics of poliomyelitis epidemics. The disease is limited almost exclusively to certain seasons, late summer and early autumn … Contrary to what is commonly believed, poliomyelitis is rather infrequent in crowded districts and among children who frequent crowded places such as schools, churches, theaters, etc. The morbidity of rural districts exceeds that in larger towns, as much as a thousand fold."

100. **G. O. Barber, 1938** - "[Polio] is definitely not highly infectious. Until recently, cases were nursed from the start in general wards of general hospitals, and there have been no well-authenticated cases of infection to contacts. Certain of the cases in this recent outbreak occurred in crowded families and were not reported until the illness had been in the paralytic stage for several days. During this time other children had been sleeping every night in the same bed as the paralyzed child, and in no case was one of these contacts affected later."

101. **H. A. Reimann, 1938** - "There is no obvious contagion in poliomyelitis of man."

102. **J. R. Paul, 1938** - "As to the possible means whereby the virus may spread through a community there is still no convincing evidence favoring any particular route … Nor has there been new or convincing work to explain satisfactorily the summer incidence of the disease [polio] or its higher prevalence in rural areas than in urban communities."

103. **C. C. Dauer, 1938** - "No direct or indirect association could be traced in the majority of [polio] cases even after the most careful and searching investigations."

104. **L. L. Lumsden, 1938** - "We do not know…with certainty, whether the disease [polio] is infectious; We do not know…whether it is directly or indirectly communicable from person to person." […] "The general and usual epidemiological features of the disease all appear

opposed to the hypothesis that poliomyelitis is a contagious disease spread among human beings by nose-to-nose or any other direct personal contact." [...] "The efforts to reconcile the contagion hypothesis with the geographical distribution, seasonal incidence and other factual features of the disease appear to some of us more and more to compose a structure comparable to a pyramid of straw with the big end up. The contagion hypothesis may be right, but proof of it is yet lacking." […] "What is the reason for such regional distribution of the disease we call poliomyelitis? We simply do not know. None of the usual hypotheses of spread—the contagion or other—appear to apply to it to a completely satisfactory degree." [...] "It is quite usual in small [polio] outbreaks in rural counties for individual cases to develop in separate homes three or four miles apart without there being any evidence of direct or indirect personal contact having operated between persons afflicted."

105. **K. F. Meyer, 1939** - "Dr. Geiger: Before we begin to discuss the disease itself in humans, I feel we should have a clearer understanding of the laboratory side. Doctor Meyer, will you summarize the present status of poliomyelitis in the field of experimental research? Dr. Meyer: That is a difficult question to answer directly, since we are still baffled by the simple question, "What is the disease agent in infantile paralysis?" … We do not know whether it is a living germ or something else growing in the cells of the brain. We cannot isolate it like a germ in a test tube, and it is too small to be seen with a microscope. There is only one species of animal—the Old World monkey—in which we can induce the same disease as seen in children. Yet these monkeys are not as susceptible as man, since there is no record of one monkey catching the infection from another monkey by exposure." […] "Dr. Geiger: Well, if we do not know a great deal about the disease itself, do we know anything about the mode of its spread? Dr. Meyer: In answering this question, let me say frankly that theories are numerous, but facts are few. It is assumed that the disease agent is discharged from the mouth and nose and enters the body through the same channels. Thus, direct exposure of the healthy with the diseased should convey the infection; but the lack of spread in families, schools and crowded places, throws doubt on this explanation. The fact that several cases may occur in the family indicates some hereditary disposition may play a role. But this does not explain why infantile paralysis appears toward the end of the summer in the temperate zone, and is rarely ever seen in the tropics."

106. **K. F. Meyer, 1939** - "There is no record of one monkey catching the infection [polio] from another monkey by exposure."

107. **Burnet and Foley, 1940** - Attempted to experimentally infect 15 university students with influenza. The authors concluded their experiment was a failure.

108. **Thomas Francis Jr, 1940** - Gave 11 people "Epidemic Influenza." 0 people got sick.

