Citation: van Steenis, Pieter Bastiaan. (1919). “Some epidemiological remarks about the flu in the Magelang Division, 1918,” *Medical Journal for the Netherlands Indies* LIX:901-20 (Siddharth Chandra, Trans.). Mimeo, Michigan State University. Translated with assistance from Google Translate.

Original document: van Steenis, Pieter Bastiaan. (1919). “Enkele epidemiologische opmerkingen aangaande de griep in de afdeeling Magelang 1918,” *Geneeskundig Tijdschrift voor Nederlandsch-Indië* LIX:901-20.

# MEDICAL JOURNAL FOR NETHERLANDS INDIA

PUBLISHED BY THE ASSOCIATION FOR THE ADVANCEMENT OF MEDICAL SCIENCES IN NETHERLANDS INDIA

VOLUME LIX.

**Java Bookshop and Printer**
**Batavia ✞ 1919.**

# Some epidemiological remarks about the Flu in the Magelang Division, 1918.

BY

**P.B. van STEENIS,**

*Health Officer 2nd class.*

---

Before discussing epidemiology, a few remarks on etiology.

*What do we mean by Flu?*

The flu, or flu disease, first appeared in Spain. It is very similar to influenza, which became known to modern medical science from the epidemic of 1889-1893. The causative agent is often assumed to be the small bacillus that Pfeiffer found in 1892.

Diseases resembling it clinically and epidemiologically have been named flu or influenza. They have been described several times in the past and can be traced back to the 16th and 17th centuries.

Common descriptions of these epidemics include a violent disease (flu) that seizes the patients in full health and suddenly casts them onto the sickbed. In addition to fever the main symptoms involve the throat, larynx and bronchi, as well as further complications that vary across epidemics (for example, in 1830 in Paris strong intestinal symptoms reminiscent of cholera were observed, and in 1837 in France there was frequent pneumonia and severe hemorrhagic disorders). The diffusion pattern is that of a highly contagious disease, which spreads rapidly over large regions and does not miss a town or village. It affects half or more of the population, expanding at a speed that is limited by that of the movement of people.

In the 19th century there was a similar epidemic from 1830 to almost 1840. A subsequent one occurred only in the year 1889, going almost undetected by doctors, and lasting until 1893.

While this epidemic was the first to be investigated bacteriologically, it was not until 1892 that Pfeiffer discovered the bacillus influenzae, which is generally regarded as the causative agent.

We can therefore only use the latter epidemic as a comparison with the present epidemic. We then see:

1. There is great agreement on the clinical picture; in my opinion the differences can very well be explained by the fact that every epidemic of a disease has its own location- and time-specific character. The Indies epidemic of 1918 can be called an influenza epidemic marked by severe hemorrhages, pneumonia, and infirmities of the heart.

2. As Dutch doctors we are accustomed to associate the term influenza with the unpleasantly bleak foggy and rainy days, which start in October. Therefore, it is striking that the influenza was able to rage so violently in the tropical regions of Java.

It should be noted here that, although in Holland the trachea and bronchi are frequently affected in the cold autumn and spring months, in Netherlands India we often see these ailments at completely different times, chiefly at the end of the dry monsoon.

Moreover, influenza does not seem to keep to certain seasons in Europe; thus says Graves: It has not yet been possible to determine the factors favoring the development of the flu, for they seem to interact with the conditions of climate, season and temperature.

3. In Europe an extensive bacteriological study of the etiology of influenza has been started, and the results of the various researchers differ; while some found Pfeiffer's germ, many others did not.

Not much bacteriological research has been carried out in the Dutch East Indies and the bacillus of Pfeiffer has only been demonstrated in *Medan* by SNIJDERS.

This will lead many to question the similarity in the etiology of the epidemic of 1889 and the present one; in my opinion incorrectly for now.

A cursory overview of what was found in Europe may be briefly summarised here:

LAND and SLUIJTERMAN N.T.v.G. N°. 4 2nd half 1918. Influenza bacilli and gram negative diplococci from the throat scrape in many cases.

KRONER. B.K.W. N°. 27 1918. No influenza bacilli.

BRASCH. M.M.W. N°. 30 1918. Never influenza bacilli.

MANDELBAUM M.M.W. N°. 30 1918. Epidemiologically and symptomatologically the same picture as 1890. The influenza bacillus certainly plays no role. Causes unknown.

NICOLAI. N.T.v.G. N°. 6 2nd half 1918. The micrococcus catarhalis was always cultivated, (according to Prof. SPRONCK) a side infection.

OLJENICK. N.T.v.G. N°. 5 2nd half 1918. In Germany the influenza bacillus was repeatedly found.

LEVINTHAL. B.K.W. N°. 30 1918. A new breeding medium, with which the influenza bacillus was repeatedly identified.

LITTLE. Lancet N°. 4950 1918. Always gram positive diplococcus in throat scrapings. Never influenza bacilli.

SIMMONDS. M.M.W. N°. 32 1918. In Hamburg the influenza bacillus was often detected, repeatedly mixed with streptococcus.

MATTEUS. Lancet N°. 4952 1918. A new breeding medium: Blood, digested by trypsin with agar. A swab from the pharynx of 100 patients was taken. In all cases the influenza bacillus of Pfeiffer was found.

OSKAR MEIJER. B.K.W. N°. 34 1918.29 Autopsies. A streptococcus was always found in the spleen, which is certainly related to the disease. Never influenza bacilli.

SELTER. D.M.W. N°. 34 1918. Never influenza bacilli. Passing throat scrapings through a Berckefeld filter and aspirating this filtrate gave Selter and his assistant mild influenza symptoms after 24 hours. Is there a filterable substance in play here?

