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# DID WIRED AND WIRELESS TELEGRAPHY AND TELEPHONY CAUSE THE EMERGENCE OF EPIDEMIC POLIOMYELITIS ?

Vincent Lauer*

Abstract: epidemic Poliomyelitis is one of the diseases that emerged as major public health problems during the 20th century, and can thus be suspected of being caused by artificial electromagnetic waves. Detailed examination reveals that early epidemic outbreaks coincided with changes in emissions of artificial electromagnetic waves, including the intentional emissions of wireless telegraphy and the unintentional emissions of wire telephony. The fundamental change from irregular outbreaks to yearly epidemics in the US also coincided with the onset of wireless voice communications. These facts provide strong support to the idea that epidemic Poliomyelitis was triggered by exposure to electromagnetic waves and had a high degree of sensitivity to exposure. Examination of further evidence reveals that Poliovirus PV3 is the ancestor of PV1, PV2 and two other group C enteroviruses, and that PV3 was the dominant Poliovirus serotype in 1875, before the onset of most electromagnetic emissions except wire telegraphy. The time coincidences of Poliomyelitis epidemiology and electromagnetic waves emissions, together with the evolution of Poliovirus, bring strong support to the hypothesis that exposure to electromagnetic fields triggered both epidemic Poliomyelitis and the diversification of Poliovirus.

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## 1 Introduction.

During the 20th century, most diseases that had plagued the humanity during its recorded history were largely overcome. Plague, Tuberculosis, Cholera and Syphilis were almost eliminated in developed countries through improved sanitation and use of antibiotics. Smallpox was eradicated by vaccination and Measles was largely overcome, also by vaccination. Although measles caused great damages after being introduced in the New World, it was relatively harmless in populations that were used to it and thus immunized. The only viral disease which was historically a major killer was thus Smallpox.

In sharp contrast to these remarkable achievements, several diseases emerged as major killers that were either inexistent or rare or benign in previous centuries: Influenza (which was occurring in outbreaks prior to the 20th century but had a much lower death rate than the pandemic of 1918), AIDS, and Poliomyelitis were major issues. The epidemiological changes that affected these viral diseases were wholly unexpected. Until recently, little effort was made to convincingly explain them. Influenza was attributed to increased travel (yet travel may explain a faster dissemination of the virus but not its emergence). AIDS is often attributed to changes in sexual behaviors (but homosexuality also existed before the 20th century). Poliomyelitis was attributed to improved sanitation (a somewhat unexpected explanation; also, why was Poliomyelitis also rare, before the 20th century, in the countryside where sanitation is not a problem or in ancient civilizations which had good sanitation?). Each disease was given its particular explanation, which in each case is unsatisfactory. No effort was made to seek for a common cause for so major an epidemiological change in such a small amount of time. Unlike the epidemiological changes that followed the discovery of America, the epidemiological changes that took place in the 20th century remained without a rational explanation.

It was recently proposed that AIDS was caused by the onset of artificial electromagnetic waves (Lauer 2015b). However, merely on the basis that a thousands years – old status quo was broken in the 20th century, with no consistent explanation proposed other than causation by the onset of electromagnetic waves, essentially all major emerging diseases of the 20th century (as compared to the remainder of recorded human history) are highly suspect of being due in whole or in part to the onset of artificial waves. This brings in a rational explanation to a change which is otherwise not understood.

One of these diseases is Poliomyelitis. In developed countries, it was overcome by vaccination, so that interest in this disease has now faded out – but were it not for the vaccine, poliomyelitis would probably be a major health concern today.

In this paper, I explore the hypothesis that the emergence of epidemic Poliomyelitis in the 20th century was due to the onset of artificial electromagnetic waves (in combination with Poliovirus), a possibility which results from the known effects of electromagnetic waves on the immune system (Lauer 2013, 2014a,b, 2015a). I discuss the history of epidemic

Poliomyelitis in the 19th and early 20th century, and the evolution of Polioviruses, both of which support the proposed hypothesis.

# 2 History of Poliomyelitis in relation to exposure to electromagnetic waves.

## 2.1 Calculation of p-values for coincidences.

Let us consider that on N years there are E timed events of polio epidemics and T timed events in onset of electromagnetic systems, of which Te coincide with a polio epidemic and Tn do not coincide with a polio epidemic. The probability of observing the specified values of Te and Tn is:

$$Pex(Tn, T, E, N) = \frac{C_E^{Te} C_{N-E}^{Tn}}{C_N^T}$$

The probability ("p-value") of observing Tn or less than Tn timed events that do not coincide with a polio epidemic is:

$$P(Tn; T; E; N) = \sum_{S=\max(0,T-E)}^{Tn} \frac{C_E^{T-S} C_{N-E}^{S}}{C_N^T}$$

This p-value represents the probability of observing by chance at least as many coincidences as observed. Coincidence of a timed event with a polio epidemic is defined as the timed event and the polio epidemic being in the same year.

When there is uncertainty over the date of the timed event in onset of electromagnetic waves, it is possible to consider 2-year periods, in which case N becomes the number of 2-year periods (half the number of years) and a timed event in onset of electromagnetic waves is considered coincident with a poliomyelitis epidemics if the polio epidemic starts during the two-year period corresponding to the timed event.

## 2.2 Sweden/Norway

Sweden and Norway (which were united until 1904) appear to have been hit early by Poliomyelitis and it also seems that the evolution of the disease was followed carefully since very early, with even small epidemics being reported.

Major epidemics in Sweden/Norway (Figure 1, Figure 2) tend to occur following major changes in wired telephony and radiotelegraphy systems. Epidemics are rare and decreasing in intensity in the interval periods. Effects appear to be mostly transient, as they are stronger the first year of a triggering event but diminish or disappear thereafter.

Considering the period of 1876 (invention of telephony) to 1911, and considering only major timed local events, there are 5 timed local events in emissions of electromagnetic waves:

- Onset of telephone network for administrations in 1881
- Onset of world's major telephone exchange in 1887
- Arrival of ships equipped with wireless telegraphs in 1889
- Onset of wireless telegraph coastal stations in 1905
- Onset of wireless telegraph coastal stations in 1911.

In epidemic Poliomyelitis there are local events:

- 8 epidemics between 1876 and 1904
- The epidemic of 1905
- The epidemic of 1911 (epidemics of 1906 to 1910 are ignored because they were progressively decreasing and can thus be connected to the 1905 epidemic.)

So during a period of 36 years (N=36) there were a total of 5 timed local events in emission of electromagnetic waves (T=5) and 10 timed events in poliomyelitis epidemics (E=10). Each timed local event corresponded to an epidemic of poliomyelitis (Tn=0). Using the formula of section 2.1 yields:

P(Tn=0; T=5; E=10; N=36)=0.0007

The p-value of 0.0007 is highly significant (usually p-values of 0.05 and below are considered significant).

How is this result influenced by possible errors?

If there are 4 additional local events in transmission of electromagnetic waves which do not coincide with timed local event in epidemic poliomyelitis the p-value becomes:

P(Tn=4;T=9;E=10;N=36)=0.046

Which remains significant. Therefore up to 4 local events in emission of electromagnetic waves not coinciding with poliomyelitis epidemics may have been ignored without affecting the significance of the observation. The conclusion as to the fact that local events in wired telephony and wireless telegraphy caused epidemic Poliomyelitis is thus little sensitive to the possibility that some events may have been ignored.

How is this result modified if we take into account international "firsts" ?

We could have chosen to additionally take into account all international "firsts" in wireless telegraphy as events (because "firsts" are more likely to have distant consequences, see section 2.6): the experiences of Calzecchi-Onesti (1883), Hertz (1886), Marconi (1895). Then we have N=36, E=10, T=8, Tn=1. This yields the p-value:

P(Tn=1;T=8;E=10;N=36)=0.0001

Note that precise dates for international "firsts" in wireless telegraphy are determined based on local observations (section 2.3) in the countries where such firsts originated: there is uncertainty, for example, on whether the Calzecchi-Onesti experiment could trigger Poliomyelis in 1883 or 1884, but observation of epidemic Poliomyelitis in Italy in 1883 shows that the appropriate date is 1883.

Adding 7 more events in electromagnetic waves emissions which do not coincide with epidemic Poliomyelitis outbreaks yields P(Tn=8; T=15; E=10;N=36)=0.04 which is still significant.

