Extraordinary curtailment of massive typhus epidemic in the Warsaw Ghetto

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The highly dependent interplay of disease, famine, war, and society is examined based on an extreme period during World War II. Using mathematical modeling, we reassess events during the Holocaust that led to the liquidation of the Warsaw Ghetto (1941–1942), with the eventual goal of deliberately killing ~450,000, mostly Jewish residents, many through widespread starvation and a large-scale typhus epidemic. The Nazis justified genocide supposedly to control the spread of disease. This exemplifies humanity’s ability to turn upon itself, based on racially guided epidemiological principles, merely because of the appearance of a bacterium. Deadly disease and starvation dynamics are explored using modeling and the maths of food ration cards. Strangely, the epidemic was curtailed and was brought to a sudden halt before winter, when typhus normally accelerates. A far more massive epidemic outbreak was prevented through the antiepidemic efforts by the often considered incompetent and corrupt ghetto leadership and the Herculean efforts of ghetto doctors.

INTRODUCTION

Epidemics, pandemics, and infectious diseases have played major roles in shaping the trajectory of human history (1–4). Their ability to rapidly invade, infect, and then ravage human populations has, not a few times, left millions of dead victims in their wake. In times of war and famine, the presence of infectious diseases magnifies existing hazards, often resulting in catastrophes of extreme proportions. Thus, the Spanish influenza pandemic at the end of World War I (WWI) killed more than 50 million people; the introduction of smallpox to the Americas almost eliminated its indigenous peoples entirely, while tuberculosis, the leading cause of death from infection globally, is up to 20-fold more transmissible during wars, conflicts, and population displacements (5). Despite the dangers of these massive public health catastrophes, their social and political dimensions are often poorly explored by the scientific community and, sometimes, even misrepresented. Thus, famines, which many times go hand in hand with disease, are often erroneously viewed as natural or climate-induced phenomena when, in most recent cases, they should more accurately be viewed as man-made weapons of war (6).

Only in recent years has there been serious recognition of the importance of both quantifying datasets of wartime events and working with mathematical models to help understand more deeply underlying interrelationships (7, 8). Here, using mathematical modeling, we study the impacts of disease and starvation in the Warsaw Ghetto during the Holocaust and provide a new reconstruction of events that occurred there.

The Holocaust (9) refers to the Nazi-German annihilation of some 6 million members of European Jewry during WWII (1939–1945). It is not widely known that this genocide was triggered in no small part by alleged public health concerns and Germany’s obsession with disease and fear of epidemics. More specifically, there was a fanatical fear of typhus spreading to the German people and its army, given its previous impact as the cause of 5 million deaths after WWI. This was the pretense given by the Germans for the relocation of Jewish victims en masse into isolated closed ghettos and camps in wartime Europe (10–14). However, the same fear of epidemics was also a pretext used by the Germans as justification to liquidate ghettos, including their residents.

Initially, the Germans did not even want to establish ghettos because these were considered to be nourishing ground for infections (10, 12). However, the German discourse on hygiene was very much influenced by the anti-Semitic idea of Jews being notorious bearers of diseases. In the Nazis’ ideology, this evolved into Jews being the actual disease, so epidemics were to be naturally expected and dealt with, which in the end meant annihilating the Jews (13). Of course, hygiene was only one pretext driving Nazi anti-Semitism; but it played an important part in propaganda efforts to justify anti-Jewish hatred and politics in this period before the industrial killings.

By October 1941, when an epidemic was raging in the Warsaw Ghetto, Jost Walbaum, the Chief Health Officer of the General Government (Occupied Poland) famously made the accusation: “The Jews are overwhelmingly the carriers and disseminators of typhus infection. ... There are only two ways [to solve this]. We sentence the Jews in the ghetto to death by hunger or we shoot them... We have one and only one responsibility, that the German people are not infected and endangered by these parasites. For that, any means must be right” (14). These words were followed by applause and clapping from 100 attendees, mostly medical doctors. Previous to this, SS General Heydrich, the main architect of the Final Solution, specifically requested that the chief SS physician initiate an epidemic in Warsaw Ghetto to exterminate the Jews (15). In addition, the highest German administrative authority in the General Government, Governor General Hans Frank, alleged in 1943 that the genocidal murder of 3 million Jews in Poland “was unavoidable for reasons of public health” (16, 17). These are obvious cases of disease being used as a weapon of war and pretexts for genocide. Today, more than ever, society needs to grasp how a virus or bacterium can create utter havoc, dragging humankind to this terminal point of evil.