BERGMANN. D.M.W. N°. 34 1918. The influenza bacillus was almost always found in sputum. Not post mortem in the lung.

HIRSCHBURG. D.M.W. N°. 34 1918. Never influenza bacilli.

CITRON. B.K.W. N°. 33 1918. Never influenza bacilli. Always diplococcus.

DITRICH. M.M.W. N°. 34 1918. In the places where the inflammation is most noticeable, the influenza bacilli are detectable.

NEUFELD and PAPAMAEKU. D.M.W. N°. 43 1918. New breeding medium. Agar with defibrinated blood, in which the dye is dissolved. In many cases the bacillus of Pfeiffer can be detected in the sputum. Agglutination by the serum of patients.

CARL ROSE. B.K.W. N°. 44 1918. In 18% of cases influenza bacilli (in 15% pure culture).

R. DUJARRIE DE LA RIVIÈRE. Soc. of Biology October 12, 1918. Repeated detection of the influenza bacilli. A new working method: 10 cc. urine aseptically cultured in peptonized water with some chicken blood. In almost all cases, when other methods fail, this method will find Pfeiffer's bacillus.

So we have 10 researchers who were able to demonstrate the presence of the influenza bacillus more or less regularly, compared to nine who never succeeded. It is curious that several researchers using new culture media (those who do not mention this will have used blood agar according to the old prescription) had very good results; a change in the blood seems to be of great importance here: let it digest with trypsin or defibrinate and dissolve the dye.

This indicates a slight change in the biology of the Pfeiffer's bacillus, which is also not unusual in epidemics that are separated by a long gap in time. Perhaps this is the reason that so many negative culture trials were recorded.

We may, therefore, assume that Pfeiffer's bacillus occurred very frequently in Europe during the 1918 epidemic and that many more positive results would have been obtained if the culture media were improved.

What about the bacillus influenzae of 1889-1893?

Since the influenza bacillus was first described by Pfeiffer in 1892, there are no data on the rate of detection of the influenza bacilli, and the influenza bacillus was also not always identified in 1893. (However, this may be due in part to insufficient use of the bacteriological technique with the blood agar culture media). Comparisons cannot therefore be made here.

First, however, an important question must be answered:

Was Pfeiffer's bacillus influenzae really the cause of the influenza epidemic of 1889-1893 as well as the current epidemic?

Koch's postulates require: 1°. The bacillus must always be detectable. 2°. It should never be found in other diseases. 3°. It will produce an analogous clinical picture in animals by experiment. Even though this is the case for most bacterial diseases, none of the three postulates are fulfilled in the case of bacillus influenzae.

There is more.

*a*. The influenza bacillus is not found exclusively in cases of influenza. An accompanying bacillus that is indistinguishable from the bacillus influenzae is also very often found in cases of whooping cough, and likewise in measles patients, who also cough when healthy, albeit rarely.

*b*. Agglutination reactions of bacillus influenzae with the serum of influenza patients do not give consistent results, although clotting is sometimes very marked.

It is increasingly apparent that agglutination is not always specific; for example, in the case of intestinal flora it has been found that coli bacilli also clump together in dysentery serum if they have lived in the same environment with dysentery bacilli for a long time.

Variable agglutinations therefore do not support the assumption of a relationship between serum and bacillus.

It is very doubtful whether Pfeiffer's bacillus influenzae is the cause of the Spanish flu; nor has it been shown with any certainty that it was the cause of the epidemic of 1889.

So we would do well to name the pandemic of 1918:

*Flu or influenza, a disease with an unknown causative agent, epidemiologically and symptomatologically identical to the pandemic of 1889.*

However, new points of view are emerging as regards etiology.

NICOLLE and LEBAILLY (Presse médicale) found a filterable virus. They summed up their research for the Académie thus:

1. The sputa of flu patients are infectious during the acute period.

2. Macacus cynomolgus is susceptible to nasal and subconjunctival infection by the influenza virus.

3. The agent of the flu appears to be a filterable organism. Successful infections with the filtrate in laboratory animals and humans.

4. There is no virus in the blood.

GIBSON, BOUWMAN and O. CONNER (Br. Medic. Journal 1918 N°. 3024) fully confirm these conclusions.

In macacus rhesus (nasally or subconjunctivally infected) they found foci of haemorrhagic fibrinous exudate in the lower lobes without typical bacilli.

The flu will therefore probably have to be classified under the group of diseases caused by a filterable virus.

Since we must for the time being deny Pfeiffer's bacillus a certain etiological significance, it is not necessary to discuss this micro-organism.

I will only briefly indicate a possible explanation for the fact that the influenza bacillus can be detected in such a large number of cases in influenza sufferers, often in pure culture.

In 1917, before the current pandemic, DAVIS (Journal of Infectious Diseases) started an investigation into the most favorable breeding ground for influenza bacilli and found that the influenza bacillus always needs hemoglobin and with it can grow on agar, in broth, etc. The most favorable media, however, were obtained by adding unheated living material, e.g. a piece of potato or fresh meat; the presence of other microbes, especially the staphylococcus aureus, is also strongly growth-promoting.

We have already seen bacillus influenzae (or at least a micro-organism that cannot be distinguished from it) as a secondary infection in whooping cough and coughing measles patients, both conditions with strong mucus formation in the bronchi. With influenza we also see strong secretions in the bronchi, always slightly hemorrhagic, and sometimes quite strongly so.

Naturally we have to consider the possibility that Pfeiffer's bacillus influenzae is an ordinary harmless throat or bronchus inhabitant, which is seldom detected because it occurs in small numbers or is not coughed up. In addition, the culture media commonly used may be unsuitable for its growth.