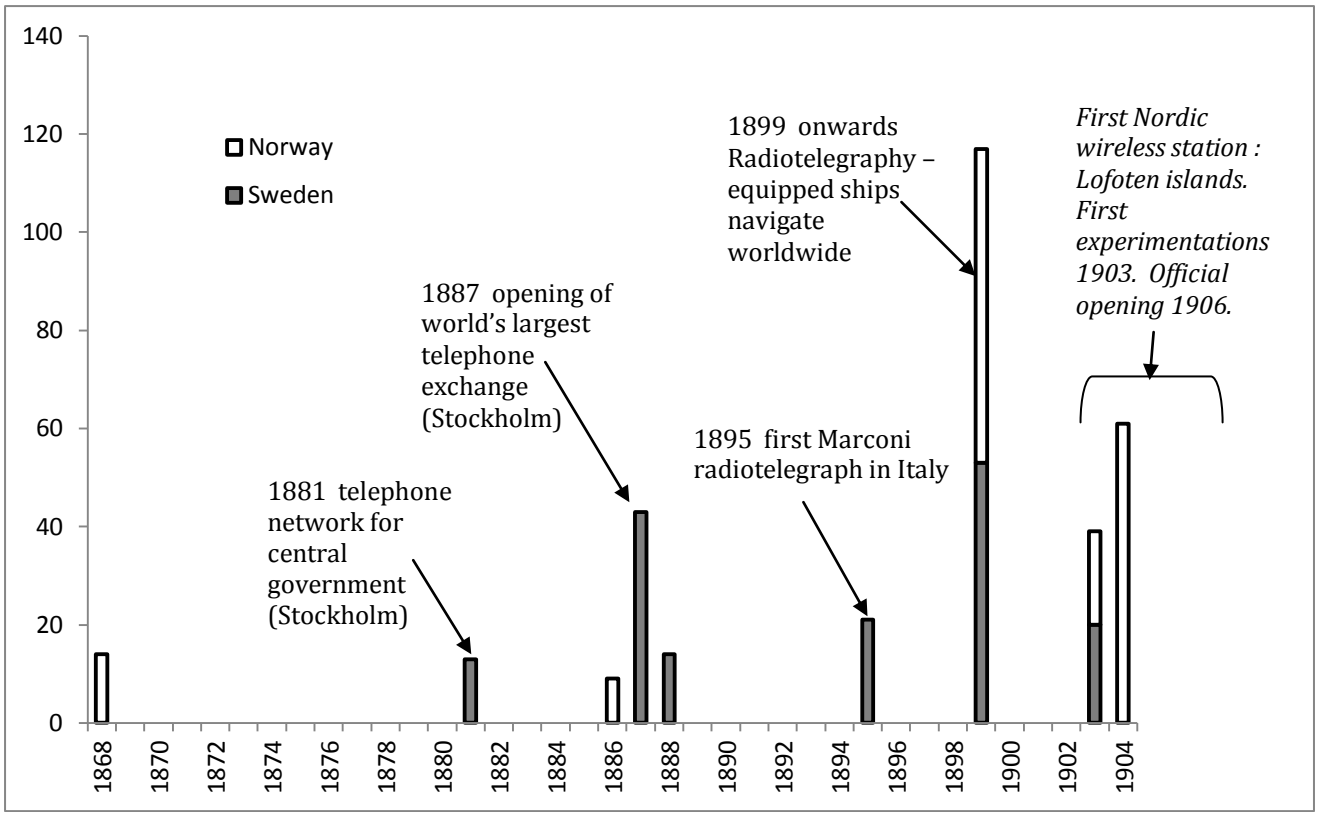



**Figure 1: Poliomyelitis epidemics in Norway/Sweden, number of cases, 1868-1904. Note that the Lofoten islands are far north, away from main cities; but one of the two epidemics of 1904 was in Aafjorden on the north coast, 300 km from Oslo over land and 400 km from the Lofoten over sea. Based on (Lavinder et al 1918) and Appendix.**

It is aso possible to proceed to an analysis restricted to events in wireless telegraphy (including international firsts), over the same period. Then P(Tn=1;T=6;E=10;N=36)=0.003, which is still significant. Further, up to 3 non-coinciding events can be added, yielding P(Tn=4; T=9;E=10;N=36)=0.046, which remains significant. The more specific conclusion that wireless telegraphy triggered outbreaks of epidemic Poliomyelitis is thus robust and reliable. Restricting the analysis to local events in wireless telegraphy yields P(Tn=0;T=3;E=10;N=36)=0.017 which is significant but quite sensitive to the possibility of having neglected an event: adding one non-coinciding event yields P(Tn=1;T=4;E=10;N=36)=0.057.

To analyze specifically the events related to wired telephony, one can consider the 1876-1894 period (19 years between invention of telephony and invention of wireless telegraphy). Then P(Tn=0;T=2;E=4;N=19)=0.035, which is still significant. Adding one non-coinciding event yields P(Tn=1;T=3;E=4;N=19)=0.097 and adding 2 non-coinciding events yields P(Tn=2;T=4;E=4;N=19)=0.18.

However, if we assume that wireless telegraphy including international "firsts" could trigger Poliomyelitis epidemics, then we must exclude from the analysis of wired telephony the years 1883 and 1886 corresponding to the Calzecchi-Onesti and Hertz experiments respectively, yielding P(Tn=0; T=2;E=3;N=17)=0.022. Adding one non-coinciding event yields P(Tn=1; T=3;E=3;N=17)=0.063 and adding 2 non-coinciding events yields P(Tn=2; T=4;E=3;N=17)=0.12.

The conclusion that wired telephony triggered Poliomyelitis epidemics is thus less robust than the conclusion as to wireless telegraphy; yet it is significant. Validity of the conclusion depends on whether or not essential events may have been ignored.

The most essential event which may have been ignored is the worldwide or local first use of wired telephony. But unlike the first uses of wireless telegraphy, the first uses of wired telephony did not appear to trigger any Poliomyelitis epidemic worldwide, including in the US where wired telephony was invented. This may have been due to a more progressive onset of wired telephony, starting with a few local lines and progressively increasing coverage: since effects were transient (see section 2.7) a progressive onset is likely to eliminate corresponding Poliomyelitis epidemics. In any case, the lack of Poliomyelitis epidemic worldwide (including the US) following onset of wired telephony confirms that this event was not a major triggering event worldwide and was thus unlikely to be a major triggering event in Sweden. Indeed, the events of 1881 and 1887 in Sweden characteristically departed from the usual progressivity which dominated in the US and in most other nations, due to political decisions resulting in unusually fast network growth. However, should such an initial event have been ignored, the conclusion on wired telephony remains near significant (p=0.063), so that it is likely that wired telephony triggered Poliomyelitis epidemics.

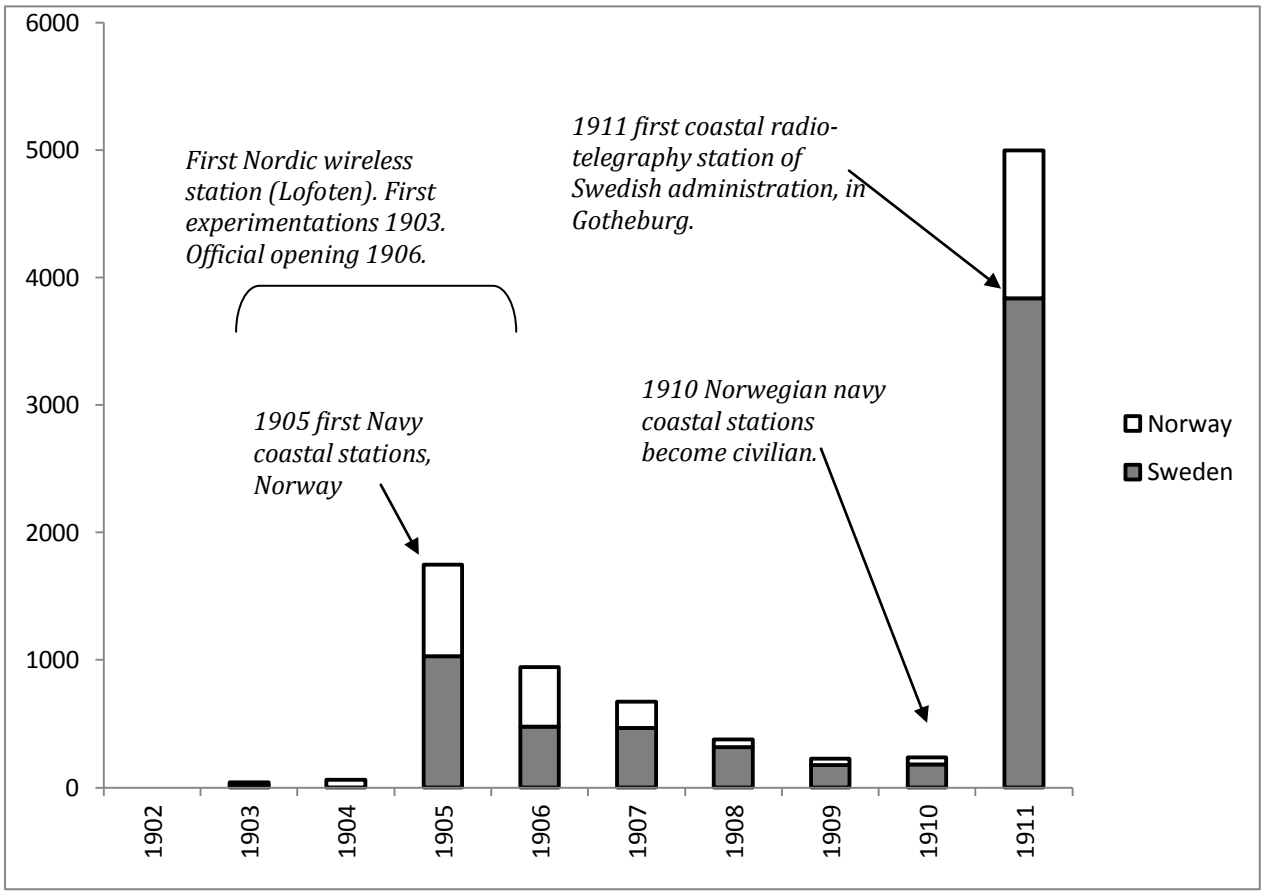



**Figure 2: Poliomyelitis epidemics in Norway/Sweden, number of cases, 1902-1911. Based on (Lavinder et al 1918) and Appendix.**

| Hypothesis | p-value | Maximum added non-coinciding events and p-value |
|---|---|---|
| Local events (wired telephony and/or wireless telegraphy) and international firsts in wireless telegraphy triggered Poliomyelitis epidemics (for international firsts : in synchronism with epidemics in the country of origin). | 0.0001 | 7 (0.04) |
| Local events in emission of Hertzian waves (wired telephony and/or wireless telegraphy) triggered Poliomyelitis epidemics. | 0.0007 | 4 (0.046) |
| Wireless telegraphy (including local events and international firsts) triggered Poliomyelitis epidemics. | 0.003 | 3 (0.046) |
| Wireless telegraphy (local events only) triggered Poliomyelitis epidemics. | 0.017 | 1 (0.057) |
| Wired telephony triggered Poliomyelitis epidemics ("knowing" that wireless telegraphy triggered Poliomyelitis epidemics) | 0.022 | 1(0.063);2(0.12) |
| Wired telephony triggered Poliomyelitis epidemics. | 0.035 | 1 (0.097) |

**Table 1: p-values, added non-coinciding events.**

The probabilistic findings are summarized in Table 1. Overall, it is also almost certain that changes in exposure to Hertzian waves (telephony and wireless telegraphy, whether or not limited to local events) triggered Poliomyelitis epidemics in Sweden between 1876 and 1911. Looking into the details, it is almost certain that wireless telegraphy triggered epidemic Poliomyelitis in Sweden between 1876 and 1911, and it is very likely that wired telephony triggered Poliomyelitis epidemics in Sweden between 1876 and 1894.