109. **T. D. Deakin, 1940** - "One of the most striking features of poliomyelitis is the lack of obvious connection between cases … it is only in a small percentage of instances that a definite series of cases and contacts may be secured. The New York City Health Department investigated carefully the epidemic in Brooklyn in 1931. Of the first 500 cases, in 31 or only 6.2 per cent was evidence of contact with previous cases established. No proof of association with other cases could be obtained in any of the remaining 93.8 per cent. In an epidemic of 100 cases in Glasgow in 1928, in only two cases was it possible to trace any direct connection between the cases." […] "[Ivar Wickman's] reports of several small epidemics in Sweden in the early 1900's still stand as the best evidence we have of the contagiousness of poliomyelitis."

110. **A. I. Kendall, 1940** - “At times numerous [polio] cases would appear suddenly in a limited area. Sometimes cases appeared simultaneously miles apart with no detectable contact of one with the others. In epidemic areas it was exceptional rather than the rule to discover more than one patient in a single family where ordinarily intimate contact should produce multiple infections. This irregular discontinuity between individual cases of poliomyelitis, together with an unequivocal time space relation in their incidence, was not wholly in accord with the usual pattern of a contagious disease.” […] “There is no evidence of spread of poliomyelitis among doctors, nurses or ward attendants in hospitals where large numbers of cases of flaccid paralysis may be interned.”

111. **John Toomey, 1941** - A veteran polio researcher: "no animal gets the disease from another, no matter how intimately exposed."

112. **A. I. Kendall, 1945** - “The epidemiological facts of poliomyelitis are these: … (2) A majority of cases of clinically diagnosable poliomyelitis (polioparalysis) occur sporadically, with no history of contact with previous cases. (3) Two cases of polioparalysis in one family are unusual, even though no precautions are taken to prevent cross infection. (4) Clinically diagnosable cases of poliomyelitis (polioparalysis) show little tendency to spread, even in schools or other places of public gathering. (5) Incidence of polioparalysis is no greater among doctors and nurses, in intimate contact with acute cases than it is among the civil population, even though the former are exposed freely to infection.” […] “Polioparalysis is not contagious.”

113. **A. B. Sabin, 1947** - “It is remarkable that, unlike certain other infections of childhood, the epidemics of paralysis occur during the very months when the children are away from school.”

114. **D. M. Horstmann, 1948** - “The fact that poliomyelitis is a summer disease has always been an obstacle in the acceptance of simple person-to-person contact as an explanation of its epidemic spread. The sudden burst of cases with the appearance of warm weather repeats itself again and again; and, if summer comes early, so do epidemics … Why, if contact alone is the answer, does not the virus spread in winter as do other contact diseases? … crowding and close quarters in winter seem more suitable for its spread than do summer conditions.” […] “In spite of all the information collected by many investigators in many lands we still cannot say … how it [polio] is spread”

115. **E. B. Shaw & H. E. Thelander, 1949** - “The epidemiology of the disease [polio] remains obscure. There has been a tendency to depart from an early theory that the disease spreads by means of direct contact.”

116. **A. B. Sabin, 1949** - “Among students of the epidemiology of poliomyelitis there are now three main views on the mode of spread of the disease, all regarding the human being as the primary reservoir of the virus. These views or hypotheses, for the sake of emphasis, may be called the respiratory, the alimentary, and the alimentary plus the nonbiting flies. The “respiratory” hypothesis postulates that transmission occurs when the virus is breathed out or otherwise expelled from the nose or mouth of one person and is then breathed in by another … According to the “alimentary” hypothesis the virus is transmitted by being put into the mouth as by contaminated fingers or food … Neither the “respiratory” nor the “alimentary” hypothesis attempts to account for the fact that 90 per cent or more of the cases and most of the epidemics occur during the late summer and autumn.”

117. **W. J. McCormick, 1950** - “The disease [polio] rarely attacks more than one member of a family, and cases developed by contact are conspicuously rare.”