If a strongly secreting bronchitis develops, always with small blood admixtures and, especially in influenza, often quite strongly haemorrhagic, then, according to DAVIS, we have the ideal breeding medium in the bronchus, a protein containing fluid with haemoglobin and living tissue in the vicinity.

As a result, the bacillus influenzae will grow, even into a pure culture. The real agent may also be present, but it is still inaccessible to our methods of research.

*Epidemiology of the Flu.*

Given that the epidemic of Europe is the same as that of the Indies, the course of events, so far as can be deduced from newspapers, points to this: Japan, China and Singapore were infected from America and, via Singapore, Java was infected in the port cities of Batavia, Semarang and Surabaya.

In Holland it could be conclusively shown that the disease spread by contact (v.d. VELDE N.T.v.G. 2nd half N°. 7. 1918) and quite certainly the most common mode of spreading was the coughing up of infected mucus particles directly from the sick to the healthy. Bacilli carriers also play an important role here.

*The observations made in the Magelang department have taught us the following:*

First of all, it should be noted that Dr. VISSER and I did not have time for accurate observations; [pandemic] control was our N°. 1 priority.

Moreover, in view of the ignorance of the population and their limited ability to observe themselves and their environment, such an endeavor would often be a waste of effort. Accurate observations can therefore be expected more from the practicing physicians, who sometimes happen to be able to make good observations.

---

*I. Some statistical data on the epidemic in the Magelang division.*

*a*. Severity of the epidemic

If we compare the epidemic with that in Europe, we see here a death rate that is appalling, even compared to the death rate in Europe.

STATEMENT

*Mortality rates over 7 consecutive weeks from the influenza epidemics in Amsterdam, Magelang division and Krasak sub-district.*

| Influenza epidemic in | Weekly mortality in ‰ (calculated per year) | | | | | |
|---|---|---|---|---|---|---|
| Total inhabitants | Amsterdam 600,000 | | Magelang division 563,208 | | Krasak sub-district 19,887 | |
| | Total mortality | Influenza mortality | Total mortality | Influenza mortality | Total mortality | Influenza mortality |
| Total mortality and influenza mortality in ‰ (calculated on an annual basis) during seven consecutive weeks of the epidemic. | 11.1<br>14.3<br>26<br>39.2<br>52.7<br>42<br>31.5 | 1.3<br>1.9<br>13.6<br>26.8<br>40.3<br>27.6<br>19.1 | ---<br>92<br>196<br>274<br>221<br>151<br>98<br>67 | ---<br>66<br>170<br>248<br>195<br>125<br>72<br>41 | 86<br>252<br>605<br>507<br>301<br>161<br>78 | 60<br>226<br>579<br>481<br>275<br>135<br>52 |
| Time period | 29 Sept.- 16 Nov. 1918 | | 1 Nov.- 19 Dec. 1918 | | 25 Oct.- 12 Dec. 1918 | |

Among an approximately equal number of inhabitants in Amsterdam and the Magelang division, we see that the mortality in the latter region is much higher than in Amsterdam, and in the two highest weeks, it is six times as high. In smaller areas, including the sub-district of Krasak, the difference, calculated on annual basis, is even more noticeable, and in Tinjomoyo village, with 822 inhabitants, 60 people died in the week of 22-28 November, which works out to 3800 ‰ per year.

*b.* As to the relationship of both the sexes and the ages of the deceased during the epidemic, no data (of a reliable nature) are available to me for the entire Magelang division; [however, we have data for] some sub-districts, including the sub-district Magelang. We then see:

*Mortality in the Magelang sub-district October 1 - December 31, 1918.*

| | Total | 0-9 years | 10-19 years | 20-29 years | 30-39 years | 40-49 years | 50-59 years | 60 years and higher |
|---|---|---|---|---|---|---|---|---|
| Men | 386 | 47%<br>181 | 5.7%<br>22 | 3.6%<br>14 | 9.1%<br>35 | 10.1%<br>39 | 7.2%<br>28 | 17.3%<br>67 |
| Women | 391 | 45.8%<br>179 | 7.7%<br>30 | 8.2%<br>32 | 14.3%<br>56 | 9.7%<br>38 | 3.8%<br>15 | 10.5%<br>41 |

*Mortality during the Amsterdam epidemic of 1918.*

| | 0-1 years | 1-4 years | 5-13 years | 14-19 years | 20-49 years | 50-64 years | 65 years |
|---|---|---|---|---|---|---|---|
| Persons | 3% | 13% | 8.7% | 8.3% | 51.9% | 8.7% | 6.4% |

The child mortality rate in the Indies is appalling; while 25% of all deaths in Amsterdam occurred among people up to the age of 13, the rate in the Indies was double that.

It is also notable that in the 20-40 year age group the mortality among women is so much higher; this is partly due to extremely frequent influenza-associated deaths during childbirth (at term or premature).

*2. How can the high mortality be explained?*

On this it can be noted:

*a.* There are unknown factors which cause local variations in the intensity of the epidemic; thus we see that particular households have been severely affected, as well as certain cities, such as Amsterdam, without an identifiable cause. It is unlikely that such factors could explain the severity of the Indies epidemic, since here too mortality varies greatly in different places.

*b.* A circular has been sent by the Headquarters of the Civil Medical Service to indicate the great importance of proper nutrition and authorising the Assistant Residents to provide food from the government. Certainly there was a shortage in several places in the months of the influenza, but in the Magelang ward the nutritional status was excellent, just as good as in other times. While the generally poor nutrition of the Javanese people may have contributed to a low resistance to influenza, it is still wrong to consider the high mortality to be the result of the scarcity of food.

In places where real food shortages existed, this factor was of paramount importance, and the foregoing is not in the least intended to call into question the importance of an adequate food supply for influenza patients.