## 2.3 England, Italy and Germany.

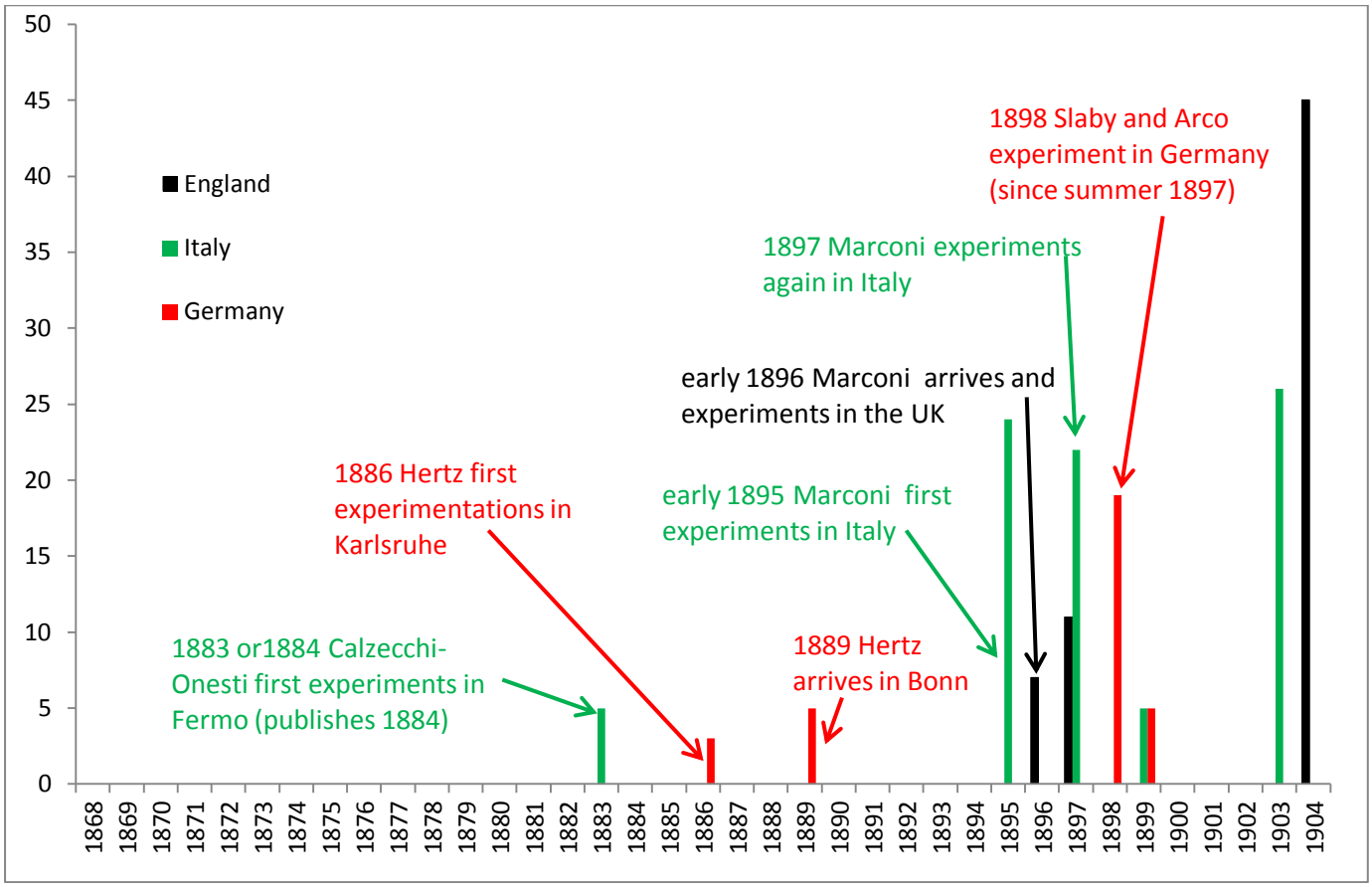



**Figure 3: Poliomyelitis epidemics in several European countries, number of cases, 1868-1904. Based on (Lavinder et al 1918) and Appendix.**

The number of cases in other European countries (Figure 3, Figure 4) is generally a lot lower than in Sweden/Norway, especially in view of the fact that the population in Sweden/Norway is lower. For example the UK cases per population ratio in 1911 is less than $1/20^{th}$ of the Swedish/Norwegian ratio.

There is virtually no detectable reaction to the onset of wired telephony in England, Italy and Germany, likely due to excessive population densities (section 2.5). These countries lead the development of wireless Telegraphy. Early Poliomyelitis cases in these countries followed the first developments of wireless Telegraphy:

- The initial experiments of Calzecchi-Onesti in Italy and Hertz in Germany are correlated with the first recorded epidemic outbursts of Poliomyelitis in these countries. There are uncertainties on the exact timing of these experiments: Calzecchi-Onesti arrived in Fermo in 1883 and published in 1884; it is not known precisely when he started experimenting. Hertz was experimenting on electromagnetism in October 1886, but it is not known precisely when he started.
- The first outburst in the UK and the second outbursts in Italy and Germany are correlated with the early systematic developments (with a view to industrial production): Italian cases rise in 1895 corresponding to Marconi's earliest developments; the English cases rise in 1896 corresponding to Marconi's arrival and first experiments in the UK; the German cases rise in 1897: Slaby visited one of Marconi's test series in the spring of 1897 in the UK, started experiments with Arco in Germany in the summer of 1897, and continued developments in 1897/1898 with substantial support from the German Navy.

Taking into account only the "firsts" (Calzecchi-Onesti 1883; Hertz 1886; Marconi 1895) in Italy and Germany and assuming there is a 2-years period (due to uncertainties on the beginning of the experiments) in which each of these firsts could have taken place, further considering the period from 1868 to 1896 (14 two-years periods), the probability of these "firsts" coinciding with corresponding epidemics is:

For Italy: $P_I=P(Tn=0;T=2;E=2;N=14)=0.011$

For Germany: $P_G$=P(Tn=0,1;T=1;E=1;N=14)=0.07

The overall p-value for these three coincidences is p= $P_I.P_G$ =0.0008

Now looking at the order in which first epidemics took place in England, Italy and Germany in the 1895 to 1898 period:

Italy-UK -Germany

The probability of observing this result by chance (p-value) was:

P=1/6=0.16

Which is not significant per se yet is an additional indication that epidemics were triggered by experimental work on radiotelegraphy.

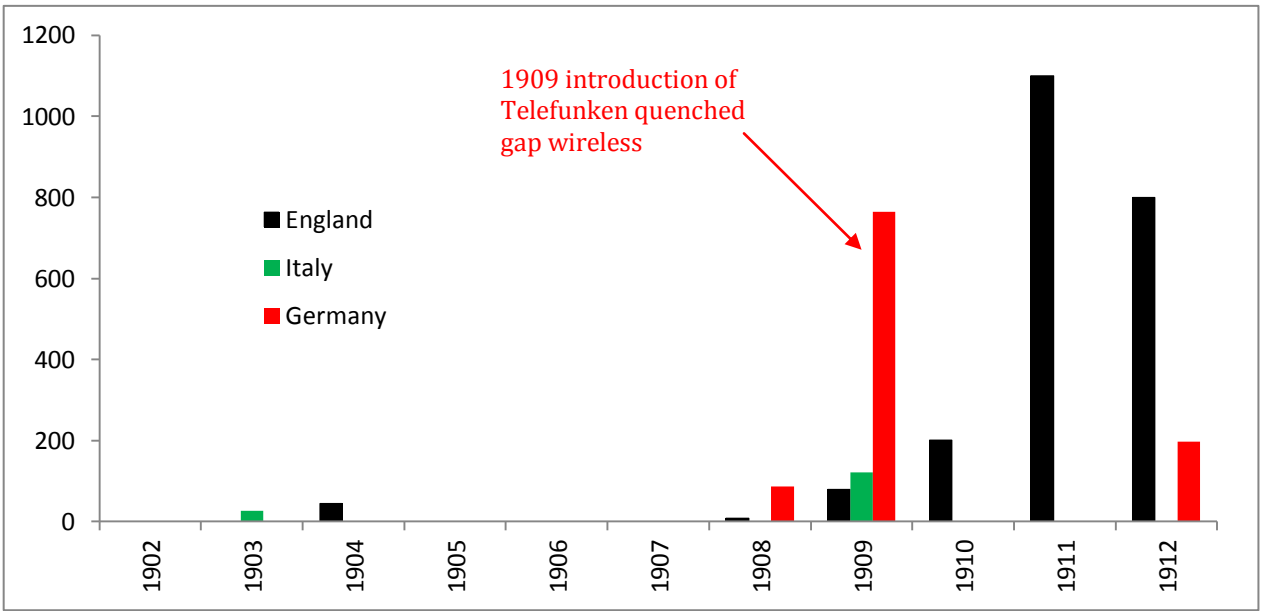



**Figure 4: Poliomyelitis epidemics in several European countries, number of cases, 1902-1911. Based on (Lavinder et al 1918) and Appendix.**

In the 1908-1912 further changes in epidemic Poliomyelitis affected the UK, Germany and Italy; they may be due to the introduction of new technologies in spark gap transmission; but details of such changes are unclear; the situation is generally complex as the number of existing emitters of varying technologies was high and details of new emitters have not been obtained, except for the introduction of quenched gap technology by Telefunken. Figure 4 is provided for completeness.