Figure 1 makes clear how several major events in the history of the Warsaw Ghetto over the period 1939–1942 synchronized perfectly...
with the epidemiological dynamics to help achieve the Nazis’ aim of exterminating the ghettos and its residents. The figure presents a time series of the monthly number of new reported typhus cases over two typhus epidemics that broke out in the ghetto. The first smaller outbreak initiated in September 1939 during Nazi Germany’s siege of Warsaw just after their aerial bombing severely damaged Warsaw’s sewage system and contaminated the water supply. Simultaneously, large numbers of refugees and exiles streamed into Warsaw, increasing the likelihood of an infection invading from the outside, which is exactly what happened (11, 18). The first smaller typhus epidemic over the winter ended naturally during the summer season (~August 1940).

The Germans feared another outbreak of typhus and thus created a Seuchensperrgebiet—literally a restricted disease area, which later would become the ghetto. On 5 October 1940, Jews were forbidden to leave this territory. After 15 November 1940, a brick wall was built that surrounded the area. It was 3 m tall and 18 km long—the “epidemic wall,” and all of Warsaw’s then ~400,000 Jewish population were confined to remain within (10, 13). With barbed wire at the top, only the most agile residents could escape, mostly through small gaps in the wall, burrowing underground or via the sewers. The sealing was an extreme form of quarantine. The Germans prohibited the internees from leaving the ghetto for fear of the possibility that typhus might spread to the external Warsaw population and especially to the occupation personnel. At a certain point, the death penalty was enacted on escapees.

Our main interest is trying to quantify what happened in the so-called “Period of Indirect Extermination” (19) from the time the Warsaw Ghetto was sealed off, until the liquidation of the ghetto began on 22 July 1942 when more than 250,000 residents were quickly deported by train to their deaths in the gas chambers of the Treblinka extermination camp. To help untangle what occurred, it is important to understand that the population dynamics over this period was largely controlled in two phases.

**Phase 1: Starvation**

Additional food supplies beyond the small amount the Jewish Council was able to buy were deliberately blocked from entering the ghetto until May 1941, but even later on, the official ration provided by the authorities often was no higher than 200 calories per day (18). Predictably, within a few months, residents began starving to death, leaving the impression of an epidemic of deaths. Mortality rates rapidly increased to large levels with 4000 to 5000 registered deaths per month, as shown in Fig. 2 (blue line). There is reasonable evidence demonstrating that the registered number of deaths is substantially less than the true number, which might be closer to that indicated by the red line in Fig. 2, even exceeding 9000 deaths per month as claimed by Penson (20), the head of the typhus ward of the Czyste Hospital (see section SM5). As described by ghetto commissar Heinz Auerswald: “A quantum leap in deaths for May of this year [1941] showed that the food shortage had already grown into a famine” (11).

The situation progressively worsened until human corpses covered with newspapers were a common sight on the streets (21, 22).

**Phase 2: Typhus epidemic**

The out-of-control rampant spread of typhus followed quickly on the heels of the horrific starvation phase. Typhus is a bacterium (*Rickettsia prowazekii*) spread by its vector, the human body louse (*Pediculus humanus humanus*). The latter multiply prolifically under conditions of poor hygiene, filth, overcrowding, and cold weather—exactly the conditions of the ghetto (section SM2) (23, 24). Given its population density and overall conditions, little wonder that the ghetto was commonly referred to as an “incubator” of disease and “breeder of epidemics.” Ludwik Hirszfeld, the eminent bacteriologist and Nobel Prize nominee living in the ghetto, had no doubts: “In the case of WW2, typhus was created by the Germans, precipitated by lack of food, soap, and water, and then—when one concentrates 400,000 wretches in one district, takes everything away from them, and gives them nothing, then one creates typhus. In this war, typhus is the work of the Germans” (16).

Lice thrive in large numbers on their human hosts, feed daily on their blood, and have rapid spatial spread, which allowed them to infest the entire cramped ghetto. For those infected, typhus typically has a ~14-day incubation period and results in a high fever, headache, muscle pain, nausea, chills, and extensive body rashes. As the disease progresses, increasing weakness and delirium develop, and in some cases, loss of consciousness. Death can result within a few days for the worst cases. Accounts may be found of sufferers jumping through windows in agony to their death. When the human host dies, the vector simply moves to another victim.