*c.* Poor housing conditions among the Javanese population in the Indies are blamed for all kinds of ailments, including influenza.

One should not exaggerate this factor too much; lately all salvation seems to have been expected from a housing ordinance. It is certain, however, that most tropical diseases would change only slightly if all native dwellings were upgraded to a presently attainable level. The influenza certainly would have changed little in character; we saw infections in well-built houses that were as severe as those in the slum dwellings. The villages in the south of the Magelang division, generally with large, spaciously built houses covered with tiles and large spacious yards, suffered a toll from the influenza just as severe as the poor, small shacks built on top of each other in the city.

The mortality in the rural villages (all with large yards) was certainly no lower than in the cities, while the medical treatment of the native population is uniformly inadequate everywhere.

*d.* While all of these factors contributed to the great mortality, the *reason* that thousands died of influenza in Java and around it is the folly and utter ignorance of hygiene among the Natives, as well as the complete lack of medical care for the native population.

*3. Severity of the infection in certain homes.*

I noted that the infection can be much more severe in one house than in another, with no apparent cause.

For example, one child and four grandchildren died in the house of a Dokter-Java; most had come to visit from outside after the first grandchild had died. The nutritional status of all was very good, and medicines were also used from the beginning.

In Kalikuto village I found that, of a family of a mother with four children and a grandfather, the mother and a child had died next to each other, while a second child had died on the ground in front of the main room. It was at the beginning of the epidemic and I suspected pneumonia; the other two children had a high fever and dyspnea. I isolated both of them with the old man in another house; both recovered. We completely miss the causes, perhaps there are changes in the virulence of the causative agent of the influenza; adherents of the parasitic transfer hypothesis could more easily explain these observations.

*4. Spread of the influenza in villages.*

In most villages we saw that the influenza raged like a fire, sometimes with small outbreaks of 5 to 10 houses, and sometimes with larger ones of up to 25 houses. We can expect this in the case of a contagious disease such as influenza --- the coughing patients are nodes of infection, infecting their neighbors. These in turn infect those living next door, and thus the outbreak develops. As a result, we see clusters of recovered people adjacent to outbreaks where the infection is at its highest, and still other outbreaks, which are just developing, in the same village. By contrast, an entire village can also be affected almost all at once, with immediate and dire mortality. This form of the epidemic, which strikes a village in its entirety at the same time, we see, for example, in Tinjomoyo, Sukorejo and Ngadirejo villages.

*Deaths per day.*

| Date | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | November. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Tinjomoyo pop. 822 | — | — | — | 1 | 2 | 6 | 11 | 6 | 27 | 4 | 6 | |
| Sukorejo pop. 898 | — | — | — | — | — | 3 | 2 | 3 | 3 | 11 | 5 | |
| Ngadirejo pop. 892 | — | — | — | — | — | 2 | 2 | 3 | 1 | 9 | 10 | |

I will return to this observation of the extremely rapid spread of the influenza, which only occurs in higher regions. It gives the impression that the entire atmosphere is a carrier of large quantities of infectious germs, so that the whole village could be infected all of a sudden.

*5. The spread of influenza from village to village.*

In order to obtain further information to answer this question, I have produced weekly maps on which the newly infected villages are shown in color, while previously infected villages are colored black.

To be able to record a new infection, we should have reliable disease data; however, there are none.

Given the rapid spread of influenza and the high mortality, however, we can assume that the mortality in a village will significantly rise approximately one week after the start of the epidemic. While we set the baseline mortality at 0.5 ‰ per week, I assumed that a village was infected with influenza when the mortality was 2 ‰ per week. (This number is not chosen arbitrarily, but is taken from the numbers of weekly deaths.) The whole epidemic may therefore be displaced by plus or minus one week, but the data hold their value for comparison.

It should be noted that no epidemics of any other nature occurred during the time of observation. The first minor epidemic [of influenza] remains outside the scope of this discussion.

Week of 11-17 October. Two villages in Magelang and one village close to Muntilan have been infected, all located on the main road connecting Yogyakarta, Magelang, and Ambarawa. I also already pointed out the role of proximity of the main bazaars. It is understandable that the infections have taken place here, because this is where, for example, salesmen from Yogyakarta, where the influenza was already prevalent at that time, come.

Week of October 18-24. The infection is spreading in Magelang. Another case has been recorded in Muntilan in addition to two cases, at Mendut and Ngluwar bazaar, both on fairly busy roads, and at a village at Kayoran, where there is also a bazaar.

In the south of the Magelang division, many people came from Yogyakarta, where there was already a severe food shortage. They especially visited the bazaars to try to satisfy their hunger in the well-provisioned Magelang.

Week of October 25-31. The Magelang outbreak is expanding, as is the one in Muntilan, albeit slowly. An outbreak is also developing around the Ngluwar bazaar. Furthermore, Kalisalak village near Salaman bazaar, a very busy market, has been newly infected as has Krasak with its small bazaar and Banjarsari village at the busy Tempuran bazaar. Senengsari village was infected by contact with the severe influenza outbreak in Tembarak (Temanggung division).

Week 1-7 November. The infection is spreading in the districts of Salam and Muntilan. Several villages away from the main road, although still somewhat linked to the roads, are infected.

The Magelang outbreak is constantly expanding, as are outbreaks in the sub-districts of Bandongan and Candimulyo.

The bazaars at Krasak and Salaman have become centres of outbreaks.

Kaweron village is infected via Kalegen bazaar, while two villages are also affected by the Grabag bazaar.

Secang and Windusari are believed to be continuously infected from Tembarak.

Week of November 8-14. The existing outbreaks are expanding rapidly. A new large outbreak is developing around the Sawangen bazaar.

