

## Supplementary Materials for

### Extraordinary curtailment of massive typhus epidemic in the Warsaw Ghetto

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Published 24 July 2020, *Sci. Adv.* **6**, eabc0927 (2020)  
DOI: [10.1126/sciadv.abc0927](https://doi.org/10.1126/sciadv.abc0927)

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### **SM1 Typhus spread, and modelling assumption of underlying contact process**

**Typhus transmission:** Typhus is a disease transmitted by the microorganism *Rickettsia prowazekii* but spread by its vector the human body louse (*Pediculus humanus humanus*) (24, 25). Lice bite infected humans for blood meals, and in the process become infected with typhus (*R. prowazekii*) themselves. *R. prowazekii* multiply and eventually appear in the louse's faeces. After being bitten, the host responds by scratching, and rubs the faeces direct into the scratches. In this way *R. prowazekii* enters the host's body to eventually reach the blood stream. At the same time the lice leaves large numbers of nits (eggs) in the host's clothing to regenerate itself. Typhus spreads rapidly when there is overcrowding, cold, poor hygiene, and filth - exactly the conditions of the ghetto (11, 16, 18, 30, 44).

The following excerpts from the chief historians and diarists of the Warsaw ghetto, comment on the importance of the contact process in spreading typhus. The SEIR model should give realistic results since it is built on the assumption that infection is transmitted via such a contact process.

**Stanislaw Adler (21)** (p.42) "...the death toll had just begun to climb. The spotted typhus epidemic spread through the ghetto in thousands of ways. The refugee center blazed with its fire. From there, thousands of beggars spread it through the streets. Lice infestation was everywhere. Merely walking a hundred meters along the street exposed one to great danger. One had only to brush against a person infested with lice to discover these dangerous vermin in the evening while examining each piece of clothing; then there was the terrible wait in fear and trepidation for the fourteenth day to see whether the temperature would rise sharply. The children of the streets contributed most to the spread of the epidemic. They were everywhere, running at dizzying speed through the crowds, rubbing against every pedestrian. Among children, the disease is not dangerous but they spread it, without being aware of what they were doing. "

**Adler (21)** (p.304): "From time to time somebody passing by brushes against me. I am horrified because so many people are infected with lice... The danger of that infection [typhus] terrifies me."

**Isaiah Trunk (17) (p.39):** "Over-crowding in the ghetto created the most favourable conditions for the spread of contagion, both in the street where people moved in thick masses, and in the homes."

**The Diary of Mary Berg (p.66):** "The chief carrier of this terrible disease [typhus] is the clothes louse, and it is hard to avoid encountering this repulsive insect. It is enough to walk in the street and rub against someone in the crowd to become infected."

**Kaplan (45)** (p.128) wrote: "There is a great confusion of pedestrians, street vendors, overloaded porters, carriages and delivery carts, beggars and all sorts of creatures whose proximity you cannot bear for fear of lice. The fear of lice obsesses all of us, for the tiny creatures are carriers of lice." p.276

**Bernard Goldstein (28)**\_ "The street was packed with people: death, death, and more death; yet there was no end to the overcrowding. People elbowed their way through the noisy throngs, fearing to touch each other, for they might be touching typhus."

## **SM2 Other factors leading to typhus and its control**

Here, we present examples from the literature concerning various factors that both contributed to the epidemic and could be controlled to reduce the intensity of the epidemic.

**1) Disposal of waste.** Rubbish and household waste disposal outside the ghetto was prohibited by the Germans. Adler (21) noted that the epidemic intensified because of the buildup of waste: “Month by month ... more and more piles of decomposing waste grew. ...., making it the perfect breeding place for lice. In the middle of 1941, when the Nazis eventually permitted waste disposal from the quarter.” Nevertheless, the problem plagued the ghetto throughout 1942 as well (17).

**Stanislaw Adler (21):** “The epidemic intensified not only because people were herded in from different places, or because of the cramped living conditions or extreme starvation resulting from the impossibility of regular provision of food, but also because, despite continual requests submitted by the Judenrat, the Germans did not permit the disposal of rubbish and household waste. Month by month, in each courtyard, more and more piles of decomposing waste grew. The burning of rubbish or its transport into burnt-out buildings was a palliative which did not solve the problem at all. This situation did not change until the middle of 1941, when the Nazis eventually permitted waste disposal from the quarter.”

**Isaiah Trunk (17) (p.39):** The primary cause for the high rate of mortality in the Warsaw ghetto, from which all other causes flowed, was the ghetto itself. (1) In the first place, the ghetto brought about a deterioration of the hygienic and sanitary condition of the population. Because of the shortage of electricity, gas, and fuel, people washed less frequently and hardly if ever were able to take hot baths. The shortage of soap and fuel meant that clothing and linens were changed less frequently. The dwellings being unheated, were not aired, the houses and the courtyards were dirty, the garbage was removed very seldom (from Krochmalna Street no garbage was removed for six months). The incessant worries and misfortunes, the constant hunger resulted in laxness and indifference to personal cleanliness. Lice infestation became widespread and increased especially among the refugees and deportees. (2) Over-crowding in the ghetto created the most favourable conditions for the spread of contagion, both in the street where people moved in thick masses, and in the homes. (3) The maintenance of the food supply in the ghetto on a starvation level not only yielded a large number of direct victims of hunger but also weakened resistance to disease.”

**Chaim Kaplan (45) p.299;** “The negligence of the Health Department ... has turned the ghetto into a garbage dump and a huge public privy. Frozen water and sewage pipes have forced us to make latrines of stairways and yards. We are surrounded by stinking filth, and when the spring thaws melt the frozen dung heaps who knows what ghastly diseases will let loose on us then?”  
.....

**2) Change of ghetto administration in May 1941.** According to Browning, the initial German administrators (Schön and Palfinger) of the ghetto were “attritionists” with the goal of liquidating the ghetto purely through starvation. Their anti-Semitic phantasies of immense Jewish wealth made them deliver food only in exchange of payment, which the impoverished Jews and the Jewish Council simply could not provide . This changed in May 1941, when the new “productionist” ghetto commissioner Auerswald was appointed with the intention to build a self-sufficient and self-sustaining ghetto economy. The “productionists” believed that sources of labour should not be wasted and therefore some component of the ghetto residents should be given minimal food and nutrition so they could work. By Fall 1941, the ghetto was increasing its economical value for Germans and thus was granted more food. (11).

Other methods for suppressing the outbreak were implemented by the Germans, but they had questionable effectiveness and often “worse than the epidemic itself”(17). The Germans set up quarantine stations and disinfection bath houses which mostly problematically exposed many healthy residents to typhus. AS to vaccines, they were in any case experimental, difficult to obtain, and not always effective.