Quantifying what actually occurred in these two phases is the goal of this paper. Referring again to Fig. 1, the official number of monthly reported new typhus cases for both epidemic waves sums to a total of 20,160 reported cases. Yet, according to the scattered reports of leading epidemiologists of the ghetto, there is reasonable consensus that a total of 80,000 to 110,000 residents were infected (19, 20, 25, 26). This major discrepancy can be seen from referring to many of the reports we have collected and placed in section SM3 (see Supplementary Materials). The time series data in Fig. 1 thus indicate a low reporting rate of ~20 to 25%. Many infected residents preferred not to report their illness given the potentially horrific
punitive actions that might eventuate as a consequence. The major study of Trunk (17) entitled “Epidemics and Mortality in the Warsaw Ghetto, 1939–1942” is the definitive statistical reference to what occurred in this period. Yet, Trunk bases most of his conclusions on the very low numbers seen in the official registered data, which results in a misleading representation of what actually took place. We will be examining these issues in depth as the paper proceeds and begin with a more detailed discussion and analysis of the epidemic dynamics.

RESULTS

Forest fire dynamics

The ghetto’s population numbers fluctuated but, at times, contained at least 450,000 people in an area of 3.4 km². This density is some 5- to 10-fold higher than the densest city in the world. In January to March 1941, the Germans forced 66,000 displaced refugees into the ghetto (11, 19), soon after it was sealed. Initially, new arrivals were quarantined for 14 days to prevent typhus spread, but the practice was soon abandoned. From this moment, the epidemic was unstoppable. “The first signs of disease appeared among the [refugees] … who were crowded like animals into the institutional buildings. So high was the death rate in these places that they were popularly known as death points. It was not long before the first sparks of disease spread like a forest fire throughout the entire ghetto” (27). Surprisingly, this quote from 1950 might be the very first verbal model of a disease outbreak couched in terms of what would decades later became the physicists’ famous “forest fire” epidemic paradigm.

Sudden curtailment of the typhus outbreak

As shown in Fig. 1, unusually, in late October 1941, just at the onset of winter, the typhus epidemic rapidly began to curtail and collapse. The epidemic’s “turnaround” was completely unexpected since typhus infection normally accelerates during winter. As Emanuel Ringelblum (28), the ghetto’s chronicler, wrote (November 1941): “The typhus epidemic has diminished somewhat—just in the winter, when it generally gets worse. The epidemic rate has fallen some 40 per cent. I heard this from the apothecaries, and the same thing from doctors and the hospital.” The same feature was also recently noticed in the thoroughly researched book of Miriam Ofer (29). Here, we use a modeling approach to help understand the epidemic’s unexpected collapse. Namely, we ask, did the epidemic naturally burn out? Or were other factors responsible (30)?

SEIRL model of typhus spread

The transmission dynamics of typhus in Warsaw Ghetto was usually through contact or close proximity to an already infected individual, as this allowed lice to pass from one host to another in the densely crowded ghetto. Residents were terrified of accidental contact and practiced social distancing (see section SM1). We therefore deploy an “SEIR”-type epidemic model given it assumes contact transmission. In this model, each human host can only be in one of four classes at any time t: susceptibles, exposed, infected, or recovered, and the variables S(t), E(t), I(t), and R(t) express the numbers in each class, respectively. An equation is developed for each class, and an SEIRL model is designed in which consideration is also given to the important “reproductive number” $R_0(t)$. The latter estimates the number of individuals infected by a typical infected individual over the lifetime of their illness in a wholly susceptible population and reflects the transmission ability of the bacteria.

The model was then used to simulate the number of reported typhus cases as a function of time (Fig. 3), as well as the total number of reported and unreported infected cases. Following convention, it was first assumed that the reproductive rate $R_0(t)$ is a fixed constant over time. Figure 3 plots the time series of stochastic simulations of the major outbreak generated from the best-fitting model, indicating the median and envelope of individual trajectories for reported cases. The model provides an accurate fit to the epidemic, as shown, yet this fit only holds with an unrealistically low reporting rate $\rho = 0.07$ (7%) and thus predicts an extraordinarily large total number of typhus infectives of 303,200 (median), i.e., three-quarters the population of the entire ghetto. However, in reality, the epidemic infected only 80,000 to 110,000 residents in total (section SM3). One concludes from Fig. 3 that it is impossible to fit the major epidemic in Fig. 1 with a standard SEIR-type model with constant transmission rate, for parameters that are in a realistic range of values. The approach is unusable, and so, in the analysis that follows, we move on to allow $R_0(t)$ to flexibly change with time rather than remain a constant.