## 2.4 United States

In the United States statistics prior to 1907 do not easily correlate with any changes in exposure to electromagnetic waves. The telephone network developed in a less orderly manner than in Sweden. Reported epidemics tend to be quite large, for example the reported epidemic in Vermont in 1894 represented 142 cases, which is an indication that smaller outbreaks may have been ignored. However, epidemic Poliomyelitis was regularly present since at least 1893.

The epidemics before 1889 were possibly connected to changes in wired telephony networks, but neither the epidemics nor the extension of the network are sufficiently documented to render a detailed analysis possible. The epidemics of 1899 to 1901 may be correlated to the general availability of radiotelegraphy, with ships equipped for radiotelegraphy navigating to US ports; but it is also uneasy to establish precise correlations. The large transatlantic Marconi stations had no recorded impact, probably because radiotelegraphy was well implanted before the onset of these particular emitters: epidemics were only transient in that period (see section 2.7) and the emissions of the large Marconi stations were qualitatively similar to pre-existing emissions (see section 2.9). This is not unique to the US and the large transatlantic stations in the UK also had no impact on epidemic Poliomyelitis. Generally the pre-1906 period is uneasy to analyze in the US due to the multiplicity of changes and limited availability of the records.

The first voiceband wireless transmissions were correlated to a major epidemic change: from 1907 onward Poliomyelitis became an annual epidemic. The 1916 peak was also correlated with major changes in voiceband transmissions. The 1931 peak is of lesser importance although it coincided with the emergence of commercial Television.

In probabilistic terms, the major change is in 1907. Assuming that it could have taken place anytime between 1893 and 1951, the probability (p-value) of it having taken place by chance exactly in 1907 following the onset of voice communications is P=1/59=0.016, which is significant.

If there was no specific reason for voice communications to have triggered this change, this result would be open to the counter-argument that any of the recorded political or other events in 1907 or the end of 1906 may have triggered the observed increase in epidemic Poliomyelitis. However, in view of the fact that voice communications are expected to have a stronger effect than all prior systems (see section 2.8) and of the general fact that electromagnetic waves interact with the immune system (Lauer 2014b) the 1907 change in the epidemiology of Poliomyelitis can be reliably associated with the onset of voice communications and the above-mentioned probability is to be considered a reliable figure.

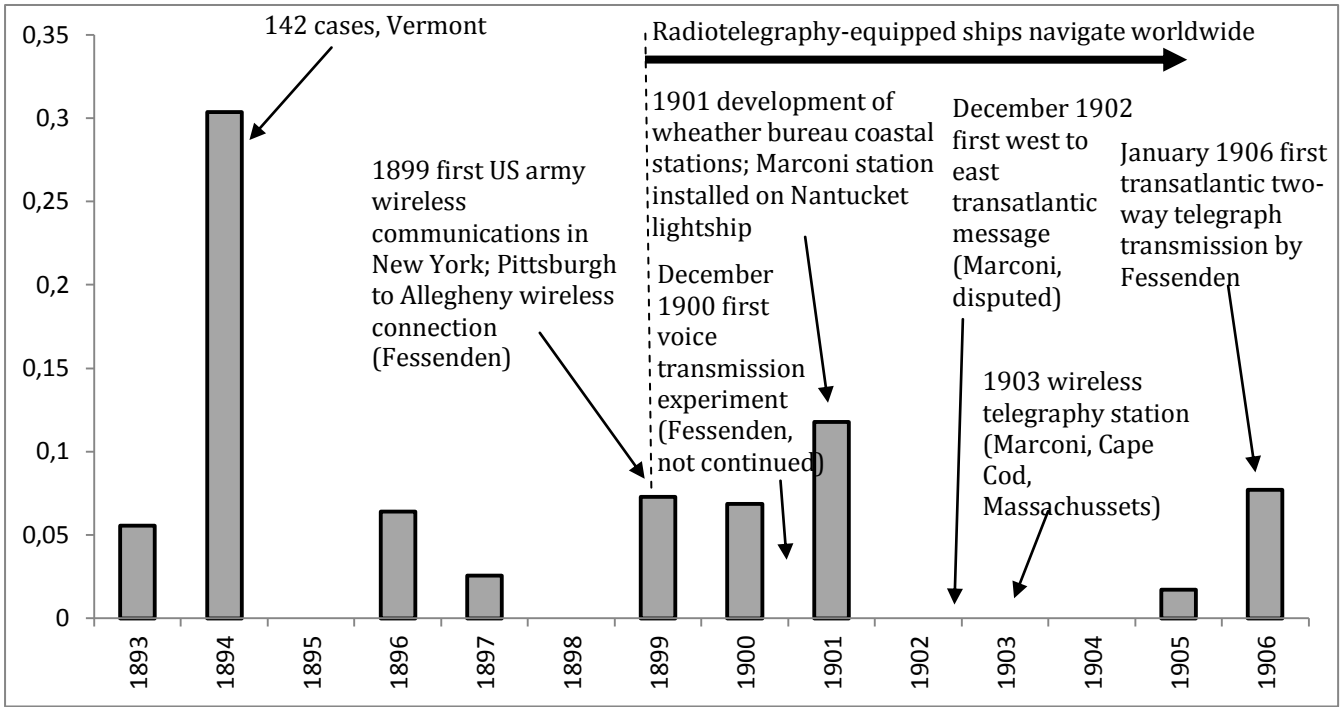



**Figure 5: Annual poliomyelitis case rate in U.S. States reporting both cases and deaths. based on Lavinder et al 1918 and the population of 1910 in Sherfling and Sherman 1953.**

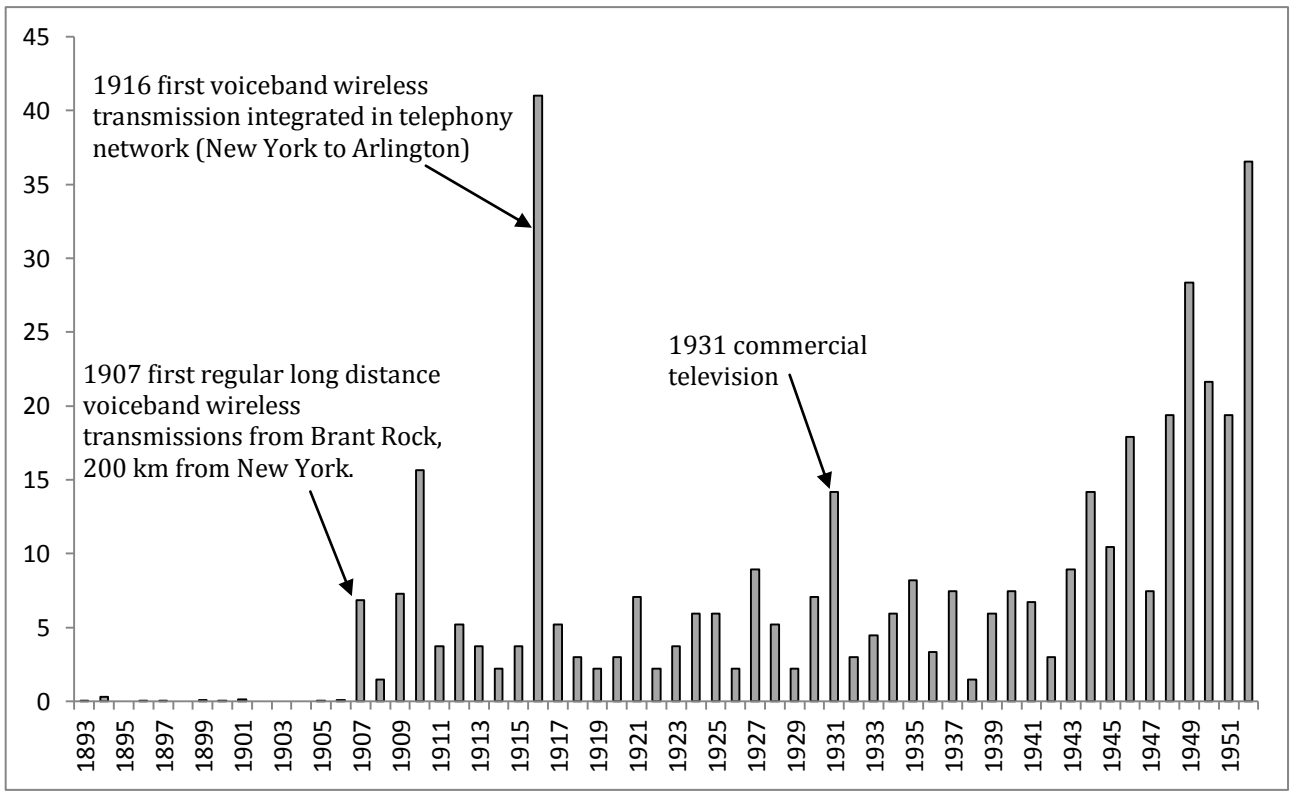



**Figure 6: Annual poliomyelitis case rate in U.S. States reporting both cases and deaths. From 1910 onwards: based on Serfling and Sherman 1953. Before 1910: based on Lavinder et al 1918 and the population of 1910 in Sherfling and Sherman 1953.**

## 2.5 The population density parameter.

In the early times of epidemic Poliomyelitis, the disease was known to occur more often in sparsely populated areas than in densely populated areas. For example Wickmann, discussing the epidemic of 1905, mentions that the disease frequently occurred in small communities in rural areas and essentially spared large cities. This may have changed at a later point but it was the case at least until 1905 in Sweden/Norway.

Sweden/Norway is much less densely populated than any other European country discussed herein and is even significantly less populated than most states in the Eastern United States where changes in wired telephony and wireless telegraphy and telephony took place. Therefore, following the general rule that epidemic Poliomyelitis occurs more easily in less populated areas, it is logical that Sweden/Norway was more strongly hit than other countries.