### **SM3 Numbers of typhus cases:**

The following quotes from the literature indicate that there were approximately 100,000 cases of typhus in Warsaw Ghetto. Some report more, others slightly less. Given the reported cases in Trunk’s dataset used in Fig.1 amounts ~20,000 cases, this would indicate a reporting rate in the vicinity of 20-25%. Many of the authors listed were highly respected epidemiologists, medical specialists or historians who were on location in the ghetto.

**Hagen:** Wilhelm Hagen was the German’s Chief Medical Officer and was a highly trained epidemiologist who specialised in typhus and tuberculosis. Christopher Browning (11) p.154 writes that Hagen estimated 20-25% of typhus cases were reported. Since some 18,530 cases were reported this implies 74,000 to 93,000 true cases of typhus.

**Professor Yisrael Gutman (19):** “In the course of that year (1941), 14,661 cases were reported, but for all practical purposes even this official figure is far from exact, and it is assumed to have represented only 25 to 30 percent of the actual number of cases (some estimates go as high as over 100,000 cases). “

**Dr. Mordechai Lenski (26)** “according to my estimate, the number of [typhus] patients should be set at about 100,000.” “... from September 1939 to September 1942, 80,000 persons died of disease, and of these, 18,000 died of starvation.”

**Professor Jakub Penson (20)** (Testimony): “Another epidemic broke out in June 1941 and continued until June 1942. It spread not only in “transit camps” [for] homeless people, like the first epidemic had done, but it swept all over the Ghetto, infecting about 100,000 people. This epidemic was a lot more acute than the first one, with a death rate of 20 per cent (in the first one it was 10 per cent); during that time, about 20,000 people died of typhus.”

**Dr. Henryk Fenigstein** By November 1941 15% of the Jews in the ghetto had typhus [i.e., ~70,000], which was only the first half of the outbreak Fenigstein *The Holocaust and I* p.xviii as cited in Roland (16) p.137.

**Dr. Tadeusz Radwański** Deputy special commissioner for epidemic control at the Ministry of Health From 1 July 1941 to 28 January 1943, I was the director of the department for epidemic control in the ghetto. Because of these orders, the typhus epidemic reached incredibly high levels. Before the closing of the Jewish district, only 140 cases of typhus had been recorded in the entire city of Warsaw. Once the ghetto was sealed, the number of sick continued to grow, not only in the Jewish quarter, but also on the Aryan side. By the end of spring 1942, around 80,000 cases of typhus had been recorded in the ghetto.

**Ryszard Zablotniak (27)** “Over 100,000 persons suffered from this disease. In all likelihood about 25,000 Jews died of typhus.”

**Ringelblum (29)** p.194-195 August 26 1941: “now, the middle of August, there are some six or seven thousand patients in [private] apartments, and about nine hundred in hospitals.” According to the official numbers August had 1805 cases. Which is 25% of the cases mentioned by Ringelblum.

#### **SM4 Typhus mortality and case-fatality rate**

Some key studies in the literature, even in recent years, have suggested the typhus epidemic in Warsaw Ghetto had little impact on population mortality (16, 17). Trunk (17), whose statistical analyses on epidemics in WG are perhaps the most influential of all, claimed that 3.6% of deaths in the ghetto were attributable to typhus. However, the widely received analysis appears erroneous on this point (Methods;SM4). Clearly, since Trunk (17) assumes there were only ~18,000 typhus cases in the first place (i.e., the “reported” number), it is obvious that his estimates of those dying from typhus must be relatively low, given there were ~100,000 or more deaths altogether.

(33)The following quotes from the literature indicate that typhus mortality as well as the typhus case-fatality rate, was roughly 20-25% over the main epidemic. Many of the authors listed were highly respected medical specialist or historians who were on location in the ghetto.

First note though that even when there were minimal cases of typhus in January to March 1941, before the outbreak was underway, Hagen examined 100 cadavers and found eight with typhus i.e., 8% of deaths (16). We know that when the typhus was reaching its maximum, in “August [1941] 5.6 thousand people died, including 3.5 thousand confirmed cases of typhus” (32) i.e., at least 62.5%. of deaths directly. But in addition to this a substantial proportion of the patients suffering from typhus died during convalescence, because of undernourishment, and were classified as dying from starvation. Thus typhus was also responsible for inducing a large number of deaths indirectly, and it seemed that one disease fed the other, as discussed in the main text. The combined effect of starvation and typhus must account for the large number of deaths we see in Fig.5.

**Professor Jakub Penson (20):** (Head of typhus epidemic ward, Czyste hospital) estimated a case-fatality-rate of 20% and also a general mortality rate as 20% of all deaths due to typhus. He wrote: “Another epidemic broke out in June 1941 and continued until June 1942. It ... swept all over the ghetto, infecting about 100,000 people. This epidemic was a lot more acute than the first one, with a death rate of 20 per cent (in the first one it was 10 per cent); during that time, about 20,000 people died of typhus.”

**Prof. Jakub Penson (20)** also writes that, “at the end of 1941 and the beginning of 1942, it reached 9,000 deaths monthly.” “In this way 25,000 people died in the second epidemic.” That is a case-fatality rate of 25-30%.

**Report of the Jewish Council (32).** Conclusions of Dr. Janczewska (2014):“In July and August 1941, the peak of typhoid fever illnesses and deaths passed. In August 5.6 thousand died people, including 3.5 thousand were confirmed cases of typhus.” That is, 63%. In Ringelblum Archiv, Underground Archive of the Warsaw Ghetto, Volume 12 Jewish Council in Warsaw (1939–1943) compiled by Marta Janczewska “Part I. The Jewish Council until July 22, 1942” p.136

**Ryszard Zablotniak (27) (1971)** writes that “[o]ver 100,000 persons suffered from this disease. In all likelihood about 25,000 Jews died of typhus,” with 85,000 deaths in total in 1941 and first half of 1942 i.e., typhus constituted directly to 29% of the deaths and a case-fatality rate of 25%.

**Yisrael Gutman (19) (1982)** In 1940, 10 percent infected with fever died; in 1941 it is estimated that 20% died. That is a case-fatality rate of 20% in the main epidemic.

**Dr. Mordechai Lenski (26)** (p.290) documents a general 15% case-fatality rate.

**Bernard Goldstein (28)** “By the early winter of 1941, shortly after the establishment of the ghetto, the death rate from typhoid, typhus, and dysentery had reached six to seven thousand every month. The wave of death swamped the doctors and undertakers. Even the bereaved were too overwhelmed by the magnitude of the calamity to express their sorrow. The dead were dumped naked - for clothes were valuable - into the streets. Every morning wagons drove through the ghetto to pick up the bodies and take them to the cemetery, where they were buried in mass graves.”