A number of independent historical reports indicate that the reporting rate of typhus cases is approximately $\rho = 20$ to 25% (section SM3). In terms of predicting the total number of cases from the observed data, we deliberately err on the conservative side and therefore make the assumption that $\rho = 25$%. The model was then again used to simulate the number of reported typhus cases as a function of time (Fig. 4) but now fitting a flexible $R_0(t)$. As seen in the resulting time series of the best-fitting model in Fig. 4A, the epidemic always peaks in late October. The total number of simulated reported and unreported typhus cases is ~72,000 (median) over the period.
September 1940 to July 1942, although it could even reach a maximum of 113,000 infected. These totals completely align with reports of ghetto epidemiologists (section SM3). For example, Penson (20) estimated ~100,000 typhus cases, which is quite plausible according to the model (section SM7 and fig. S2). In short, this form of the model with flexible $R_0(t)$ provides results that are self-consistent with available data, literature, and known parameter values.

Several independent reports from the period give estimates of the case fatality rate (CFR) as approximately 20 to 25% (section SM4), and thus, some 20,000 to 25,000 deaths due directly to typhus would seem a reasonable estimate. All of this dying took place in an area of 3.4 km². Several epidemiologists have suggested that typhus was responsible for 25,000 direct deaths (20, 26), but it is known that it led to the deaths of many others indirectly.

Normally, the crash of an epidemic is indicative of a lack of susceptibles remaining in the population to infect, thereby bringing the epidemic to its end (30). However, in Warsaw, it was only after approximately 40,000 residents (<10%) became infected, of a large population of >400,000, that the epidemic began to collapse in October. As the number of susceptibles in the population remained relatively high over the whole period as shown in section SM7, the epidemic is unlikely to have died naturally for want of susceptibles. Even stronger reasons are found by examining the disease transmission rate $R_0(t)$ over the course of the epidemic, as estimated by the model fitting procedure. In Fig. 4A, the reproductive number $R_0(t)$ is seen to steadily increase in January 1941 but then, in June, suddenly monotonically diminishes in magnitude. As is well known (30), when the effective reproductive number drops below its unity “tipping point” [i.e., below $R_{eff}(t) = R_0(t) S(t) = 1$], the epidemic collapses. Thus, whatever caused the transmission rate to decrease to 20% of its value in Fig. 4A is responsible for the collapse of the epidemic before winter. In Discussion, we argue that antiepidemic activities of the ghetto community are the likely cause here.

**The catastrophe that was stopped**

We now use the model to predict the epidemic trajectory in the absence of any form of antiepidemic activities that might reduce $R_0(t)$. This requires preventing the transmission rate $R_0(t)$ from dropping in the last months of 1941 and artificially ensuring that it remains approximately a fixed constant $R_0(t) = 2$. (Similar results were found in section SM7b for different regimes of antiepidemic activities.) The underlying assumption is that any increase in antiepidemic activities in the ghetto corresponds to a reduction in $R_0$. As Fig. 4B
shows, the end outcome is a massive outbreak with two to three times more typhus infectives. On average, 196,000 (median) residents become infected when $R_0(t)$ was constrained, as compared to 72,000 (median) when $R_0(t)$ was fully flexible. (Full statistical details may be found in section SM7.) Moreover, note that the epidemic now operates throughout winter, peaking in January, rather than late October. Hence, the curtailing of the epidemic (Fig. 4A) averted a major winter catastrophe (Fig. 4B).

**Deaths and the maths of food ration cards**

The dataset for registered deaths shown in Fig. 2 appears in many studies on the Warsaw Ghetto (18, 19, 31), but is it reliable? The German epidemiologist Hagen believed that it only gave an indication of two-thirds of the deaths (16). Moreover, various medical reports from the ghetto reflect the underreporting of these data as we document in section SM5. Another obvious problem is that from a visual inspection, the dynamics of the typhus epidemic (Fig. 1) are not superimposed or modulating the registered mortality data (Fig. 2). However, we know from reports that when typhus cases were reaching their maximum, they generated many deaths directly, and far more than in other periods. Thus, close to the epidemic’s peak, in “August [1941] 5.6 thousand people died, including 3.5 thousand confirmed cases of typhus” (32), which is 63% of all deaths.