118. **R. R. Scobey, 1950** - “The theory that poliomyelitis communicable has never been able to account for such anomalous and contradictory facts as the victimization of individuals who have had no contact with active cases; the non-communicability to doctors, nurses, and ward attendants; the absence of communicability to patients in hospitals and to individuals in communities when quarantines are not established; the rarity of multiple cases in the same family even where a child with poliomyelitis is known to have slept with another child; its greater incidence in small communities than in large cities where crowding exists and where, consequently, poliomyelitis should extort a staggering number of victims; and the increase in epidemics of this disease in spite of improved hygiene and education regarding precautionary prophylaxis.”

119. **C. Armstrong, 1950** - “We are therefore confronted with the difficulty of explaining why poliomyelitis, a disease transmitted by close contact, should be most prevalent in that portion of the year when people spend most of their time in the open and why it should tend to wane rapidly when cool weather induces people to congregate indoors in search of warmth.”

120. **Science News Letter, 1950** - “Strict and heavy quarantines for infantile paralysis does not stop polio epidemics, health and poliomyelitis authorities agree. All attempts to stop polio by quarantine have failed and authorities now consider it foolish to enforce it.”

121. **A. Taylor-Smith, 1950** - “It is a most frequent thing to find that only one of a large family of children falls a victim [of polio], or that one only has been picked out of a large school class.”

122. **A. B. Sabin, 1951** - “There is no evidence for the transmission of poliomyelitis by droplet nuclei.”

123. **A. L. Hoyne, 1951** - “There is nothing about poliomyelitis which seems stranger than its epidemiologic character … Considering that nearly all of the common acute infectious diseases predominate in the fall and winter or winter and spring, seasons when life is principally within doors and schools are in session, we are forced to ponder why poliomyelitis is epidemic in the summer.” […] “Can it be that the disease is transmitted only by person-to-person contact? It does not seem likely.” […]“It is a matter of extreme rarity for a patient to give a history of exposure to a known case of poliomyelitis.” […] “There is little to indicate that isolation has been a controlling influence in the spread of the disease during epidemics.” […] “Since the virus may be found in the intestinal tract for thirty-five days or possibly longer after onset of the disease it would seem logical to disinfect all body discharges before their disposal. However, in the Cook County Contagious Disease Hospital where the latter procedure has not been used there has never been a doctor, intern, nurse or any other member of the personnel who contracted poliomyelitis within a period of at least thirty-five years, nor has any patient ever developed poliomyelitis after admission to the hospital.”

124. **A. B. Sabin, 1951** - “One of the important unsolved problems in poliomyelitis is why in temperate zones approximately 80 to 90 per cent of the cases occur during four months of the year in late summer and early autumn.”

125. **R. R. Scobey, 1951** - “The first, and by all means the foremost fact that must be conclusively established is whether or not poliomyelitis is actually an infectious contagious disease, as has been commonly assumed and stated in the public health law. This assumption, it must be admitted, is almost entirely based on the results of animal experiments rather than on clinical investigations.” [...] ”Although poliomyelitis is legally a contagious disease, which implies that it is caused by a germ or virus, every attempt has failed conclusively to prove this mandatory requirement of the public health law. The manifest truth that we must take into consideration is that progress in poliomyelitis investigations has been impeded by this prematurely formulated public health law.”

126. **J. A. Toomey, 1952** - “Polio has not been proved to be contagious.”

127. **P. M. Holst, 1952** - “All our experience argues that the disease [polio] does not spread from contact with the clinically sick.”

128. **R. R. Scobey, 1952** - “It is extremely difficult to understand how a human can contract poliomyelitis from another individual through dissemination of a virus by contact, carriers, excrement, unclean hands, unwashed fruits and vegetables, flies, etc. when a healthy animal in the same cage with an ’infected’ animal, exposed to all of these natural factors, remains unaffected.” [...] “The fact that an extensive epidemic of poliomyelitis was prevailing in the states of New York and Massachusetts in 1907, aroused the suspicion that the disease was infectious and communicable; it was therefore incorporated into the Public Health Law as such. However, conclusive evidence of contagiousness was not established during that epidemic nor in subsequent ones.” [...] “In addition to the failure to prove contagiousness of human poliomyelitis, it has likewise been impossible to prove contagiousness of poliomyelitis in experimental animals.”