**Typhus mortality in hospitals: Trunk (17)**, “In June 1941 out of 959 patients, 176 died (18 percent); during the first half of July, out of 782 patients, 153 died (17 percent). It should also be noted that many of the patients discharged from the hospital died after leaving it. Some were discharged while still in serious condition... Others fell victims to death from starvation on arriving at home.” The actual percentage of deaths among the hospital patients was, therefore, undoubtedly much higher.” Trunk

**Typhus mortality in hospitals: Trunk (17)**, “The number of deaths in the Warsaw [ghetto] hospitals in 1941 amounted to 4,920. Of these 3,171 died of contagious diseases, including the 1,580 cases of spotted typhus. In January 1941 the number of deaths from typhus ..... Altogether, the number of deaths from spotted typhus during the whole year amounted to nearly one-third of all the deaths and nearly half of all deaths from contagious diseases.”

### **SM5. The under-reporting of the official registered death data**

Here we provide reports from the literature corroborating the “registered death” data (i.e., reported deaths, dashed blue line) in Figure S1 appears to be considerably underestimating the true monthly mortality rates. And that the food ration card can give valuable insights. We use the food ration card from Ref.(23) as we are unclear of the sources or validity of data in other reports and plot the month to month changes in Fig.2. Ref.(23) provides the registered death data also plotted in Figure S1. Caveats in using these cards are then discussed.

#### **a) Corroboration from the literature of extensive monthly deaths, far larger than official numbers:**

In Figure S1 above, all months have fewer than 5,000 reported deaths except for three months which are less than 5,500 deaths. These numbers from the official monthly registered mortality reports contradict the literature, as we show below. Many of the authors listed were highly respected medical specialists or historians who were on location in the ghetto.

**Trunk (17)** (Table VIII, p.39) cites a document from the Ringelblum Archives with figures showing that there were 7,070 deaths in July 1941.

**Ringelblum** writes that the mortality reached 5000-7000 in some months (see (30) p.381).

**Ringelblum (29)** p.199 11'th July. “The fate of the refugees is horrifying; they are dying at a fearful rate. In the refugee centre ... 35% of them died in five months time.”

**Goldstein (28)** (2005; p.63) writes that in the early winter of 1941, shortly after the establishment of the ghetto, ... the death rate had reached six to seven thousand every month.”

**Prof. Jakub Penson (20)** who writes that, “at the end of 1941 and the beginning of 1942, it reached 9,000 deaths monthly.”

In her diary, **Mary Berg (22)** writes of sometimes even 15,000 deaths a month, matching a possibly outlying point in Fig.2b obtained from the food ration card.

**Trunk (17)** p.37: “In the children’s homes the mortality rate was also very high. As an example we shall take the largest such home on 127 Leszno Street (on November 25, 1940 it was moved to 39 Dzielna Street). There were 47 deaths in this home between July 4 and August 15 1940, out of 470 children i.e., 10% [Bulletin of the Judenrat #15]. In 1941 in the ghetto, the rate of mortality in the same children’s home rose still higher: for an average of 600 children there were 178 deaths in May and 155 in June]. At this rate, if no new children were added to the home, all the children would have died out within three or four months!” This is 55% death rate in two months.

**Trunk (17)** p.37: “Thus, the large center at 15 Stawski Street had 207 deaths during the months March to July of 1941 for a population of 673 i.e., 36.2% “

**b) Caveats in using cards as a proxy for deaths:** In Figure S1, the food ration cards indicate a loss of 15,000 cards in the month of September. Based on the reports from the literature, many of which are cited here, this does seem unusually large and we would suspect this number exceeds the true number dead for September.

A close study of the population flows in Berenstein and Rutkowski (1958) indicates there may be possibilities for several sources of bias. In their Table on p.88, we know that for the year 1941 there were 10,6000 (6,000+4,600) residents who were either sent to labour camps or escaped. However, there were also 2,000 immigrants in August 1941 from Ciechanów countering this loss. That is, a nett loss of 8,000 residents that were not in fact due to death, but we know few other details. This might explain a positive bias in the food card proxy for death rates.

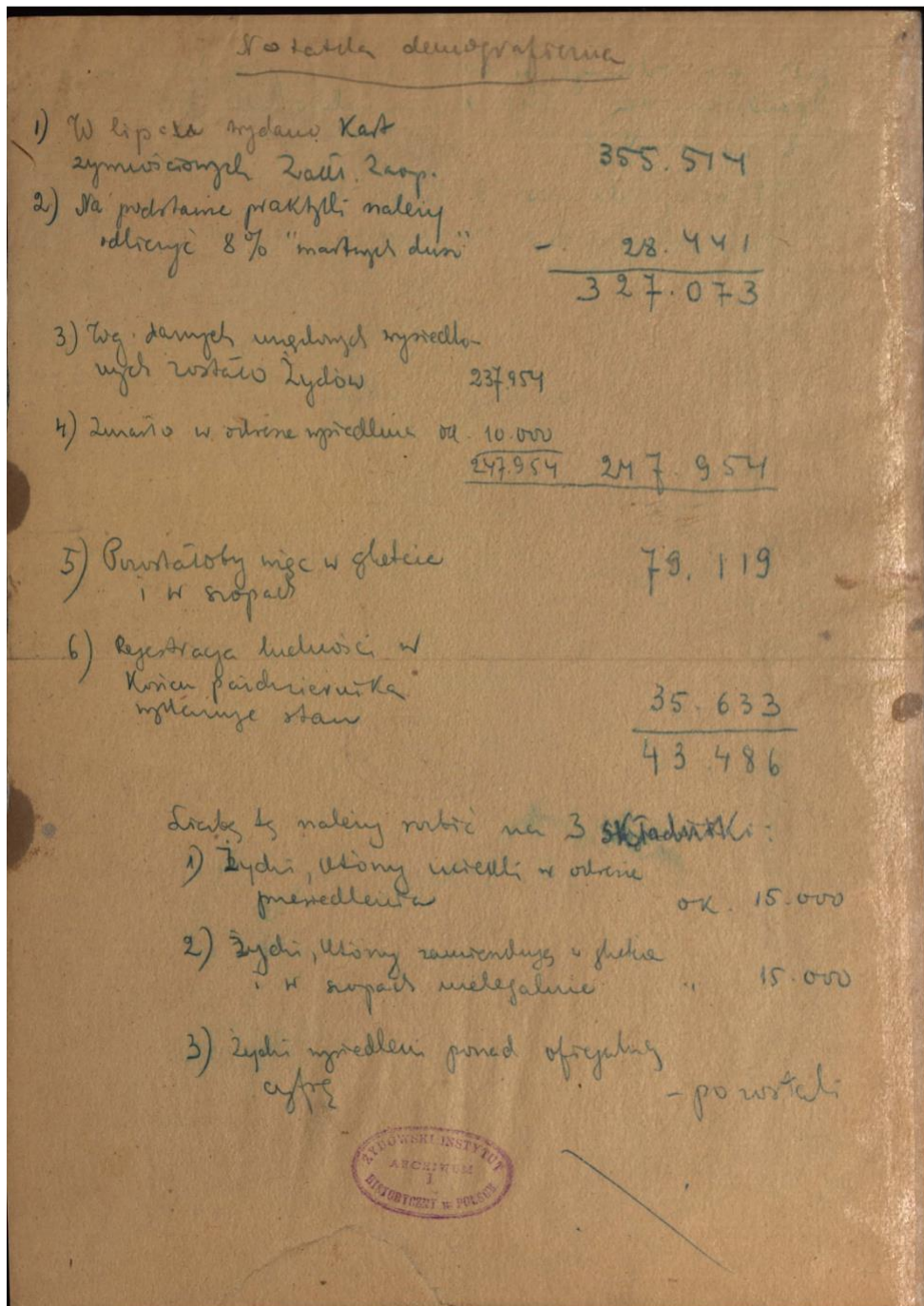
On the other hand, if this loss of 8,000 was spread uniformly over the months of May to September, it would not alter the trend seen in the monthly *differences* in food card numbers. If it were not spread uniformly, in the worst case the numbers may over-represent some months but not enough to affect the general argument. Moreover, there was a trade in the ration cards of those people who had left the ghetto, and these cards might have remained “in the system” for a period.