In particular, the first significant recorded epidemic in Sweden in 1881 did not take place in Stockholm where the wired telephony networks were installed, but in Umea, a small city of about 3000 inhabitants, in the province of Vasterbotten which is itself little populated. The most significant of the early epidemics in the United States took place in Vermont, which is possibly the Eastern US state having the lowest population density.

The dependency on population density is also confirmed by findings in very isolated populations. Among Eskimoes in the Canadian Arctic in 1948-49, about 60% of the population had paralytic disease (Paul 1955). In Nauru (a small pacific island) in 1910 the epidemic affected a third of the population, including two Europeans but no east Asians (Cushman 2013). One of the first outbreaks of epidemic Poliomyelitis ever recorded occurred on the island of St Helena.

It has been proposed that the dependency of epidemic Poliomyelitis on population density is due to populations in less populated areas being less exposed to the virus, thus exposed later, thus more likely to develop grave forms of the disease. This explanation may not be fully satisfactory, but a full discussion of the issue goes beyond the scope of this paper. I will thus simply admit, in agreement with general knowledge and with facts, that epidemic Poliomyelitis has a strong dependency on population density and that this dependency explains why Sweden was more hardly hit than other countries and why the US was more hardly hit than Germany, Italy or the UK.

| | thousand inhabitants | thousands km2 | habitants/km2 |
|---|---|---|---|
| UK | 34 000 | 244 | 139,6 |
| Germany | 56 367 | 541 | 104,2 |
| Italy | 32 000 | 310 | 103,2 |
| Sweden | 5 136 | 450 | 11,4 |
| Vasterbotten | 111 | 55 | 2,0 |
| Norway | 2 240 | 385 | 5,8 |
| Massachussets | 2 805 | 27 | 102,6 |
| New York State | 7 269 | 141 | 51,4 |
| Pennsylvania | 6 302 | 119 | 52,8 |
| Vermont | 344 | 25 | 13,8 |
| United States | 76 212 | 9 857 | 7,7 |

**Table 2: Population densities in several geographical areas around 1900.**

The most obvious application of the dependency of Poliomyelitis on population density is the Swedish epidemic of 1905 which was triggered by the installation of local radiotelegraphy emitters and reached a level which at the time was not comparable to any other outbreak.

## 2.6 Distance between source and epidemic

The distance between the source and the epidemic is very variable (Table 3).

Shorter distances are seen when the change in electromagnetic emissions is not a “first”. For example the first outbreak connected to wire telephony in Sweden had a source-to-outbreak distance of 500 km but the second one had both its source and outbreak in Stockholm. The first Marconi emissions in Bologna were probably the cause of the 1895 Stockholm outbreak 1700 km from the source (and of the Montespertoli and Genova outbreaks 100 and 400 km from the source) but later Marconi emissions had more local effects.

In a “first” electromagnetic emission, the emission takes place above essentially a strictly thermal background. The source to background spectral power ratio is very high, which justifies a far-reaching effect. Assuming that there is a threshold value of power (in W/m2) above which emissions can cause epidemic Polimomyelitis outbreaks, a power which

is above threshold does not necessarily yield a much higher probability of an epidemic, so that the finding that epidemics occur far from the source is not generally unreasonable.

An example of a "second" emission is as follows: when the Slaby and Arco experiments of 1898 took place in Germany, comparable emissions already existed which originated from England. Locally in Germany the Slaby and Arco experiments increased the spectral power and had an effect on epidemic Poliomyelitis, triggering (or at least coinciding with) two outbreaks in Germany and one in Austria. But far from Germany the English and German emissions were received with comparable power, so that the Slaby and Arco experiments had no significant effect on epidemic Poliomyelitis and there was no epidemic in that year outside Germany and Austria.

Thus, generally, "second" emissions qualitatively similar to pre-existing emissions emanating from another place have a more local effect than "first" emissions taking place without a pre-existing background of similar electromagnetic waves emissions. Note that the emissions of Calzecchi-Onesti (1883), Hertz (1886) and Marconi (1895) are in each case considered as firsts, because the first two were temporary so that in each case the emissions took place against a background of zero emissions in wireless telegraphy.

| | | source | outbreak | Distance |
|---|---|---|---|---|
| Wire telephone | 1881 Stockholm | Stockholm | Umea | 500 km |
| | 1887 Stockholm | Stockholm | Stockholm | 0 km |
| Wireless telegraph | 1883 Calzecchi-Onesti | Fermo | Arenzano | 400 km |
| | 1895 Marconi | Bologna | Montespertoli | 100 km |
| | | | Genova (Ravecca) | 200 km |
| | | | Stockholm | 1700 km |

**Table 3: distance between source of electromagnetic emission and recorded epidemic in several early outbreaks.**

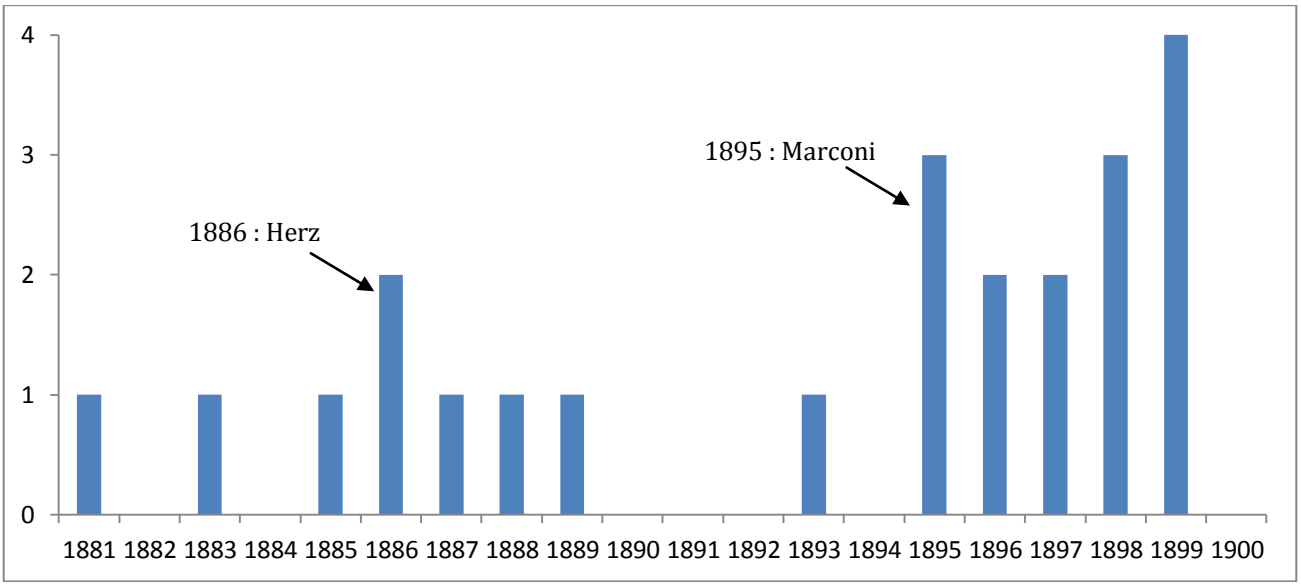



**Figure 7: number of reported epidemic Poliomyelitis outbreaks in Europe between 1881 and 1900, after (Lavinder et al 1918) with one date corrected as per (Cordier et al 1887). Note that there was no outbreak between the 1868 and the 1881 outbreaks.**

Examination of epidemics that coincided with the first experiments of Marconi (Table 3) shows that Marconi's first experiments may have triggered up to three epidemics at different distances. Indeed, the number of epidemics in 1895 in Europe was unusual (Figure 7). It is therefore likely that the abnormal number of epidemics in Europe in 1895 was not due to chance and was rather due to the triggering of these epidemics by the Marconi experiments. In particular, causation of the Swedish/Norwegian epidemic of 1895 by the Marconi experiments is not unreasonable, because although Sweden/Norway is far from the places of the experiments, it is also more likely to host epidemics due to its lower population density (see section 2.5).

## 2.7 Transient and long-lasting effects.

The onset of wired telephony in Stockholm in 1881 is an example of a long-lasting change which had only transient effects. Likewise, early changes in epidemic Polimyelitis were generally transient. This is not surprising per se: stronger

effects are generally expected in the transition period from low to high exposure for auto-immune diseases, cancer (Lauer 2014b) and diseases caused by pathogens which present near-self epitopes (Lauer 2015b).

However the 1907 change in the United States was permanent. This is an important qualitative difference with previous changes, possibly reflecting the passing of a threshold causing a major functional change in the immune reaction against Poliovirus. A permanent effect is not of itself surprising as infectious diseases which are not self-mimicking are expected to be permanently effected by exposure (Lauer 2014b). This permanent effect was likely due to the onset of wireless voice transmission. However, properly observing the permanent character of the 1907 effect also required a large enough territory as epidemics after 1907 varied in their geographical epicenters and extent.

The simultaneous existence of a permanent effect (U.S. from 1907 onwards) and of transient effects (all other observed epidemics discussed herein) is an indication that the virus yielded both near-self epitopes (non-recognition of which caused transient effects) and far-from-the-self epitopes (non-recognition of which caused a permanent effect). Although this sort of combination was not discussed in previous publications, it could be expected in view of the fact that, as discussed in (Lauer 2015b), it is unlikely that a virus can have solely near-self epitopes both after restriction by MHC class I and after restriction by MHC class II.

## 2.8 Difference between emissions.

Different emissions vary in bandwidth and power (Figure 8). Higher bandwidth and higher power generally imply more immune system-mediated effects on diseases (Lauer 2014b), including Poliomyelitis. Indeed, this is what is observed. Wireless telegraphy (resp. voice transmission) caused more Polio outbreaks than Electrical Telegraphy (resp wired Telephony), wired Telephony (resp. wireless voice transmission) caused more Polio outbreaks than electrical (resp. wireless) telegraphy.