129. **B. Eskesen & B. Glahn, 1953** - [Epidemic of polio in Greenland] “It has not been possible to find definite means of contagion or disease spreaders.” […] “Means of contagion have not been proved.”

130. **R. R. Scobey, 1954** - “It is now known that the most intimate contacts—such as healthy and sick individuals in one bed, the attendance of physicians and nurses upon the sick, the use of unclean linen, clothes, or beds, unsanitary conditions, insects and animals, post-mortem examinations of poliomyelitis victims, and other factors—have in no wise contributed to the spread of the disease.”

131. **J. F. Edward, 1954** - “According to British Law, an individual is innocent until proven guilty. Applying the same legal dictum to Poliomyelitis its cause was adjudged, between 1905 and 1911, to be contagious and infectious; this, in the absence of knowledge of its cause of spread, its only proven crime being that it could become epidemic . It was declared to be viral in origin. This implication by the Public Health Laws of many of the Provinces of Canada and of the States of the Union made Poliomyelitis legally an infectious contagious disease, and thereby opened the door for research, considering the disease as such; and closed the door to research along lines other than that which has been publicised and financed by endowment in the past forty years. […] “Viewing the disease from a clinical standpoint, in Manitoba’s Epidemics of 1952 and 1953 one notes that:

    1. Few of our cases had a history of contact with an earlier case.

    2. Few of our cases transmitted the disease to family contacts.

3. Few Medical Personnel in attendance upon Polio patients acquired the disease or transmitted it to their families."

132. **R. R. Scobey, 1954** - "Multiple cases in families present the nearest approach to the grouping of epidemiologically connected [polio] cases. There is no conclusive proof that the disease spreads under such circumstances like a contagious or infectious disease." […] "The seasonal incidence of epidemics of poliomyelitis has always been one of the puzzling features of the disease. Epidemics are reported chiefly in the temperate zones."

133. **A. L. Hoyne, 1954** - "But we may ask is poliomyelitis actually contagious? … If poliomyelitis is a contagious disease as first maintained by Wickman about 1905 it is strange indeed that no one ever contracted it at County Contagious Hospital…where, it may be mentioned, the wearing of face masks is optional." […] "Quarantine and isolation of patients have had no discernable effect in the control of epidemics."

134. **A. L. Hoyne, 1957** - "Is it not strange that we seldom hear of any hospital personnel who come in frequent contact with poliomyelitis patients during the acute stage contracting the disease? If poliomyelitis is contagious why has no one, during a period of 40 years, ever acquired the disease at Cook County Contagious Disease Hospital? In addition to the regular staff of graduate nurses, a new group of students is assigned for duty each month. Also, clinics for medical students are held almost daily. Recently, a somewhat similar experience was reported in the Baltimor city hospitals. There it was believed that the personnel must have had "inapparent" poliomyelitis and possessed antibodies which afforded protection. It was decided to investigate the matter and Wehrle conducted a highly scientific study. He found that among 75 of the personnel, which included nurses, nearly one-third lacked sufficient antibody to provide protection. In some cases, there was no antibody. If antibody is unnecessary for immunity what is the explanation for failure to acquire the infection when intimately exposed?"

135. **Douglas Gordon et al, 1975** - This study gave 10 people English type Influenza and 10 people a placebo. The study was negative. Most telling is they admit that mild symptoms were seen in the placebo group, proving that the inoculation methods cause them.

136. **N. Nathanson & J. R. Martin, 1979** - "Poliomyelitis has undoubtedly received as much attention from epidemiologists as any other viral disease of man. Yet despite intensive study over a century, many of the salient epidemiologic features of this infection must still be considered enigmas. Even some of the accepted dogmas about poliomyelitis can be debated as perhaps erroneous." […] "No good explanation was ever documented for the occurrence of epidemics." […] "One of the most characteristic features of poliomyelitis in the United States is its very marked seasonality … The regularity of this pattern over many years suggested that it was governed by a mechanism which should be ascertainable. Nevertheless, the underlying explanation has remained elusive."