Another possibility is suggested by Engelking & Leociak(18) who suggest, according to a newspaper source, that the fall in ration cards may be due to stricter community control on exploiters and “double dippers.” But again, if the control was uniformly exerted, it should not change the trend in monthly differences. Second, according to Berenstein and Rutkowski (23) we can be fairly confident that there were 445,000 or more people in the ghetto by March 1941, and we can use this as a reference point. At this point the ration card numbers were believed to be the actual numbers. So even if the ration cards began to fall after March 1941 because of extra community controls, and the numbers were dropping because of both this and deaths, where did these people disappear to if not deaths?

Finally, if people made use of the cards of “dead souls” then this would tend to increase the ration card numbers rather than decrease them as is shown in the calculations in SM7. The month to month differences in numbers should not be affected.

The Judenrat Statistical Division used the counts of the Food Ration Cards as a mechanism for censusing the population, and we follow their guidance on this, and believe that they should give an indication of population trends.

**SM6 Maths of Food Ration Cards**



**Fig. S1 Maths of Food Ration Cards.** Anonymous note found in one of the cases of the Ringelblum Oneg Shabat Archive (reproduced by courtesy of the Jewish Historical Institute Warsaw, signature Ring II/195) dug out from under ruins of Warsaw Ghetto. Presumably the note shows calculations of the Statistical Division with 355,514 being the number of food ration cards estimated in July 1942 on the eve of deportations to Treblinka. From this one deducts 8% of cards (28,441) belonging to “dead souls” and still in use. This leaves 327,073 cards. Thus on the eve of the deportations to Treblinka these calculations suggest there were 327,073 residents in the ghetto. The remaining data concerns the statistics of victims (237,954) deported to Treblinka, those who died during the deportation (10,000), and those remaining in the ghetto (79,119). We thank Professor Jan Grabowski for directing us to this document.



## SM7 Model description

### Model 1

In the Methods we outlined the vector-host SEIRL model with disease induced mortality having five classes, Susceptible, Exposed, Infective, Recovered, and Lice. Given the relatively fast dynamics of the vector (lice), we showed that at quasi-equilibrium ( $\frac{dL}{dt} \approx 0$ ) the SEIRL model reverts to a standard SEIR model of the following form:

$$\begin{aligned}\frac{dS}{dt} &= A(t) - \frac{\beta(t)}{N(t)}SI - \mu(t)S \\ \frac{dE}{dt} &= \frac{\beta(t)}{N(t)}SI - (\sigma + \mu(t))E \\ \frac{dI}{dt} &= \sigma E - \frac{1}{1-m}(\gamma + \mu(t))I \\ \frac{dR}{dt} &= \gamma I - \mu(t)R\end{aligned}$$

As stated in the Methods, the total but changing population size at time  $t$  is  $N(t) = S(t) + E(t) + I(t) + R(t)$ . The parameter  $\sigma$  denotes the rate of progression from the exposed class to infectious class, and thus the mean latent or incubation period of the disease is  $\sigma^{-1} = 14$  days.

The parameter  $\gamma$  denotes the rate of loss of infectiousness or rate of progression from the infective to the recovered class. Hence the mean infectious period is  $\gamma^{-1} = 28$  days. These parameters correspond to estimates from the literature (Raoult and Roux 1999). Adler (1982), for example, documents this (in addition to other references in main text): ‘One had only to brush against a person infested with lice to discover these dangerous vermin in the evening while examining each piece of clothing; then there was the terrible wait in fear and trepidation for the fourteenth day to see whether the temperature would rise sharply.’

Setting  $m = 0.25$  gives an infection mortality rate of  $m/(1+m) = 0.2 = 20$  percent as justified in SI4. The parameter  $\mu(t)$  is the rate of removal from the full population due to death (other than through typhus infection).  $A(t)$  denotes population displacements into the ghetto (eg refugees). We set  $A(t)$  and  $\mu(t)$ , such that the model simulated population matched the historical records (see Figure below).

We use a frequency-dependent vector-host transmission function which accounts for the term  $\beta(t)/N(t)SI$  in the SEIR equations above.

We note that for  $\beta$  and  $\mu$  constant, then:

$$\mathcal{R}_0 = \frac{\beta\sigma}{(\sigma + \mu)(\gamma + \mu)}.$$

A key goal is to fit the transmission rate  $\beta(t)$  flexibly so that it can change as a function of time. To achieve this,  $\beta(t)$  is formulated as a cubic spline function in the exponential form with nodes uniformly distributed over the whole period. When  $\gamma \gg \mu$  and  $\gamma \gg \mu$ , we have  $\mathcal{R}_0 \approx \frac{\beta(t)}{\gamma}$ .