Week of November 15-21. Considering the Ngablak sub-district with a large bazaar, but very inaccessible at a height of 1375 meters, we do not see the contamination emanating from the bazaar. However, the sub-district is infected from two sides, Grabag and Pakis.

Over the following weeks the influenza was widely diffused and almost all villages were affected.

*From these maps we see* that, with a few exceptions, the first-affected villages were located on the main roads, chiefly on the *Yogyakarta-Magelang-Ambarawa* and *Purworejo-Magelang* roads. Both *Yogyakarta* and especially *Purworejo* were affected by influenza before *Magelang* and were very important sources of infection.

Moreover, with a few exceptions, the outbreaks appear to start close to the large bazaars and to develop around them. This is very important. Everybody knows the bazaars, where thousands of natives appear twice a week, swarming together. One could not create a better opportunity for the infection to spread rapidly.

If we refer to the maps, we see that once the central bazaar is infected, the villages around it are quickly affected; dozens of people from each village visit the central bazaar, running the risk of becoming infected there and transmitting the germs to their village.

Having seen the dangers of these bazaars so clearly on the map, it seems to me that in the event of a repeat of the influenza epidemic (for example, next year), we must take measures against it. Domestic Administration officials I spoke to considered it impossible to close a large bazaar for 2 to 3 weeks - that would cause famine - but in any case measures should be taken to mitigate the dangers of these bazaars; closely monitoring and removing feverish patients would help somewhat, but how long the recovered influenza patients remain infectious, and what is the status of healthy bacilli carriers, these are unanswerable questions.

These maps show that the influenza first spread along the main roads, where human traffic is greatest; moreover, the first central village markets to be affected were locations where hundreds of people from different regions come together twice a week. There are three possibilities for propagation:

1. Person-to-person infection by the coughed up infectious sputa – airborne droplet infection.
2. Airborne infection at a great distance.
3. Parasitic transmission.

### 1. Droplet infection at a short distance

In Europe it has been fairly certainly demonstrated that in many cases this is the usual mode of infection, from person to person. All conditions are favorable for this in the Indies as well; in all the infected villages sick patients were seen huddled together, coughing in the faces of the healthy.

This also appears to have been common practice in the Magelang division; the weekly maps shown indicate the role of human traffic; the important role that the bazaar centres apparently play in this has also been explained.

2. Airborne infection at a great distance.

By this is meant that the agent is atomised and travels through the air over great distances, e.g. 10 meters to 1 kilometer, and remains virulent enough to infect healthy people. So this is basically the airborne droplet infection, except that it occurs over great distances.

With pneumonia, a good example of airborne droplet infection, this never happens over great distances in Java --- these always turn out to be infections at a short distance. In the Indies, isolation in a hut with barbed wire a few meters away is sufficient to make the pneumonia patient harmless.

In the case of influenza, however, there is evidence that that the influenza germs carry the infection over greater distances through the air.

In general, we can say that airborne pathogens will be killed by the action of sunlight and dehydration. The greatest risk of respiratory infections will therefore exist in places where these factors are absent or present in lower intensity.

Now we see in the high mountain regions, where the humidity is great (the influence of the sun is absent in the evening and morning hours), the spread of the influenza is occurring much faster than in the lower regions.

To demonstrate this, I give the following statement.

| Sub-district | Altitude in Meters above sea level | Number of villages | Number of villages newly infected in the 4th quarter of 1918 | | | | | | | | | | Mortality per sub-district in the 4th quarter |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 4th wk. | 5th wk. | 6th wk. | 7th wk. | 8th wk. | 9th wk. | 10th wk. | 11th wk. | 12th wk. | 13th wk. | |
| Borobodur. | 350 | 22 | — | 1 | — | 5 | 8 | 5 | 2 | 1 | — | — | 485 |
| Muntilan. | 350 | 16 | 1 | 1 | 9 | 4 | 1 | — | — | — | — | — | 587 |
| Krasak. | 300-400 | 19 | — | 3 | 4 | 7 | 4 | 1 | — | — | — | — | 832 |
| Pakis. | 800-1500 | 20 | — | — | — | — | 12 | 8 | — | — | — | — | 885 |
| Ngablak. | 800-1500 | 16 | — | — | — | — | 6 | 9 | — | — | — | — | 730 (1 village was not infected). |

We can see from this that in the high mountain sub-districts all villages had a serious influenza infection in a span of 2 weeks, while this took 5 to 7 weeks in the lower sub-districts.

In addition, in the mountain sub-districts the distance from one border to the other is 3 to 4 times greater than in the plains districts; the villages at the top are at a distance of some 2 *paal* [one *paal* is approximately a mile] while the villages below are 300-500 meters apart.

The infection in Krasak was just as severe as that in Pakis and Ngablak.

The traffic among villages at the top is certainly not greater.

*I therefore consider it not impossible that in the mountainous regions of Pakis and Ngablak, with their great humidity, the influenza was blown from hamlet to hamlet and thus caused the extremely rapid spread.*

I will recall briefly the rapid spread throughout the village, as was observed in Tinjomoyo at an altitude of 800 meters.

### 3. Parasitic mode of infection.

Those who declare the epidemic to be identical with fevers such as pappataci fever and dengue believe this. Epidemiology argues against this view:

*a*. Extremely rapid spread, although this would be possible [with the parasitic mode]; however, it much more strongly argues against it:

*b*. The fact that from the North of Europe to the equator we should accept that a parasite should transmit the causative agent. The existence of such a parasite is very unlikely.

*c*. Numerous observations would be inexplicable, for example the manner of distribution outlined above in the Magelang division.[1])

In summary, I consider the spread of the influenza in the Magelang division to be explained:

I. By coughing up infected mucus from the throat and nasal cavities, particles of which are directly inhaled by healthy persons. This mode of infection is the most common and explains the outbreak of influenza in the village.