Penson (20) also points out that the death rate increased from 4000 to 5000 deaths per month in early 1941 to 9000 at the peak of the typhus outbreak. However, the impact of the typhus peak is not seen in the official mortality data (Fig. 2). Moreover, those who recovered from typhus often died by starvation in convalescence, indicating the important indirect impact of typhus on the mortality rate. As such, it is unusual that signs of the typhus outbreak in the dynamics of the registered deaths are so minimal. Hence, there are serious indications that the reliability of the dataset is questionable. It is well known that when the deaths in the ghetto were at their peak, the community was unable to deal with the large number of corpses on the streets and in the overflowing cemeteries, let alone their registration.

We therefore check for other means to quantify the death rate and take advantage of an alternative scheme devised by the ghetto’s Statistical Division. The statisticians ingeniously made use of monthly counts of food ration cards to obtain a regular census of the population of close to a half million people (18, 19, 31). The cards were used by almost all members of the ghetto to obtain food-ration cards. Their total numbers per monthly census from (31) plotted in Fig.5a in blue, and range from 445,000 cards in March 1941 to 355,000 in July 1942. Taking into consideration information in (31), the former figure is a likely to be an underestimate and the latter figure requires adjustment to 327,073 (see Fig. 6). Thus approximately 118,000 residents went unaccounted for, nearly all of whom in all likelihood died of disease or starvation. Yet the official registered rates of deaths, as found in Trunk (17) and (31), amounted to ~70,500 in total for this same period. A forthcoming work will give a deeper accounting of these statistics (see also SM5).

While the accuracy of this census method has its own problems (19, 31), the monthly differences in number of food ration cards should be a reasonable proxy for the number of monthly deaths in the population, after demographic adjustments if needed (Methods). The large drops in the number of ration cards indicate large death rates in the April to October 1941 period followed by a wind-down in 1942, as seen by changes in the slopes in Fig. 5A. In Fig. 5B, the monthly differences in ration card numbers are plotted directly, when monthly data are available. We see that card counts mimic features of the typhus epidemic dynamics. New deaths are seen to be proportional to the number of new typhus cases (after some delay). On the other hand, lice find new hosts as soon as their current host dies, which should lock in the relationship further—the number of new infectives being related to the number of new deaths.

Engelking and Leociak (18) argue that ration cards were almost never forged, and this should not be considered a source of bias.
However, it is interesting to speculate whether the Jewish Council might have had their own motives for manipulating the number of needed ration cards and introduced unwanted bias. Thus, they might have increased the number of cards or decreased it for reasons unrelated to the actual number of inhabitants. We believe that this is also unlikely: The Jewish Council was required to present the ration card data when requesting food from the Germans. While the former was interested in obtaining as much food as possible, the occupiers would have sanctioned any betrayal with concentration camp or death. Moreover, in practice, the Germans did not deliver food in accordance to the number of ghetto inmates but provided a set amount whose distribution was in the hands of the Jewish leadership. In short, the Jewish Council wanted objective figures of ration cards because it helped them with distribution inside the ghetto. In reality, the Germans did not care about this figure, so there was no reason for the Council to forge it, as it would not have helped them at all and possibly have had dangerous repercussions. (See section SM5 for a discussion of other caveats.)

**DISCUSSION**

Penson (20) referred to a 20% mortality rate in 1941 in the Warsaw Ghetto, with close to 20,000 dying of typhus. However, 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 from the other seen in Fig. 5. In addition, although not enforced. Home self-isolation was put in practice, although not comprehensively. Last, complex and highly elaborate sanitation programs and measures were developed by the Health Department and Council (14, 18, 26) with the goal of eradicating typhus.

The Nazis were aware and counted on the intertwined character of starvation and epidemics. For them, starving Jews meant more food for Germans, which was a key motivation for the onset of the industrialized mass murder (33). In the end, ideological factors amplified themselves: The starving of the ghetto increased epidemics, which seemingly proved Jews as (bearers of) diseases. Those were to be eliminated, and the elimination would then save even more food. Furthermore, by the end of 1941, it had become clear that Nazi visions of deporting Jews to Madagascar were illusions (34), and even more murderous “final solutions” were necessary. Thus, the deportation of more than 260,000 of Warsaw’s Jews to the Treblinka death camp from July 1942 onward happened despite the fact that there was no longer a raging epidemic and the much less populous ghetto was actually productive.