The observed typhus cases (Fig.1) are modeled as a Partially Observed Markov Process (POMP) making use of the Iterated Filtering and plug-and-play likelihood-based inference frameworks to fit the data (King et al. 2015; He et al. 2010). These are modern state-of-the-art statistical methodologies developed for fitting complex epidemiological datasets. The Maximum Likelihood Estimate (MLE) for model parameters is calculated using the Cran R package ‘‘POMP’’ (King et al. 2015). The simulations made use of the Euler-multinomial integration method with the time-step fixed to one day. When simulating the model, we included both demographic noise and

measuremental noise. The former follows a multinomial distribution, and the latter follows a negative binomial distribution with a dispersion parameter  $\tau$ . In simple terms, the model estimates the true number of cases but then adds a reporting layer to simulate observed data (as discussed further below).

**Table S1** – Historical record of population size

year	month	pop
1939	10	359827
1940	3	380000
1940	12	392911
1941	1	400000
1941	3	460000
1941	4	450000
1941	5	442000
1941	6	439000
1941	7	431900
1941	8	420116
1941	9	404300
1942	1	377000
1942	7	355000

Data taken from Berenstein and Rutkowski (1958) and Trunk (1953).

The initial susceptible proportion is assumed to be  $S(0)/N(0) = 0.99$  and initial exposed and infectious cases are assumed to be 1 or 2 cases. At the end of 1940, the start of the second wave, some exposed and infectious cases are introduced (180 infectious cases) to initiate the huge epidemic that follows and to mimic the impact of 66,000 immigrants arriving.

We let  $\rho$  denote the reporting ratio. The monthly reported cases are

$$Z_i = \int_{\Delta t} \rho \sigma E dt$$

where  $\Delta t$  is a month.

We denote  $C_i$  as the reported cases in  $i$ 'th month, which is assumed to follow a Negative-Binomial distribution,

$$C_i \sim \text{NegBinom}(\text{mean} = Z_i, \text{variance} = Z_i(1 + \tau Z_i))$$

where  $\tau$  controls the over-dispersion. When  $\tau = 0$ , this becomes a Poisson distribution. The logic behind this assumption is that the rate of the Poisson distribution is typically a random variable (e.g. Gamma random variable) rather than a constant in many settings. When the rate of the Poisson distribution is a Gamma random variable, it becomes a Negative Binomial distribution.

We define the log likelihood function as

$$l(\theta) = \sum_{i=1}^n \log f(C_i | C_{1:i-1}, \theta)$$

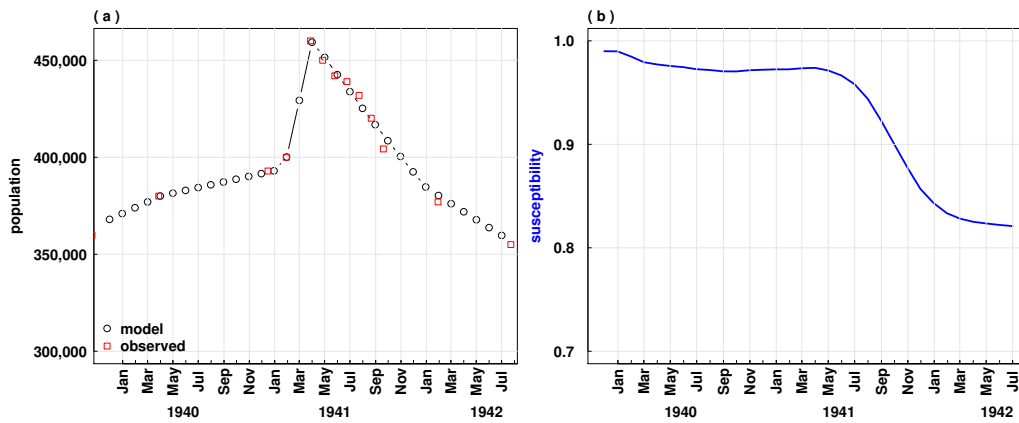
where  $\theta$  is the set of unknown parameters and  $f(C_i | C_{1:i-1}, \theta)$  are the conditional densities for  $C_i$  given  $C_{1:i-1}$ . Sequential Monte Carlo (i.e., particle filtering) is used to calculate these conditional densities. We used the iterated filtering algorithm implemented in the Partially Observed Markov

Process (POMP) R package to estimate  $\theta$  and calculate  $l(\theta)$ . More details are given in [https://en.wikipedia.org/wiki/Iterated\\_filtering](https://en.wikipedia.org/wiki/Iterated_filtering).

As mentioned, on Dec 31, 1940, a proportion ( $\rho_3$ ) of population became infected (or introduced). We noticed through fitting experiments that  $\rho$  and  $\rho_3$  are negatively correlated,

## Results

We fixed  $n_\beta = 5$  nodes in the cubic spine, based on preliminary testing of the optimal  $n_\beta$ . As mentioned,  $\sigma^{-1} = 14$  days, and  $\gamma^{-1} = 28$  days. The total population numbers (left) and susceptibility over time (right) over time for a typical simulation are plotted in the figures below. The population numbers from input data appear as red squares.



**Fig. S2 – Population and susceptibility over time.**

The following Table summarises the distribution of the total number of infected (reported and unreported) cases for the major wave only over the epidemic for 1000 independent runs.

**Table S2 – Simulated cases for the major wave under two scenarios.**

	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
actual	44664	64596	71943	72202	79251	113442
hypothetical	135680	183576	195819	196728	210103	248759