The Indies bazaar is of great importance, as the spread of the influenza takes place through human movement.

II. In the high, humid mountain regions I consider it likely that long-lived germs of infection blow through the air during the early morning or evening hours from village to village over greater distances.

*Magelang*, March 1919.

---

1) We can reasonably assume that the epidemic was spread as such by contact infections; however, this does not preclude the possibility of other infection modes in addition. In this connection, the following may be noted here.

On clinical and etiological grounds, the first initiator of this topic, Mr. NEEB, pointed to the great similarity between influenza and pappataci fever, which is transmitted by phlebotomas.

An example of the same bacillus being transmitted by parasites and as contact infections can be found in plague (bubonic plague transmitted by fleas, pulmonary plague as airborne droplet infection and contact infection).

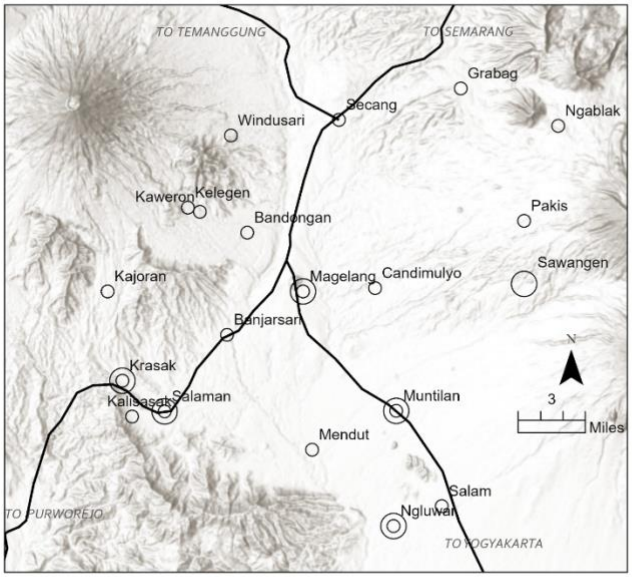
The influenza can therefore also normally be classified as a disease grouped with pappataci fever, dengue, etc. with parasitic transmission, while, as in 1918, it occurs under conditions completely unknown to us as a contact infection of the respiratory organs with a much more serious course.

(Also in the case of pneumonia, we lack any insight into why it sometimes occurs as a severe epidemic as in Manchuria in 1911).

## Appendix: Mapping van Steenis' account of the spread of influenza in Magelang

The following pages contain a sequence of maps, rendered in ArcGIS, showing the weekly progression of the influenza pandemic across Magelang. While van Steenis' report mentions maps, none were available in the digital version of the article used for this translation. These maps follow the text in the preceding report.

## Centers of Outbreaks and Locations of Observed Cases in Magelang Regency: Week Ending October 10, 1918




LEGEND

- MAIN ROAD

WEEK OF FIRST OBSERVED CASE

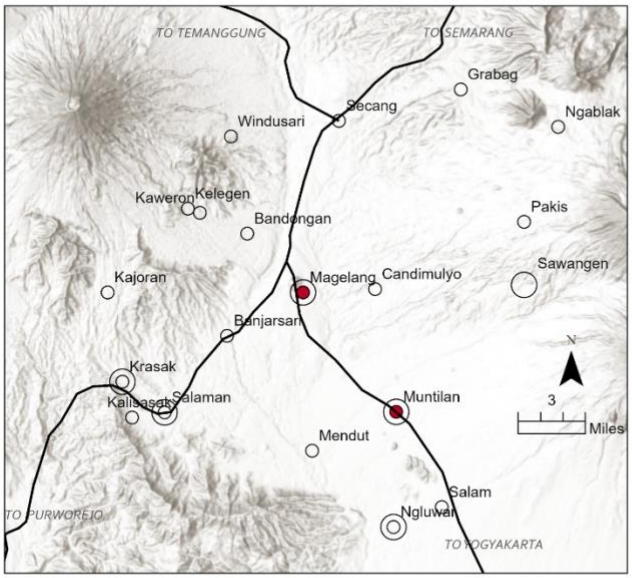
- OCTOBER 11 TO 17
- OCTOBER 18 TO 24
- OCTOBER 25 TO 31
- NOVEMBER 1 TO 7
- NOVEMBER 15 TO 21

WEEK LOCATION BECOMES CENTER OF OUTBREAK

- OCTOBER 25 TO 31
- NOVEMBER 1 TO 7
- NOVEMBER 8 TO 14

SOURCES: ESRI, AIRBUS DS, USGS, NGA, NASA, CGIAR, N ROBINSON, NCEAS, NLS, OS, NMA, GEODATASTYRELSEN, RIJKSWATERSTAAT, GSA, GEOLAND, FEMA, INTERMAP AND THE GIS USER COMMUNITY

## Centers of Outbreaks and Locations of Observed Cases in Magelang Regency: Week Ending October 17, 1918




LEGEND

- MAIN ROAD

WEEK OF FIRST OBSERVED CASE

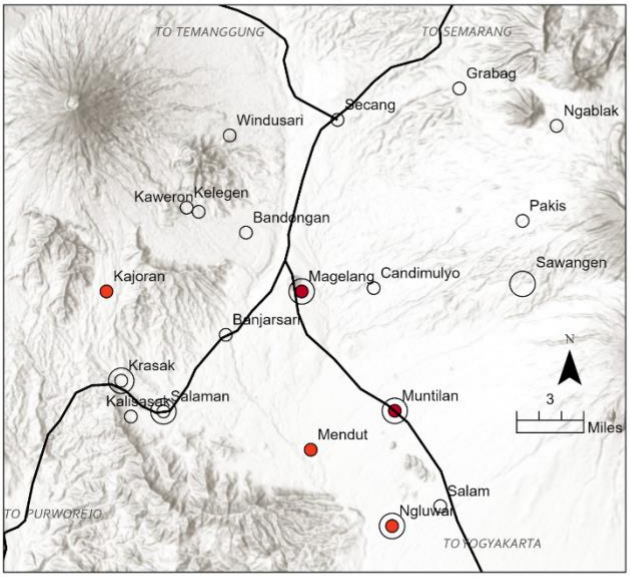
- OCTOBER 11 TO 17
- OCTOBER 18 TO 24
- OCTOBER 25 TO 31
- NOVEMBER 1 TO 7
- NOVEMBER 15 TO 21