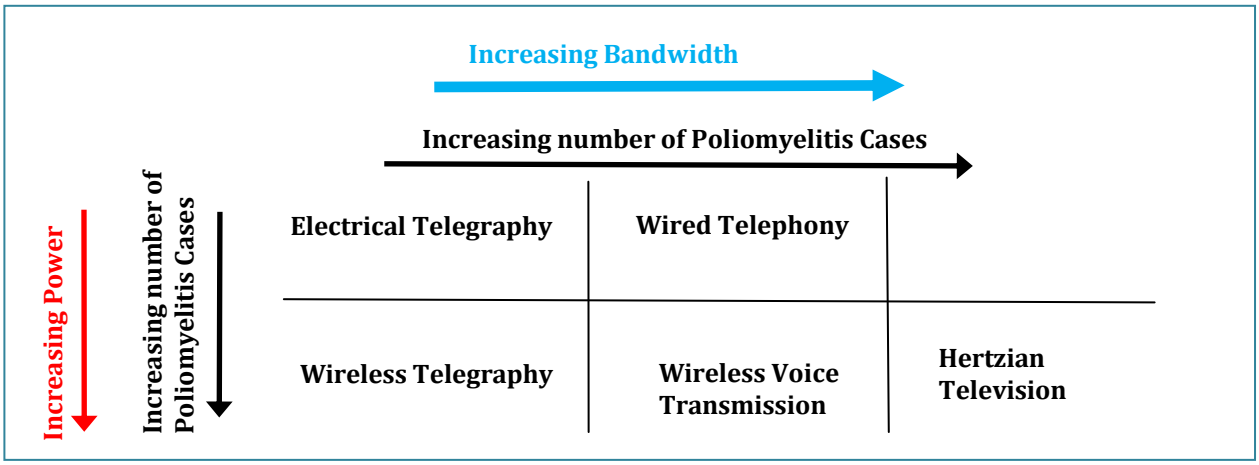



**Figure 8: Comparison of emission types.**

## 2.9 Variations within Spark Gap systems.

Wireless telegraphy uses spark gap technology. A slightly idealized wireless telegraphy signal is a repetitive periodic signal lasting the duration of a "bit" (push on the telegraph key). Its frequency representation is a set of low bandwidth components at discrete frequencies ("frequency comb"). The bandwidth (sum of the individual bandwidths of the discrete components) is low, despite the fact that the signal globally extends over a wide frequency range. Due to this low overall bandwidth, this signal is expected to have a weaker effect on the immune system than voiceband transmissions which have a larger bandwidth [the effect of "frequency combs" as compared to wideband signals was also discussed in (Lauer 2015a) in the context of experimental results on the animal]. However, real spark gap transmitters use a variety of technologies which depart from this ideal model. For example, a transmitter using an asynchronous rotary spark gap does not produce a periodic signal. Even transmitters which theoretically generate periodic signals may be affected by time/frequency instabilities, which can increase considerably the summed bandwidth of the system and thus its impact on the immune system. So the properties of a wireless telegraphy system are expected to heavily depend on the specific technology used. Technological differences could explain in part the strong increase of Poliomyelitis cases which took place in Norway/Sweden in 1911 following the onset of a wireless telegraphy system whilst there was already a working emitter (possibly of a different technology) since 1905.

## 2.10 Electrical Telegraphy

Poliovirus is an ancient virus but historically it affected only very young children and it was endemic rather than epidemic (Heine 1840, 1860, Laborde 1864). The first recorded cases of epidemic poliomyelitis prior to the onset of telephone networks are:

an outbreak in the island of St. Helena, not dated, but is likely to have occurred sometime between 1824 and 1836 in view of the dates of some other cases in the same publication (Bell 1836).

A small outbreak in Worksop, UK, with 4 cases about august 14, 1835, only children about 2 years old (Badham 1836).

an outbreak of 8 to 10 cases in Louisiana in 1841 (Colmer 1843).

an outbreak of 14 cases in Odalen, Norway, in 1868 (Lavinder et al 1918).

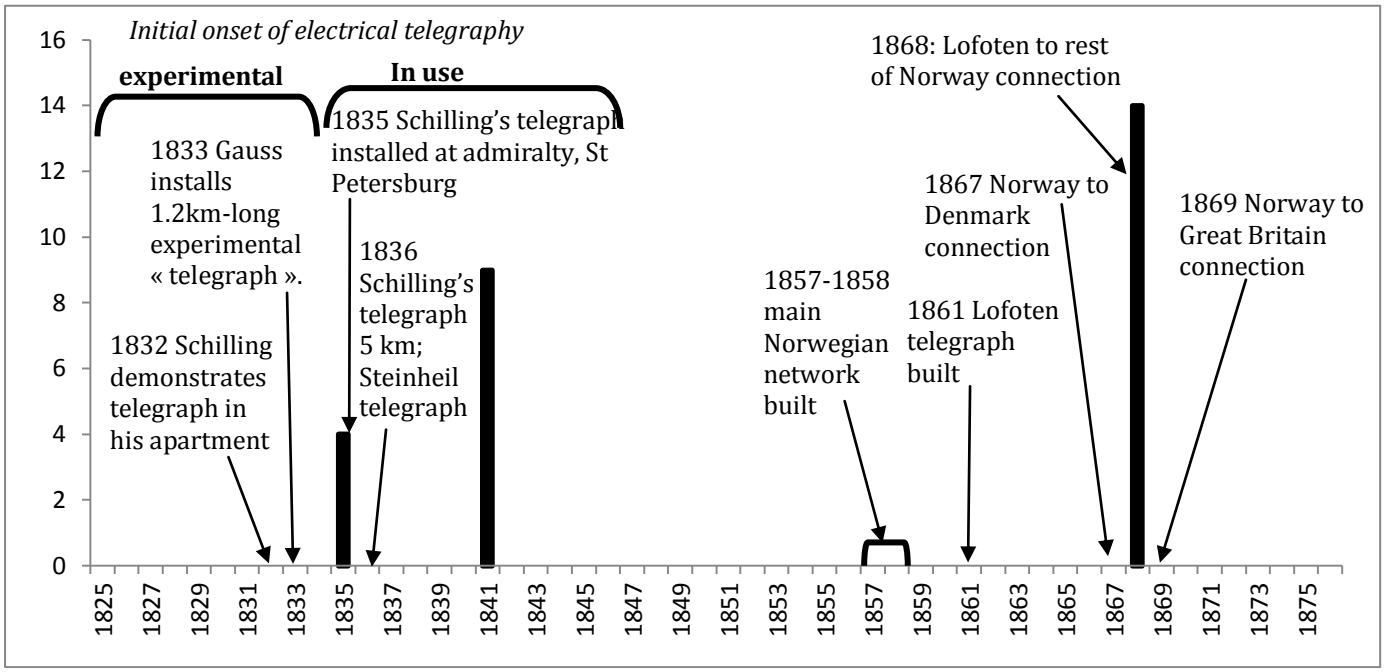



**Figure 9: Reported dated Poliomyelitis epidemic outbreaks until 1868, and changes in wired telegraphy. For changes in electromagnetic wave emissions see Appendix.**

One interesting question is whether these outbreaks were caused by electrical telegraphy or were rare but "natural" outbreaks. All dated outbreaks are dated after the invention of Electrical Telegraphy, but this is not per se a proof as these outbreaks were rare and medical coverage of the potentially affected population may have been insufficient in earlier periods to detect outbreaks.

The first telegraph capable of practical use was Schilling's telegraph which was demonstrated to the Tsar in 1832 between two rooms of Schilling's appartment. The Gauss and Weber system was essentially experimental and was installed in 1833. As compared to Schilling's telegraph it was a "a set of bulky labware of a little practical use" (IEEE Milestones), had a very low data rate (and thus a low bandwidth) and was rarely used.

The first telegraph to be installed for practical use was Shilling's telegraph which was installed in 1835 in an underground line of the admiralty's building in St Petersburg. It was a relatively modern telegraph designed for practical communications. Installation in the admiralty's building implies that it may have been regularly used.

In 1836, Steinheil had a working telegraph and Schilling had a 5 km telegraph for the Admiralty in St Petersburgh.

The effects of electromagnetic waves in the early period of Poliomyelitis history were generally transient (see section 2.7) which implies that an epidemic Poliomyelitis outbreak is more likely to have been triggered by the "first" use rather than by the later progressive developments. Schilling's installation of 1835 was the first practical use of radiotelegraphy (also implying a higher bandwidth than the previous experimental systems) and was thus particularly likely to trigger an outbreak of epidemic Poliomyelitis. Later steps were comparatively incremental/progressive.

On this basis, the probability of having one out of three dated outbreaks in the roughly 1825 to 1875 period (50 years) coinciding with this event (which is in a 1 year window) is P(Tn=0;T=1;E=3;N=50)=0.06. This is near to the significance threshold.

We may ignore

(a) the difference between the installation of Schilling in the Admiralty which provided an effective distance between the transmitter and receiver ends of the installation, triggering a reasonable unintentional Hertzian emission, and the previous Schilling installations in his apartment, where the transmitter to receiver distance was extremely low, and
(b) the difference between the Gauss and Weber apparatus and the Shilling apparatus.

In this case, the window to be considered is 1832-1836 (a 5-years window) and the probability of having one out of three dated outbreaks in the roughly 1825 to 1875 period (50 years or 10 five-year periods) coinciding with this event

(which is in a 5 year window) is P(Tn=0;T=1;E=3;N=10)=0.3. This is far from the significance threshold, so that the significance of the 1835 outbreak does depend on the correctness of the analysis concerning the Schilling telegraph.

The outbreak of 1841 took place within the period of early developments of electrical telegraphs but cannot be connected to any particular triggering event, perhaps due to lack of information.