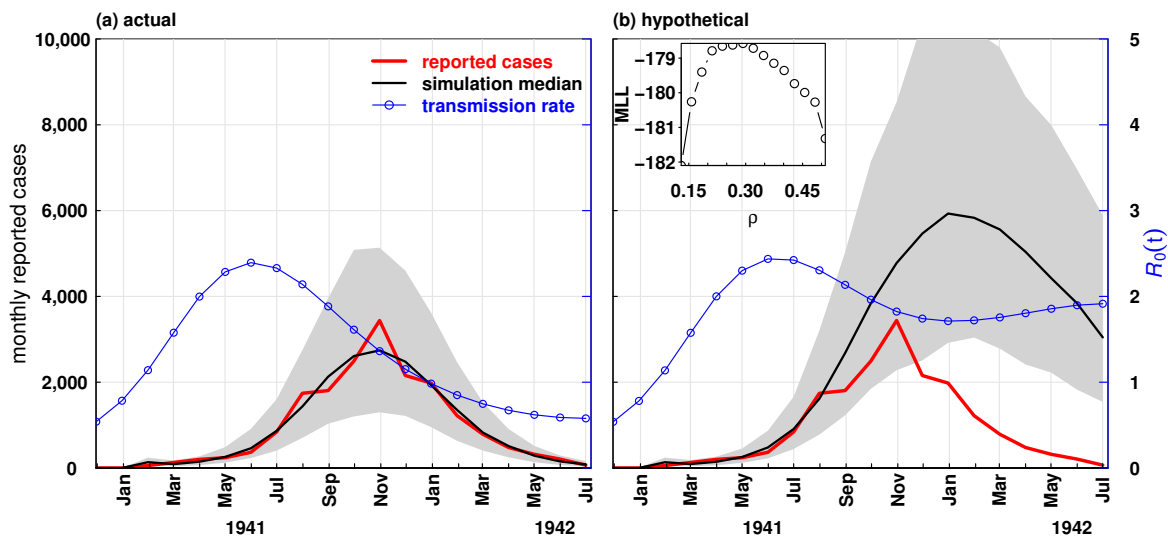
We began with  $n_\beta = 5$ , nodes in the transmission rate ( $\beta(t)$ ). In the “actual” scenario, the last two nodes the value of ‘log.beta3’ and ‘log.beta4’ decreases, ie.  $\mathcal{R}_0(t)$  decreased after May-June 1941. In the hypothetical scenario, where we assumed  $R_0$  is approximately constant after July 1941, we set

Hypothetical scenario:

```
y[i, 'log.beta3'] <- 1.0 * y[i, 'log.beta2']
y[i, 'log.beta4'] <- 1.02 * y[i, 'log.beta2']
```

such that the  $\beta(t)$  shows a more or less non-decreasing trend. Since we use an (exponential) cubic-spline function, it is hard to keep  $\beta(t)$  an exact constant, because the spline generates fluctuations. The outcome is shown in Fig.4 main text but repeated for convenience with Fig.S3. Other parameters and estimates are in the following.

	loglik	loglik.sd	nfail.max	nfail.min	sigma	gamma	lambda	mu	
[1,]	-178.5756	0.273576	0	0	26.07143	13.03571	0	0.2	
[2,]	-178.5756	0.273576	0	0	26.07143	13.03571	0	0.2	
	pop	nbeta	log.beta	log.beta1	log.beta2	log.beta3	log.beta4	log.beta5	
[1,]	365000	5	4.679734	1.366666	3.176625	2.856769	2.043029	3.456605	
[2,]	365000	5	4.679734	1.366666	3.176625	3.176625	3.240158	3.456605	
	log.beta6	log.beta7	log.beta8	log.beta9	log.beta10	log.beta11	log.beta12		
[1,]	3.329809	3.167993	3.796959	4.382058	5.899897	5.899897	5.721251		
[2,]	3.329809	3.167993	3.796959	4.382058	5.899897	5.899897	5.721251		
	log.beta13	log.beta14	log.beta15	log.beta16	log.beta17	log.beta18			
[1,]	5.465442	5.28135	5.181875	5.434717	4.480801	5.038071			
[2,]	5.465442	5.28135	5.181875	5.434717	4.480801	5.038071			
	log.beta19	tau	rho	rho2	rho3	S.0	E.0	I.0	R.0
[1,]	5.036732	0.098987	0.292857	0.1	0.000444	0.99	3e-06	3e-06	0.009994
[2,]	5.036732	0.098987	0.292857	0.1	0.000444	0.99	3e-06	3e-06	0.009994
	susc.ini	infe.ini	t0	aicc	mean.beta	R0			
[1,]	0.9	10	0	379.412	22.67748	1.7			
[2,]	0.9	10	0	379.412	26.67244	2			



**Fig. S3 – Fitting an SEIR model to typhus cases (two waves).** a) Fitting “actual” reported cases b) Simulating the “hypothetical” case in which  $R_0$  is roughly constant in the last months. The latter simulates a natural scenario in the absence of anti-epidemic activities. The inset panel shows the maximum log-likelihood (MLL) profile of  $\rho$ . The maximum likelihood estimate MLE is  $\rho = 0.262$  and the 95% confidence interval (CI) of  $\rho$  (0.158, 0.496). The 95% CI is the range where the MLL is within the maximum minus  $0.5\chi_{0.95,1}^2 = 1.92$ .

### Choice of $n_\beta$ on the two waves

In Fig.S4, we demonstrate why we chose  $n_\beta = 5$  in our analyses. This particular value was found by finding the best fitting value (see Inset) given all other parameters fixed to realistic values.

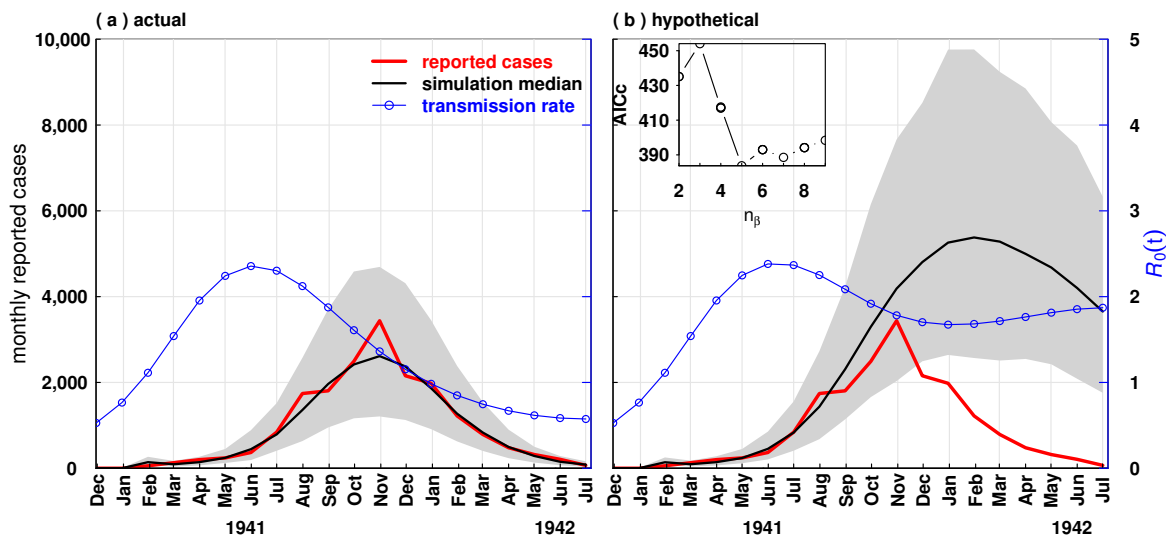


Fig. S4 – Fitting an SEIR model to typhus cases (two waves). Inset panel showed that  $n_{\beta} = 5$  attains the smallest AICc.