137. **Beare et al 1980** - (refer to reference 6 in the linked paper). Quote from John J Cannell, 2008 as follows - "An eighth conundrum – one not addressed by Hope-Simpson – is the surprising percentage of seronegative volunteers who either escape infection or develop only minor illness after being experimentally inoculated with a novel influenza virus."

138. **Nancy Padian, 1996** - A study which followed 176 discordant couples (1 HIV positive and the other negative) for 10 years. These couples regularly slept together and had unprotected

sex. There were no HIV transmissions from the positive partner to the negative partner during the entirety of the study.

139. **John Treanor et al, 1999** - Gave 108 people Influenza A. Only 35% recorded mild symptoms such as stuffy nose. Unfortunately, 35% of the placebo control group also developed mild symptoms proving the methods of inoculation are causing the symptoms.

140. **Bridges *et al*, 2003** - "Our review found no human experimental studies published in the English-language literature delineating person-to-person transmission of influenza... Thus, most information on human-to-human transmission of influenza comes from studies of human inoculation with influenza virus and observational studies."

141. **The Virology Journal, 2008** - ”There were five attempts to demonstrate sick-to-well influenza transmission in the desperate days following the pandemic [1918 flu] and all were ’singularly fruitless’ … all five studies failed to support sick-to-well transmission, in spite of having numerous acutely ill influenza patients, in various stages of their illness, carefully cough, spit, and breathe on a combined total of >150 well patients.”

142. **Public Health Reports, 2010** – “It seemed that what was acknowledged to be one of the most contagious of communicable diseases [1918 flu] could not be transferred under experimental conditions.”

143. **T.C. Sutton et al, 2014** - “Throughout all ferret studies, we did not observe an increase in sneezing, and a febrile response (i.e., elevation of body temperature) was inconsistent and was not a prominent feature of infection.”

144. **Jasmin S Kutter, 2018,** - Our observations underscore the urgent need for new knowledge on respiratory virus transmission routes and the implementation of this knowledge in infection control guidelines to advance intervention strategies for currently circulating and newly emerging viruses and to improve public health.
- There is a substantial lack of (experimental) evidence on the transmission routes of PIV (types 1–4) and HMPV.
- Extensive human rhinovirus transmission experiments have not led to a widely accepted view on the transmission route.
- However, until today, results on the relative importance of droplet and aerosol transmission of influenza viruses stay inconclusive and hence, there are many reviews intensively discussing this issue.
- Despite this, the relative importance of transmission routes of respiratory viruses is still unclear, depending on the heterogeneity of many factors like the environment (e.g. temperature and humidity), pathogen and host.

145. **Jonathan Van Tam, 2020** - Conducted these human trials of Flu A in 2013. 52 people were intentionally given "Flu A" and made to live in controlled conditions with 75 people. 0 people contracted the illness.

146. **J.S. Kutter, 2021** - “Besides nasal discharge, no other signs of illness were observed in the A/H1N1 virus-positive donor and indirect recipient animals.” The animals were subsequently euthanized after the animals experienced what the scientist describes as having breathing difficulties (no further details were given to describe their condition). **Refer to Note 1.*

147. **Ben Killingley, 2022** - Gave 36 people what he considered to be purified Covid Virus Intranasally. The Results: Nobody got sick. **Refer to Note 2.*

148. **N. Shetty et al, 2024** - The study reported that illness was moderate, with upper respiratory symptoms dominating. Most notable symptoms include Nasal/Sinus Congestion and Rhinorrhea (nasal discharge) which can, as described in Note 1, be induced through nasal swabs. This brings into question the results of the study as the only other forms of viral detection were indirect methods. **Refer to Note 3.*

Notes

***Note 1 - Jasmin Kutter, 2021:**

- From the Results section: "Throat and nasal swabs were collected from the donor and indirect recipient animals on alternating days." This procedure alone can induce nasal discharge [25, 26, 27, 28], which was the only "sign of illness" noted in this study.