The last dated outbreak of the pre-wired telephony era was in Norway in 1868. The local events in this period were the connection of the main Norwegian network to Denmark (1867), the Lofoten islands (1868; there was a pre-existing electrical telegraph in the Lofoten), and England (1869). These connections were possible triggering events because they may generated brutal increases in traffic due to the connection of pre-existing active networks: unlike the case of telephony, telegraph was mostly used for long-distance communications. These 3 connections connections are the events most likely to have generated a Poliomyelitis epidemic in Norway between 1862 (when both the main Norwegian network and the Lofoten telegraph had been completed) and 1875 (before the invention of telephone) corresponding to 14 years. On this basis the calculated p-value is P(Tn=2;T=3;E=1;N=14)=0.2, a p-value which is quite far from significance.

However, the connections to Denmark and England were probably used mostly for "big business", which is valuable but generates little traffic. The connection to the Lofoten is likely to have carried messages from fishermen to their families, and messages between salesmen and fishermen related to the fish trade, all of which is of lesser value but represents more traffic. Further, the connection to the Lofoten added a substantial amount of aerial lines, whilst the connections to Denmark and England were mostly submarine. Therefore the connection to the Lofoten is more likely to have generated a significant increase of unintentional Hertzian emissions, as compared to the England and Denmark connections. If we take this into account, the probability of a single polio epidemic of the previously mentioned 14-years period occurring by chance in 1868 is P(Tn=0;T=1;E=1;N=14)=0.07, which is near significant.

Analysis of the 1835 outbreak and of the 1868 outbreak yield p=0.06 and p=0.07 respectively, which in each case is near the significance threshold. However these p-values are highly dependent on the correctness of the analysis of the importance of the events of Schilling's 1835 telegraph and of the Lofoten to rest of Norway connection, as compared to other events. If these analyses are flawed, the respective p-values become non-significant. Since there is arguably some degree of subjectivity in these analyses, the observations of the pre-wired telephony period are at best an indication that wired telegraphy may have triggered Poliomyelitis epidemics.

## 2.11 Analysis

Analysis of coincidences was used to evaluate the hypothesis that Poliomyelitis epidemics were caused by changes in exposure to electromagnetic waves. The leading case is Sweden/Norway (section 2.2), which was the country most strongly hit worldwide in the early times of epidemic Poliomyelitis. Analysis of the sole case of Sweden/Norway yields to the conclusion that Poliomyelitis epidemics were almost certainly triggered by changes in exposure to Hertzian waves. This result is confirmed by observations in European countries that led the development of wireless telegraphy (section 2.3), and by the observations in the United States (section 2.4). Combining these results together, the overall probability of changes in epidemic Poliomyelitis being triggered by changes in emissions of electromagnetic waves appears extremely high, beyond reasonable doubt.

Observations which at first glance may seem surprising can be explained using existing knowledge and commonsense, including the higher incidence of early epidemic Poliomyeltis in Sweden (section 2.5), the large distance between the source of electromagnetic emissions and the outbreaks of epidemic Poliomyelitis (section 2.6), the existence of both transient and permanent effects (section 2.7) and their triggering by qualitatively different emissions (section 2.8), and the different effects of technologically different spark gap systems (section 2.9).

Looking into the details:

(a) Wireless telegraphy: the analysis in Sweden/Norway (section 2.2), and in European countries that led the development of wireless telegraphy (section 2.3) independently yield the same conclusion that wireless telegraphy almost certainly triggered epidemic Poliomyelitis.
(b) Wireless voice transmission: The analysis in the United States shows that wireless voice transmissions triggered a much increased incidence of epidemic Poliomyelitis as compared to wireless telegraphy only. The significance level of this observation is less than for wireless telegraphy, but since it was expected that voice transmissions should have a stronger effect than wireless telegraphy (section 2.8), this observation is viewed as reliable.
(c) Telephony: the analysis in Sweden/Norway (section 2.4) shows that it is very likely that wired telephony triggered epidemic Poliomyelitis.
(d) Electric Telegraph: the analysis in section 2.9 yields some indications that Electrical telegraphy may have triggered Poliomyelitis epidemics – but no reliable proof.

In short, the observations with regards to epidemic Poliomyelitis appear to be consistent with existing knowledge and their triggering by changes in emissions of electromagnetic waves appears to be probabilistically confirmed.

# 3 Evolution of group C enteroviruses.

## 3.1 Asymmetrical recombination of Poliovirus and C-cluster Coksackie A viruses.

In vitro recombinants generated by replacing one of the P2, P2(2C), or P3 regions (but not the P1 region corresponding to the capsid) of a standard Poliovirus PV1 by corresponding regions of a C-cluster Cocksackie A virus CVA20 were viable. In vitro recombinants generated by replacing the P1 region (but not the P2, P2(2C), or P3 regions) of a C-cluster Cocksackie A virus CVA20 by corresponding regions of a standard Poliovirus PV1 were viable (Jiang et al 2007). This shows that the non-structural part of CVA20 can recombine with the capsid of PV1 but the non-structural part of PV1 cannot recombine with the capsid of CVA1. Essentially the same relation was obtained between PV3 and CVA20, or between PV1 and CVA21, as between PV1 and CVA20.

Following vaccination with Oral Polio Vaccine (OPV) comprising a mix of the three Sabin strains S1, S2, S3 serologically equivalent to PV1, PV2 and PV3 respectively, patients excreted the three types of attenuated Poliovirus, and recombinants which had nucleotides from other strains in their non-structural region. Serotype S3 recombined mostly with the non-structural part of serotype S2 (more than 50% S3/S2 recombinants amongst S3 serotype). Serotype S2 recombined mostly with the non-structural part of serotype S1 (more than 40% S2/S1 recombinants amongst S2 serotypes) . Serotype S1 did not usually recombine (less than 2% recombinants amongst S1 serotype) (Cuervo et al 2001). Assuming the Sabin strains essentially behave the same way as corresponding wild Polioviruses, this shows that the PV3 capsid recombines frequently with the PV2 non-structural part but the PV2 capsid recombines rarely with the non-structural part of PV3; and that the PV2 capsid recombines frequently with the PV1 non-structural part but the PV1 capsid recombines rarely with the non-structural part of PV2.

These asymmetrical recombination possibilities are summarized in Figure 10.

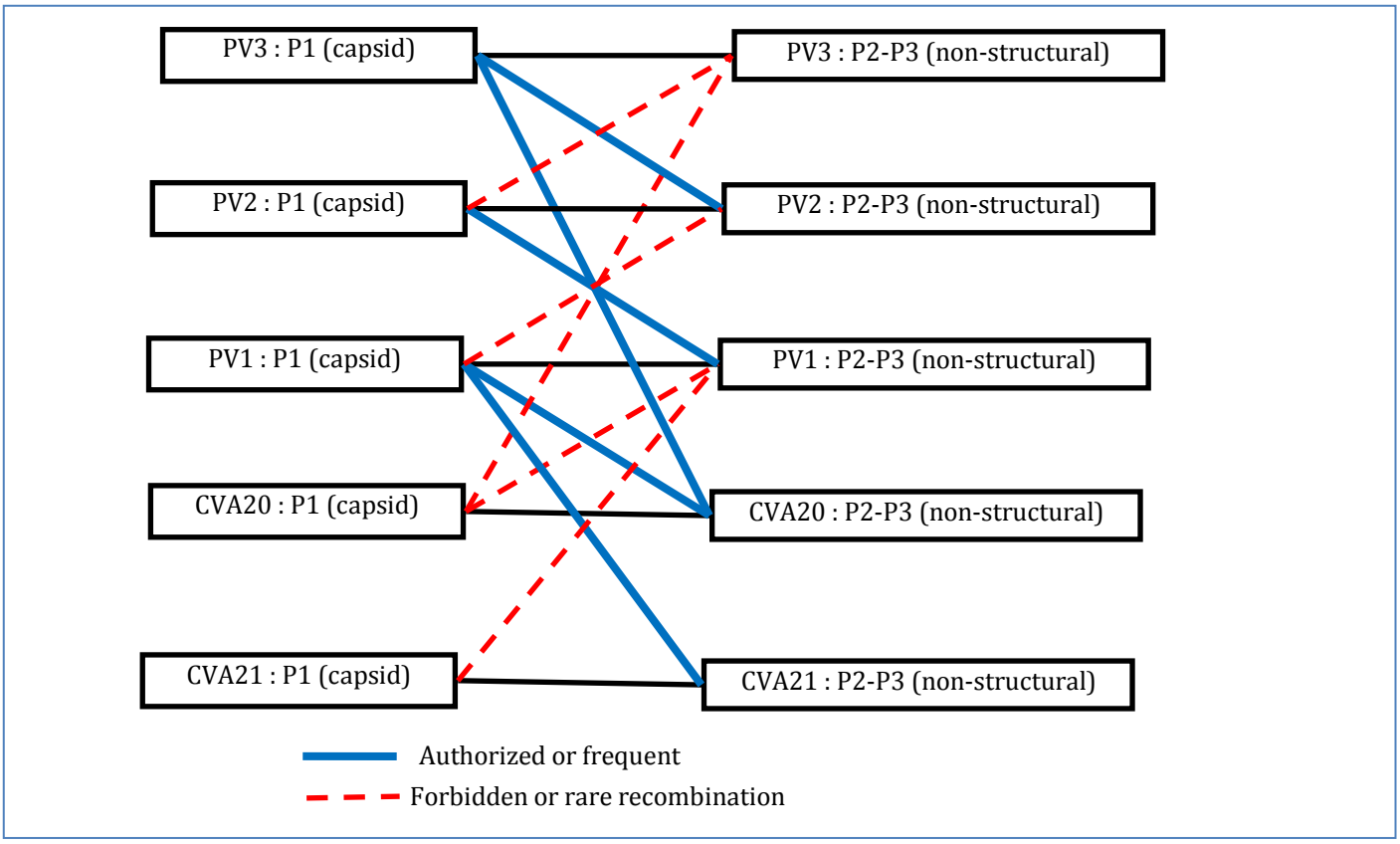



**Figure 10: asymmetrical recombination of Poliovirus and other Cluster C enteroviruses.**

## 3.2 Viral Persistence.

Post-polio patients have mutated Poliovirus genomic sequences in their Cerebro-Spinal Fluid (CSF) whilst polio survivors without post-polio syndrome do not (Leon-Monzon and Dalakas 1994, Leparc-Goffart et al 1996). This is objective evidence that Post-polio syndrome is due to the persistence of a mutant form of Poliovirus in polio survivors. Such genomic sequences were not detected in all post-polio patients, suggesting that the virus persists in minute amounts (Leparc-Goffart et al 1996). High titers of IgG and IgM antibodies to Poliovirus were detected in the serum of post-polio patients but not in healthy blood donors. But high titers of IgG antibody to poliovirus were detected in some patients with Amyotrophic Lateral Sclerosis (ALS). High titers of IgM antibodies to poliovirus were not detected in patients with other neurodegenerative diseases. The IgM antibodies to Poliovirus are dominantly produced by B cells inside the CSF as

corresponding oligoclonal bands were found in post-polio patients but not in controls (Sharief et al 1991). It thus appears that post-polio is associated to persistence of mutant versions of Poliovirus, accompanied with a humoral immune reaction comprising the production of IgM antibodies in the CSF.