### SM7b Parameter estimation and exploring

#### Exploring parameter values $\sigma$ , $\gamma$ and $\rho$ :

In all analyses in this paper, we fixed latent or incubation period at  $1/\sigma = 14$  days given that it has been mentioned repeatedly in the historical literature of Warsaw Ghetto (see eg., SM1 (Adler)). A reasonable estimate from what is known of the infectious period is  $1/\gamma = 28$  days. That it can be an extended time can be seen from passages as that found in Snyder (1947): “It is important to recall that the typhus infection in lice establishes itself after an incubation period, just as it does in man. The period of maximum danger of spread of typhus from a louse-infested patient to other persons extends into the first two weeks of convalescence when the victim becomes ambulatory. The hazard in handling corpses within a few hours after death is obvious.” Or from Bechah et al. (2008): “Infection through aerosols of faecal dust has been reported and provides the main risk of epidemic typhus contamination for physicians. Indeed, *R. prowazekii* can remain viable for 100 days in lice faeces.” The period to convalescence can be quite lengthy as pointed out in Ciesielska (2015): “In a typical course of the disease, the rash disappears after 5-6 days, turning into bran skin exfoliation at week 2 or 3 of the disease, sometimes preceded by so-called skin readiness for exfoliation. Hemorrhagic eruptions and bruising disappear later, only after 10-14 days of illness.”

We now proceed to verify that the infectious period is  $1/\gamma = 28$  days is indeed reasonable.

**Additional test 1.** We begin by fitting the second wave and simultaneously estimating both the reporting rate and  $\gamma$ . The results are given in the Figure S5 and the associated Table below. The overall best result is with  $n_{\beta}=2$ , (i.e., constant beta) as seen from the inset.

The estimates for the parameters so found give extremely low reporting ratios  $\rho$  (5-9%) that are unrealistic (see main text). The estimated infectious period are long  $1/\gamma = 26-37$  days.

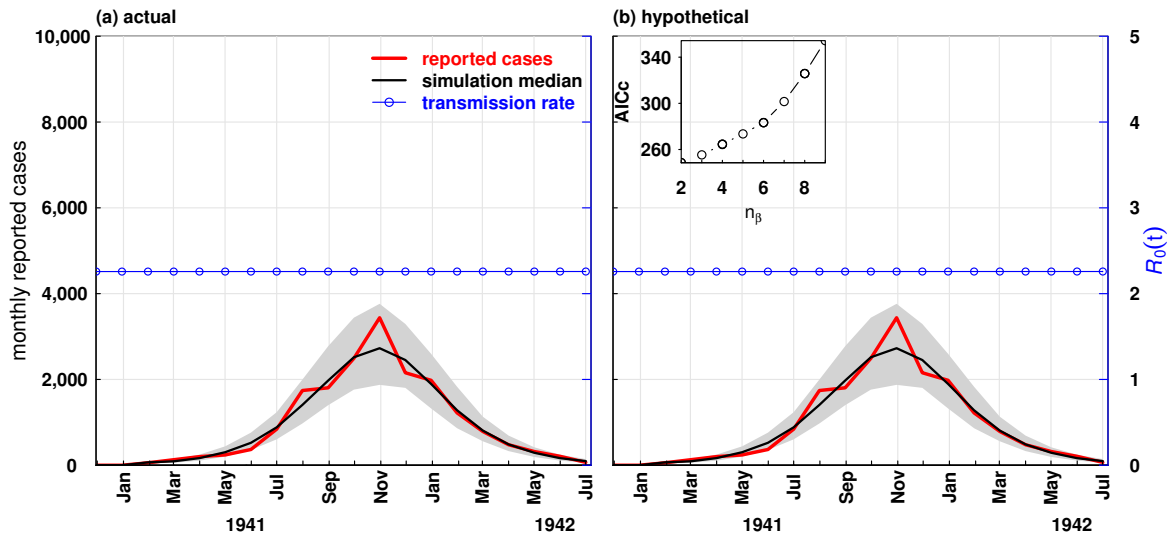


Fig. S5 – Fitting an SEIR model to typhus cases (two waves) and simultaneously estimating both the reporting rate and  $\gamma$ . Inset panel showed that  $n_\beta = 2$  attains the smallest AICc.

$n_\beta$	$\rho$	$1/\gamma$	$n_\beta$	$\rho$	$1/\gamma$
2	0.06184638	26.53967	6	0.06338225	30.18195
2	0.05969612	30.32556	6	0.06150539	47.82063
2	0.05909456	31.52845	6	0.06856682	38.12394
2	0.05770588	34.37639	6	0.05201700	57.06015
3	0.06277339	37.33442	6	0.05729979	52.52728
3	0.07348999	37.93717	7	0.06469663	31.65357
4	0.07732673	34.97222	7	0.06545347	33.85535
4	0.08254114	32.44651	8	0.06293641	39.19181
4	0.09309374	32.00618	8	0.04996599	88.01055
4	0.09259728	30.02939	8	0.05216815	78.55982
4	0.08794224	31.28654	8	0.06621162	41.32385
4	0.10538832	27.86694	8	0.05421956	48.68633
5	0.06047626	53.23725	8	0.06038673	50.74655
5	0.06873125	48.87142	9	0.05980729	41.65555
6	0.06465285	49.85577	9	0.05883102	47.05755

The main result here is that we cannot fit the data with realistic parameters after examining a wide range of conditions. And certainly not with constant beta or equivalently constant  $\mathcal{R}_0$ .

**Additional test 2** We now fix the reporting rate to  $\rho=25\%$ , which we believe to be realistic (see main text), and estimate  $\gamma$ , with  $n_\beta$  varying from 2 to 9. We see that  $n_\beta=2$  (scenario of constant beta), gives a very poor fit to the one wave (based on minimizing the AICc) as compared to other values of  $n_\beta$ . The best result (with the smallest AICc) is shown in (a) when we fix  $n_\beta=2$ . Clearly this graph is a terrible fit to the data, (b) The overall best result is obtained when  $n_\beta=3$ . The estimated  $\gamma$  ( $1/\gamma$  in days) in the table below and inset, occurs when  $n_\beta=3$ . The point estimate is  $1/\gamma = 35-37$  days and the graph Fig. S6 gives a nice fit.