We return now to the unusual curtailing of the typhus outbreak in late October 1941, and note that the idea that behavioral response can influence the course of epidemics is well understood in theoretical epidemiology (35). In the case of Warsaw Ghetto, a number of factors are likely to have been responsible for the drop in transmission rate \( R_0(t) \) that led to the epidemic’s early collapse. Until April 1941, the main goal of the German “attritionist” ghetto administrators was to starve every ghetto resident who could not produce valuables to buy food. This kind of lethal depopulation of the ghetto was also considered as an effective means against epidemics. For the Germans, outright murder to stop an epidemic outbreak is known to have occurred at the Gypsy camp inside the Litzmannstadt/Lódź ghetto in January 1942 (36). The argument of mass murder to prevent epidemics was also used by Romanians after consultations with Germany when killing some 50,000 Jews in Bogdanovka, occupied Transnistria, in December 1941 (37).

However, from May 1941, the new “productionist” administrators in Warsaw made plans to build a self-sustaining ghetto economy. The productionists believed that sources of labor should not be wasted, and therefore, some component of the ghetto residents should be given minimal food and nutrition so they could work. Thus, in mid-1941, the ghetto was improving economically with some increases in food aid, and for a short period, a blind eye was given to food smugglers. During April to September, the ghetto was stocked up more than ever and started working economically in German eyes (11). A new “feeding program” was introduced, whereby large numbers of community kitchens run by volunteer agencies became vital for one-quarter of the population in supplying basic (albeit very meager) nutrition. In December 1941, ghetto residents had between 784 and 1665 daily calories, depending on wealth and social status that allowed, e.g., for acquiring smuggled food (18). The community’s diverse network of Social Self-Help and medical organizations were intensely involved in fighting the epidemic, and health education and hygiene became major thrusts (18, 26). Numerous sanitary courses were initiated, covering public hygiene and infectious diseases, in which >900 people sometimes participated. There were hundreds of public lectures on the fight against typhus and epidemics. An underground university was set up to train young medical students, and scientific studies on the phenomenon of starvation and of epidemics were undertaken. Building and apartment cleanliness was encouraged and often enforced. Social distancing was considered basic common sense by all, although not enforced. Home self-isolation was put in practice, although not comprehensively.
These efforts under the given conditions were what Adina Blady-Szawürger, a surviving doctor of the Warsaw Ghetto, called “superhuman medicine” after the war (18). It is proof of the successful politics of the Jewish Council, which often was blamed as being corrupt and incompetent (38).

Nevertheless, under abysmal ghetto conditions, these efforts often ran into major obstacles and were often difficult to implement in practice. On the other hand, there are no other reasonable alternative hypotheses to explain the early demise of the epidemic at the onset of winter. The only alternative explanation available we are aware of is the excellent fit to the data in Fig. 3 based on a constant transmission rate. The fit suggests a reporting rate of $r = 0.07$ (7%), implying a total number of typhus infectives of $303,200$ (median), which is far larger than any estimate of epidemiologists who were on location and is thus unlikely.

The spread of typhus was hardly restricted to the Warsaw Ghetto, and in WWII, it appeared all over Europe—thus its pandemic nature. Arad (39) provides details of some of the horrors in the Ukraine in late 1941 and early 1942. In the town of Shargorod in the Vinnytsia oblast, we learn that the medical services established in the ghetto were inadequate and “made it impossible to block a typhus epidemic early in 1942.” While thousands of people died in the typhus epidemic, “[o]nce a cleaning service and a workshop for the manufacture of soap had been established in the ghetto, as well as public lavatories and baths, the number of typhus victims dropped and the epidemic was curbed.” This demonstrates the plausibility of our argument that the Warsaw Ghetto typhus epidemic was curtailed through the community’s attempted antiepидemic activities. Second, these and similar descriptions of other ghettos tell us that outside of Warsaw, the epidemic continued through the winter months, and this would seem to be the expectation for Warsaw Ghetto as well. Third, we learn of the far higher infection and death rates from typhus in the Ukraine. Fourth, we learn that “the Germans’ ideological connection between typhus and Jews as racially inferior” was used again and again as a pretext for massaging Jewish victims (37).