WEEK LOCATION BECOMES CENTER OF OUTBREAK

- OCTOBER 25 TO 31
- NOVEMBER 1 TO 7
- NOVEMBER 8 TO 14

SOURCES: ESRI, AIRBUS DS, USGS, NGA, NASA, CGIAR, N ROBINSON, NCEAS, NLS, OS, NMA, GEODATASTYRELSEN, RIJKSWATERSTAAT, GSA, GEOLAND, FEMA, INTERMAP AND THE GIS USER COMMUNITY

## Centers of Outbreaks and Locations of Observed Cases in Magelang Regency: Week Ending October 24, 1918



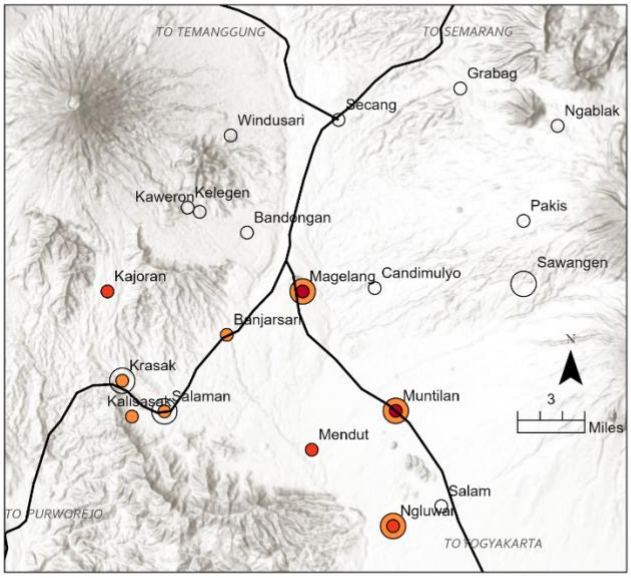

LEGEND
- MAIN ROAD

WEEK OF FIRST OBSERVED CASE
- OCTOBER 11 TO 17
- OCTOBER 18 TO 24
- OCTOBER 25 TO 31
- NOVEMBER 1 TO 7
- NOVEMBER 15 TO 21

WEEK LOCATION BECOMES CENTER OF OUTBREAK
- OCTOBER 25 TO 31
- NOVEMBER 1 TO 7
- NOVEMBER 8 TO 14

SOURCES: ESRI, AIRBUS DS, USGS, NGA, NASA, CGIAR, N ROBINSON, NCEAS, NLS, OS, NMA, GEODATASTYRELSEN, RIJKSWATERSTAAT, GSA, GEOLAND, FEMA, INTERMAP AND THE GIS USER COMMUNITY

## Centers of Outbreaks and Locations of Observed Cases in Magelang Regency: Week Ending October 31, 1918



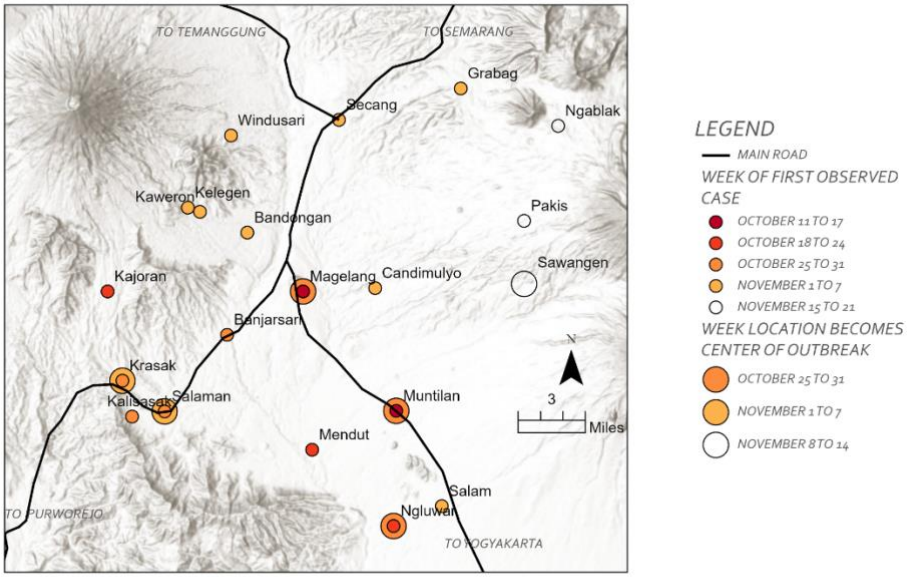

LEGEND
- MAIN ROAD

WEEK OF FIRST OBSERVED CASE
- OCTOBER 11 TO 17
- OCTOBER 18 TO 24
- OCTOBER 25 TO 31
- NOVEMBER 1 TO 7
- NOVEMBER 15 TO 21