***Note 2 - Ben Killingley, 2022:**

- Ben Killingley also conducted a study around 2010 in which he inoculated individuals in a room with 75 others, some wearing masks and others as controls. Not a single person tested PCR positive. Some of his previous studies include those from 2011, 2019, and 2020. It is assumed that his latest 2022 study is a follow-up to employ methods that overestimate viral disease as those discussed in Section 2.3. Additional notes on the referenced study include:
    - 10 participants received Remdesivir which is considered nephrotoxic.
    - Illness was assessed using PCR tests, which are not definitive indicators of disease as they can yield positive results in asymptomatic cases.
    - Even if a nasal discharge post-swabbing is considered indicative of COVID-19, a 50% outcome to a direct challenge does not suggest causation, which would need to be at least 90%.
    - The inoculation methods used during the study could cause nasal congestion/discharge, which was a measure of disease transmission [25, 26, 27, 28].
    - No participants were administered Regeneron due to none of them exhibiting symptoms of illness.

***Note 3 - Symptoms in Viral Transmission Studies**

- Indirect methods of virus transmission detection, such as viral RNA detection in nasopharyngeal swabs, saliva, stool, urine samples, respiratory aerosols, environmental swabs, and seroconversion assessments, provide valuable supplementary information but have limitations. These methods often detect viral RNA rather than infectious viruses, are susceptible to contamination and false positives, and lack clinical context. Symptoms, on the other hand, offer direct evidence of disease, are clinically relevant, guide treatment decisions, and correlate with infectiousness. Therefore, while indirect methods are useful, symptoms are a more valuable source of information for understanding and managing viral infections (also refer to Section 2.3 and 2.4).

3 - Summary

The findings in this paper highlight significant gaps in the empirical basis for viral transmission, raising critical implications for public health policies. Current strategies, including vaccination programs, quarantine measures, and antiviral treatments, rest on the assumption that viruses are reliably transmissible agents of disease. However, the lack of consistent evidence supporting natural person-to-person transmission challenges this foundational belief.

As demonstrated, historical and contemporary studies often fail to replicate natural transmission dynamics under experimental conditions. Instead, virology frequently relies on artificial inoculation methods that bypass natural barriers and provoke reactions unrelated to actual viral activity. Such methods, while yielding measurable effects, do not confirm the infectious nature of the viruses under investigation. This disconnect between experimental outcomes and real-world scenarios undermines the reliability of the evidence upon which public health measures are based.

Furthermore, the critique of methodological approaches underscores the ethical and practical dilemmas posed by interventions found on inconclusive data. For example, policies advocating widespread vaccination or prolonged quarantines assume a high degree of transmissibility and pathogenicity, which have not been conclusively demonstrated. The resulting allocation of resources and enforcement of societal restrictions may therefore lack sufficient justification, raising concerns about the proportionality and efficacy of such measures.

Additionally, the review of experimental studies reveals a pattern of over-reliance on proxy indicators, such as the detection of viral genetic material, which may infer but not confirm infectivity. This reliance has far-reaching consequences for diagnostic practices, surveillance systems, and the evaluation of intervention efficacy. For instance, policies designed to mitigate respiratory virus transmission, such as mask mandates or contact tracing, may overestimate their impact if the underlying evidence for airborne or contact-based transmission is flawed.

To address these concerns, this review advocates for a reevaluation of virological methodologies and the assumptions guiding public health strategies. By prioritizing studies that validate natural transmission mechanisms and by refining experimental designs to reflect ecological validity, virology can contribute more robust and actionable insights. Policymakers must adopt a critical stance toward existing evidence, ensuring that interventions are proportionate, ethically grounded, and supported by rigorous science. Only through such a reassessment can public health initiatives achieve greater transparency, accountability, and effectiveness.

4 - Acknowledgements

I would like to extend my heartfelt gratitude to everyone who contributed to the completion of this study. First and foremost, I thank Jamie Andrews (website - https://controlstudies.substack.com/) and Aldhissla (website - https://aldhissla.substack.com/) for their meticulous work in reviewing many of transmission papers discussed in this study. Their dedication and expertise have been instrumental in shaping the critical analysis presented here.

I also wish to express my appreciation to my colleagues and mentors for their invaluable guidance and support throughout this research. Their insightful feedback and encouragement have greatly enhanced the quality of this work.

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