There are interesting parallels of the eradication of typhus in Warsaw Ghetto and attempts to contain COVID-19 (coronavirus disease 2019). Both actions sprang from the same fear and panic typifying cities in the grip of a plague (18). COVID-19 CFR is 1 to 2% (40) and quite likely less, while in Warsaw Ghetto, the typhus CFR was close to 20%. Containment in mainland China was reached through draconian quarantining, social distancing methods, and travel bans. In contrast, in Hong Kong and Taiwan, the COVID-19 infection numbers stabilized despite less rigid approaches. Similar to the aforementioned regions, the Warsaw Ghetto had numerous internal institutions, civil, medical, and social self-help, working intensely over many months to stamp out the typhus epidemic. A whole range of strategies were explored (18). However, many failed due to lack of experience and knowledge, inability to carry out programs in practice, sabotage for financial gain, or the diabolical methods of the Nazis. Nevertheless, the constant ongoing efforts of the community and self-help organizations to destroy the epidemic must have certainly contributed to lowering transmissibility below a critical threshold, bringing the epidemic to an early sudden halt. Richards (41) described a similar outcome in his anthropological study of Ebola epidemics in Sierra Leone. In this way, our quantitative analysis predicts that antiepidemic activities greatly contributed to the prevention of typhus infection to >100,000 residents and helped prevent tens of thousands of deaths. Our results thus give support to Gutman (19), one of the ghetto’s most respected historians, who wrote that “while almost 100,000 ghetto residents died mostly from starvation and disease in the period up until July 1942, a similar or greater number were saved thanks to the dedicated relief workers and self-help relief agencies operating.” The Warsaw Ghetto residents were completely unaware of what had been achieved.

**METHODS**

**The SEIRL model**

We begin with a vector-host model applying the definitions from the section, SEIRL model of typhus spread (Results), for the standard $S, E, I,$ and $R$ components (30), but now a new variable for the vector (louse) $L(t)$ is added. The equations read

$$
\begin{align*}
\dot{S} &= A(t) - \frac{\beta(t)}{N(t)} SL - \mu S; \\
\dot{E} &= \frac{\beta(t)}{N(t)} SL - (\sigma + \mu) E; \\
\dot{I} &= \sigma E - (\gamma + \mu) I - m(\gamma + \mu) I; \\
\dot{R} &= \gamma I - \mu R; \\
\dot{L} &= \kappa I - \lambda L
\end{align*}
$$

The total but changing population size at time $t$ is $N(t) = S(t) + E(t) + I(t) + R(t)$. The mean latent or incubation period of the disease is $\gamma^{-1} = 14$ days, which is well documented in the historical literature (21, 24). The mean infectious period is also documented, and we set $\gamma^{-1} = 28$ days (24), and in section SM7b, we explore other values and justify this choice further. Setting $m = 0.25$ gives an infection mortality rate of $m = 0.25 = 20\%$ as documented in section SM4. The parameter $\mu$ is the rate of removal due to death (other than through typhus infection). $A(t)$ denotes population displacements into the ghetto (e.g., refugees). These parameters are fitted to satisfy the trends in the historical population numbers $N(t)$ as discussed in section SM7.

We use a frequency-dependent vector-host transmission function that accounts for the term $\frac{m(t)}{1 + m(t)} SL$. The main goal is to fit the transmission rate $\beta(t)$ and the reproductive number $R_0(t) = \frac{\beta(t)}{\gamma}$. As the louse is capable of moving from host to host with great rapidity (relative to other dynamics), this leads to a slow-fast system, which we assume reaches quasi-equilibrium so that $dL/dt = 0$ and thus $L(t) = \frac{\kappa}{\lambda} I(t)$. The SEIRL model then simplifies to an SEIR model, which aids in fitting the model parameters, especially given the very small size of the monthly dataset (Fig. 1).

The typhus cases (Fig. 1) are modeled as a POMP, making use of iterated filtering and plug-and-play likelihood–based inference frameworks to fit the data (42, 43). These are modern state-of-the-art statistical methodologies developed for fitting complex epidemiological datasets. The maximum likelihood estimate for model parameters is calculated using Cran R package “POMP” (42). The simulations made use of the Euler-multinomial integration method with the time step fixed to be 1 day (43) with both demographic and observation noise. That is, the model estimates the true number of cases but then adds a reporting layer to simulate observed data. See section SM7 for details.

**SUPPLEMENTARY MATERIALS**

Supplementary material for this article is available at http://advances.sciencemag.org/cgi/content/full/6/30/eabc0927/DC1

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