WEEK LOCATION BECOMES CENTER OF OUTBREAK
- OCTOBER 25 TO 31
- NOVEMBER 1 TO 7
- NOVEMBER 8 TO 14

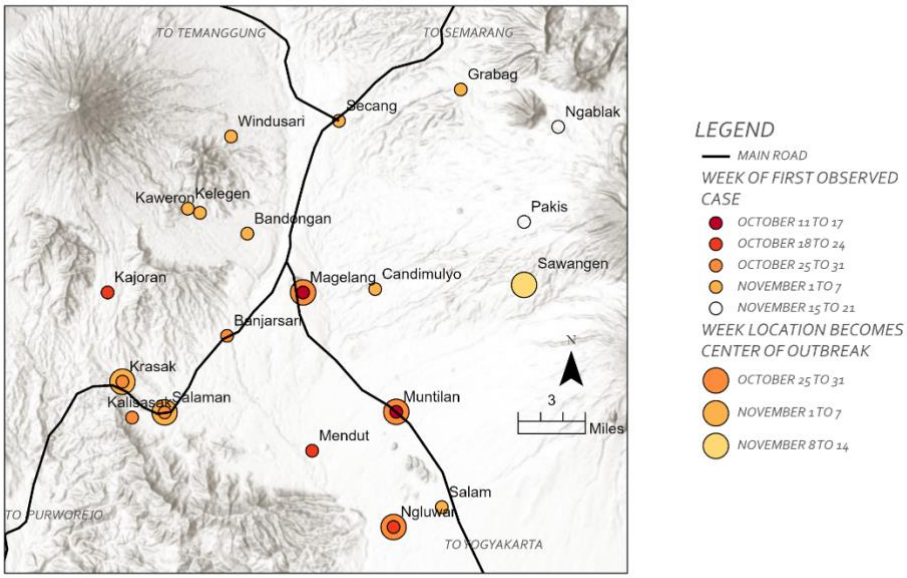
SOURCES: ESRI, AIRBUS DS, USGS, NGA, NASA, CGIAR, N ROBINSON, NCEAS, NLS, OS, NMA, GEODATASTYRELSEN, RIJKSWATERSTAAT, GSA, GEOLAND, FEMA, INTERMAP AND THE GIS USER COMMUNITY

## Centers of Outbreaks and Locations of Observed Cases in Magelang Regency: Week Ending November 7, 1918



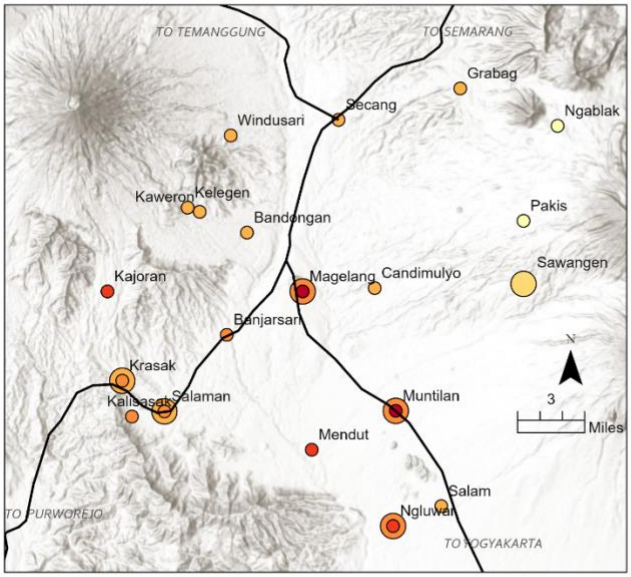

SOURCES: ESRI, AIRBUS DS, USGS, NGA, NASA, CGIAR, N ROBINSON, NCEAS, NLS, OS, NMA, GEODATASTYRELSEN, RIJKSWATERSTAAT, GSA, GEOLAND, FEMA, INTERMAP AND THE GIS USER COMMUNITY

## Centers of Outbreaks and Locations of Observed Cases in Magelang Regency: Week Ending November 14, 1918




SOURCES: ESRI, AIRBUS DS, USGS, NGA, NASA, CGIAR, N ROBINSON, NCEAS, NLS, OS, NMA, GEODATASTYRELSEN, RIJKSWATERSTAAT, GSA, GEOLAND, FEMA, INTERMAP AND THE GIS USER COMMUNITY

## Centers of Outbreaks and Locations of Observed Cases in Magelang Regency: Week Ending November 21, 1918




LEGEND

MAIN ROAD

WEEK OF FIRST OBSERVED CASE

- OCTOBER 11 TO 17
- OCTOBER 18 TO 24
- OCTOBER 25 TO 31
- NOVEMBER 1 TO 7
- NOVEMBER 15 TO 21

WEEK LOCATION BECOMES CENTER OF OUTBREAK

- OCTOBER 25 TO 31
- NOVEMBER 1 TO 7
- NOVEMBER 8 TO 14

SOURCES: ESRI, AIRBUS DS, USGS, NGA, NASA, CGIAR, N ROBINSON, NCEAS, NLS, OS, NMA, GEODATASTYRELSEN, RIJKSWATERSTAAT, GSA, GEOLAND, FEMA, INTERMAP AND THE GIS USER COMMUNITY

## Glossary

Following are the titles of the journals for which abbreviations are presented in the literature review on the second page of the report:

| | |
|---|---|
| N.T.v.G.: | Nederlands Tijdschrift voor Geneeskunde (Dutch Journal of Medicine) |
| B.K.W.: | Berliner Klinische Wochenschrift (Berlin Clinical Weekly) |
| M.M.W.: | Münchener Medizinische Wochenschrift (Munich Medical Weekly) |
| D.M.W.: | Deutsche Medizinische Wochenschrift (German Medical Weekly) |

In the discussion of transmission of influenza, the term 'paal,' which is approximately one mile, is used to signify distance.