Outside the Central Nervous System (CNS), Poliovirus spreads by cell lysis. However, Poliovirus dominantly spreads through the nervous system via trans-synaptic transmission rather than cell lysis (Daley et al 2005). The normal immune reaction in the CNS involves synaptic stripping of synapses by microglia (Kettenmann 2013). This yields to containment of the infection within affected neurons rather than to elimination of the infection. This explains why poliovirus can persist in the CNS.

## 3.3 Age of patients in Poliomyelitis and post-polio syndrome.

Prior to the onset of artificial electromagnetic waves, poliomyelitis was endemic; thereafter it became epidemic. Endemic Poliomyelitis affected only very young children, 6 months to 3 years old (Heine 1840, 1860), sometimes up to 4 years (Laborde 1864). In epidemic poliomyelitis the age profile was variable but generally included patients aged more than 15 years (Wickman 1913).

Age is a major determinant in post-polio syndrome (Ivanyi et al 1999) and it is likely that the syndrome occurs after thymus involution (after the age of about 35-45 years) when thymic output slows down and the number of recent thymic emigrants diminishes. However, at least until 1875 (which we may consider to be in essence prior to the onset of artificial electromagnetic waves, although wired telegraphy was already present) post-polio syndromes affected younger men:

- Gautier had paralytic poliomyelitis when aged 6 months, recovered partially when aged 6 years, and had a post-polio syndrome starting at the age of 16 or 17 (Carrieu 1875).
- Basset has paralytic poliomyelitis including convulsions between 6 months and 2 years of age. His health status was then stationary until age 15 years. Aged 15 years he developed a post-polio syndrome (Carrieu 1875).
- X. had paralytic poliomyelitis aged 6 months. He partially recovered when aged 7 years. He had a post-polio syndrome starting at age 17 (Raymond 1875).
- D .had a childhood paralysis followed by complete recovery. He developed a post polio syndrome when aged 24 following a period in which he suffered much from cold. He died of his post-polio syndrome (unable to breathe) (Cornil and Lépine 1875).

Overall, these early cases show onset of post-polio syndrome at the age of 15-17 years in patients having had early paralytic poliomyelitis with no recovery or partial recovery. D. had post-polio syndrome later, aged 24, but had a mild paralytic poliomyelitis with full recovery.

## 3.4 Duration of immunity to Poliovirus attenuated strains

Immunity to Poliovirus attenuated strains is longest for Sabin 1 corresponding to wild type PV1 (up to 16 years) and shortest for Sabin 3 strain corresponding to wild-type PV3 (in the order of 2 years) (Trivello et al 1988). It is likely that immunity induced by the corresponding wild-type strains is similar, i.e. shortest for PV3 and longest for PV1.

## 3.5 Fast recombination.

Following infection with OPV comprising the three attenuated strains S1, S2, S3 derived from PV1, PV2, PV3, the first excreted S2 recombinants and non-recombinants appear simultaneously on day 2. The number of excreted viruses is maximal on day 14 and the number of S3 recombinants on day 14 is 80% of the number of non-recombinants (Cuervo et al 2001). This shows that recombination is a fast process which can take place in a single host during the short period of the initial infection.

## 3.6 Analysis.

Figure 10 shows that there is an asymmetrical relation between group C enteroviruses. Such systematic asymetrical relations are unlikely to appear by chance. There are two reasonable possibilities: either (i) a virus B which non-structural part can recombine with the capsid of a virus A is its descendent, or(ii) it is its ascendant. If hypothesis (i) is correct, PV3 is the ancestor of all viruses of Figure 10. If hypothesis (ii) is correct, all Polioviruses are descendents from CVA20 and/or CVA21.

At onset of acute Poliomyelitis, an immune response develops, which is protective against Poliovirus. Post-poliomyelitis is associated with viral persistence, i.e. the presence of Poliovirus or a Poliovirus mutant. In 1875 post-poliomyelitis could develop at the age of 15, a relatively short time after the acute symptom, and it is thus likely that it was due to the original Poliovirus with relatively few mutations. The original immune response, developed during the acute phase, was probably efficient against the Poliovirus mutant. It is thus unlikely that post-polio syndrome could appear whilst the original immune response was still conferring immunity. This excludes PV1 which confers an immunity of up to 16 years, and PV2 although to a lesser extent. Thus in 1875, PV3 existed but there is no indication that PV1 or PV2 existed.

Thus, it is likely that PV3 is the ancestor of PV2 and PV1 rather than the opposite, and therefore it is likely that hypothesis (i) is correct. Thus, the descendent "B" of a group C enterovirus "A" would tend to maintain capsid compatibility with its ancestor.

Is this explainable ? Polioviruses recombine quickly within an infected host. They are known to cause an essentially humoral immune response targeted to the capsid proteins. Recombination of a poliovirus with capsid elements from another virus yields a mutant which may escape an ongoing immune response. Thus a descendent "B", which originally developed amongst dominant ancestral "A" infections, was favored if it maintained capsid compatibility with "A". This explains why the descendent "B" of a group C enterovirus "A" would tend to maintain capsid compatibility with its ancestor rather than the opposite. This reasoning confirms that hypothesis (i) is valid, rather than hypothesis (ii) which would not confer any particular benefit to the descendent.

Since in 1875 there is a strong indication that PV3 was present and no indication that PV1 or PV2 was present, it is likely that PV1 and PV2 appeared after 1875.

In short, there is good support for the hypothesis that PV3 is the ancestor of PV2, PV1, CA20 and CA21 and that PV2, PV1, CA20 and CA21 emerged after 1875.

## 4 Conclusions.

There is strong support for the following hypotheses:

A) The emergence of epidemic Poliomyelitis was triggered by the onset of artificial electromagnetic waves, including wireless telegraph and wireless voice transmission, and probably including wired telephony.
B) PV2, PV1, CA20 and CA21 emerged after 1875 from a PV3 ancestor.

These hypotheses comfort each other: the mechanism of PV3 infection was somehow disturbed by the onset of artificial electromagnetic waves, yielding the emergence of epidemic Poliomyelitis; and following that disturbance new virus variants evolved from PV3, which were better adapted to the new environmental conditions (i.e. the presence of artificial electromagnetic waves).

The herein presented findings confirm the existence of a causal relation between onset of artificial electromagnetic waves and Poliomyelitis, which was originally suspected in view of the fact that onset of electromagnetic waves is the only common explanation reasonably likely to have caused the major epidemiological changes observed in the 20th century.

Not every viral disease reacted in the manner that Poliomyelitis did. Indeed, Poliomyelitis may be the only disease which reactions to exposure to electromagnetic waves can be traced back as far as wireless telegraphy and probably wired telephony. In contrast to AIDS (Lauer 2015b), sensitivity of Poliomyelitis to electromagnetic waves pre-existed artificial electromagnetic waves, independent of the later evolution of Poliovirus. However, the diversification of Poliovirus following exposure to electromagnetic waves parallels the evolution of HTLV towards HIV.

Guglielmo Marconi, merely by experimenting wireless telegraphy in Bologna, without personally suffering any harm and without causing a Poliomyelitis epidemic in Bologna itself, caused a Poliomyelitis epidemic in Montespertoli at a distance of 100 km, another in Genova at a distance of 200 km, and probably a worst one in Stockholm at a distance of 1700 km: this was not really expected, and it looks somewhat unreasonable as first glance. Yet, this is one of the conclusions which are strongly supported by facts and scientifically explainable. Generally, the high sensitivity of Poliomyelitis to electromagnetic waves, and the manner and extent in which emissions of electromagnetic waves influenced Poliomyelitis epidemics, are explainable but are somewhat at odds with "commonsense" intuition and expectations. However facts are a more reliable guide than preconceived ideas in understanding observations, and in the present case facts yield to the conclusion that epidemic Poliomyelitis was caused by emissions of artificial electromagnetic waves (in combination with Poliovirus).

The present findings are of interest with regards to the problem of Electro-Hyper-Sensitivity: the claim that some persons would be affected by very low power exposure, possibly including wired telephony, appears more "believable" when viewed against the finding that epidemic Poliomyelitis reacted to wireless telegraphy and most likely wired telephony, under extremely low exposure power. If "Electro-sensitive" means a person who is diseased due to exposure to electromagnetic waves and with some degree of synchronism with exposure, then victims of epidemic Poliomyelitis were probably the first electro-sensitives in history, for even if infected with Poliovirus, they would not have been paralyzed in the absence of artificial electromagnetic waves.

Whilst the present paper clarifies the underlying causes of the emergence of epidemic Poliomyelitis and of the diversification of Poliovirus, it does not propose a complete understanding of Poliomyelitis. Thus, work remains to be done in order to improve our understanding of the interaction between poliovirus, electromagnetic waves and the human being.

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