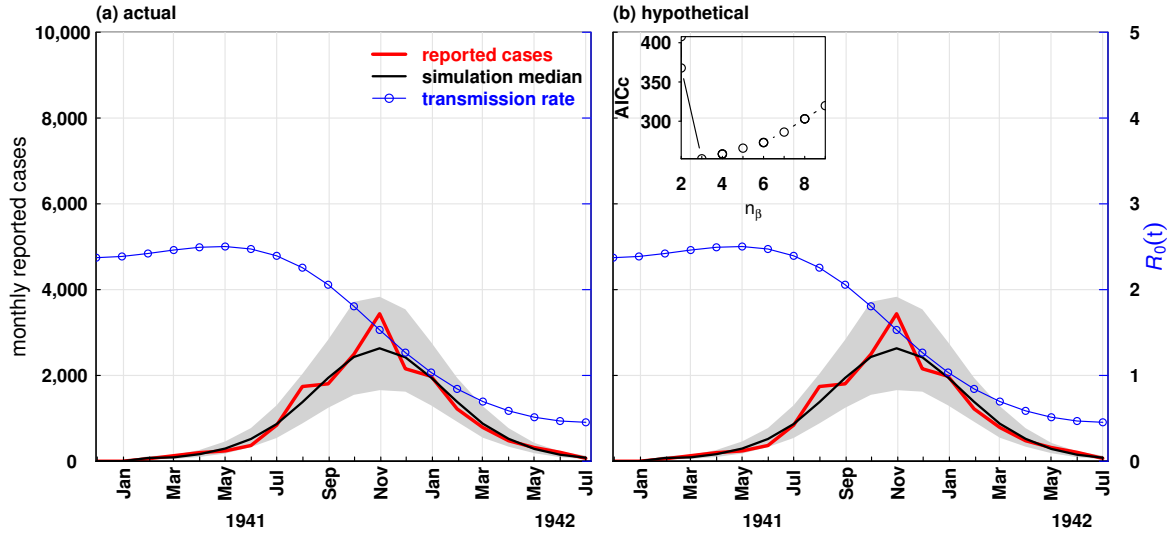


Fig. S6 – Fitting an SEIR model to typhus cases (two waves) and estimating  $\gamma$  while fixing  $\rho = 25\%$ . Inset panel showed that  $n_\beta = 3$  attains the smallest AICc.

$n_\beta$	$\rho$	$1/\gamma$	$n_\beta$	$\rho$	$1/\gamma$
2	0.25	3.364533	6	0.25	20.943313
2	0.25	3.797988	6	0.25	23.740654
2	0.25	4.445179	6	0.25	28.722364
2	0.25	4.524587	6	0.25	25.079699
3	0.25	37.937523	6	0.25	26.683352
3	0.25	35.047778	7	0.25	18.627172
4	0.25	24.752449	7	0.25	21.849003
4	0.25	23.150640	8	0.25	25.688992
4	0.25	22.716135	8	0.25	21.557641
4	0.25	27.765804	8	0.25	27.366969
4	0.25	22.218937	8	0.25	23.297960
4	0.25	21.823898	8	0.25	24.710986
5	0.25	36.852162	8	0.25	28.015218
5	0.25	31.834336	9	0.25	24.903118
6	0.25	28.705139	9	0.25	19.583486

### Exploring scenarios of different anti-epidemic:

In Figure 4 of the main text we simulated the epidemic curve that would result in the hypothetical absence of anti-epidemic activities. This was achieved by preventing  $\mathcal{R}_0$  from dropping below a base value of  $\mathcal{R}_0=2$  corresponding to what the simulation indicated was the capacity of typhus i.e., close to the peak value of  $\mathcal{R}_0$  it was potentially capable of maintaining. The underlying assumption was that any increase in anti-epidemic activities in the ghetto corresponds to a reduction in  $\mathcal{R}_0$ . Here we check what would occur if  $\mathcal{R}_0$  is prevented from dropping below  $\mathcal{R}_0=1.25$ . This is because we know that  $\mathcal{R}_0=1$  corresponds to a scenario in which a virus or bacteria would be unable to generate an epidemic in a totally susceptible population, and  $\mathcal{R}_0=1.25$  might correspond to the minimal possible value we would expect for a minimal type of typhus outbreak, certainly far smaller than the explosive one in the Warsaw Ghetto. With  $\mathcal{R}_0=1.25$ , the simulation is plotted in Fig.S7.

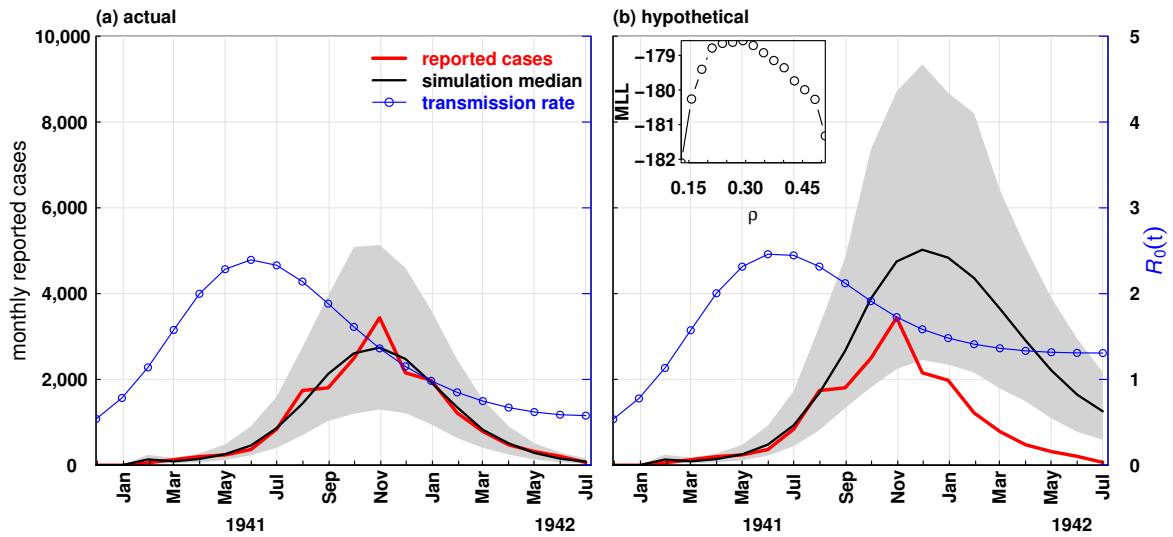


Fig. S7 – Fitting an SEIR model to typhus cases (two waves) and keeping  $\mathcal{R}_0 > 1.25$ .

We see that even for this value of  $\mathcal{R}_0=1.25$  which we consider unrepresentatively low, the same conclusions hold. The hypothetical epidemic is typically more than 2 times intense and doesn't begin to significantly decline until February 1942.



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