New Historical Evidence on the 'Russian Influenza' of 1889: Primary Source Documentation from the Bukharan Archive

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Abstract

The pandemic of 1889–1890, commonly known as the "Russian Influenza," is conventionally attributed to influenza and assumed to have originated in Central Asia before spreading globally via trade routes. However, the scholarly identity of this pathogen remains contested: while some researchers argue for H3N8 or H3N2 influenza on the basis of seroarchaeological and phylogenetic evidence, a growing body of clinical analysis from European sources has advanced a coronavirus hypothesis. Both sides of this debate have been constrained by a shared limitation: the absence of systematic clinical and epidemiological documentation from the probable outbreak region itself.

We present newly translated and systematically analyzed primary sources from Bukharan administrative archives documenting an outbreak beginning in 1884 in the Qarshi region and continuing through the 1889 spread through Bukhara, Samarqand, and into the Ferghana Valley. These records describe specific clinical signs (jaundice, cyanosis, progressive edema), mortality patterns, and epidemiological progression that are more consistent with coronavirus evolutionary adaptation than with influenza dynamics. We interpret this evidence through a three-stage model of coronavirus host adaptation—from high-mortality zoonotic spillover with broad tissue tropism, through stuttering chains of limited human-to-human transmission, to a pandemic-capable strain characterized by efficient respiratory spread and attenuated hepatic involvement—and argue that the Bukharan archival record documents precisely the pre-pandemic phase this model predicts. Critical context includes the predominance of karakul (Qaraqol) fur trade in the region—an animal husbandry practice already documented to enable transmission of related coronaviruses in other species—and the relative absence of livestock typically associated with sustained influenza transmission.

We present these sources as a curated dataset for researchers studying 1889 pandemic origins and early coronavirus emergence, without claiming to definitively resolve the pathogen's identity. These data may constrain epidemiological and phylogenetic models of spillover and early pandemic dynamics and motivate targeted viral surveillance in the Kyzylkum region.

Keywords: 1889 pandemic, coronavirus spillover, historical epidemiology, Central Asian outbreaks, zoonotic emergence, HCoV-OC43, host adaptation

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1. Introduction

The pandemic of 1889–1890 is one of the best-documented pandemic events prior to the era of virology, yet its etiology remains genuinely contested. In the immediate aftermath, government commissions converged around a consensus: attributing its origins to the Bukharan Emirate with westward spread through Russia, facilitated by the newly established Trans-Caspian Railway, and identifying the clinical manifestation as resembling influenza rather than dengue or other vector-borne fevers.[1] Within six months, the illness had circumnavigated the planet, with mortality peaking in Saint Petersburg in December 1889, then in Paris and New York in January 1890.[2] The pandemic was mild in relative terms, with a case fatality ratio worldwide between 0.1% and 0.28%, making it roughly ten times less deadly than the 1918 influenza pandemic.[3]

The modern scholarly debate over pathogen identity has unfolded on two fronts. On the influenza side, Worobey and colleagues have argued that birth-cohort patterns of 1918 pandemic mortality are consistent with antigenic imprinting from prior exposure to an H3 subtype influenza circulating from roughly 1889 onward—suggesting H3N8 as a candidate.[4] On the coronavirus side, Brüssow and Brüssow (2021) have presented clinical and demographic evidence from European sources—multisystem involvement, elderly-predominant mortality, neurological sequelae—arguing that the 1889 pandemic was caused by an ancestral lineage of HCoV-OC43 or a closely related embecorvirus.[5] Phylogenetic molecular dating has placed the most recent common ancestor of HCoV-OC43 in approximately this era, though recent reanalysis has contested the precise dates (see Section 6.3).

We do not claim to settle this debate. We note, however, that the influenza and coronavirus hypotheses are not necessarily mutually exclusive: seroarchaeological evidence demonstrating H3 exposure in birth cohorts of the early 1890s is compatible with concurrent seasonal H3 influenza circulating alongside a pandemic coronavirus. Meanwhile, Central Asia's role in this history is significant but poorly characterized.[6] Contemporary sources suggest the outbreak emerged or intensified in the Bukharan oasis before spreading via railway. Yet detailed epidemiological and clinical documentation from this region has remained difficult to access and is often cited at second hand through colonial administrative reports.

We present newly translated and systematically analyzed primary sources from the Bukharan administrative archive documenting outbreaks from 1884 through 1890. These records, written in Russian and Persian/Tajik by administrators, physicians, and local officials, describe specific clinical signs, mortality patterns, and the geographic and temporal progression of illness. Critically, they document an outbreak beginning in the Qarshi region in 1884 and its subsequent spread to Bukhara and beyond, providing a chronological arc often absent from existing accounts. Our interpretation of these documents is informed by a distinctive ecological context—intensive karakul sheep herding—that is epidemiologically distinct from the waterfowl-agriculture interface typically associated with influenza emergence.

Our contribution is deliberately curated rather than conclusive. We merely aim to render the archival evidence legible to virologists and epidemiologists studying coronavirus spillover, zoonotic

emergence, and pandemic origins. To that end, we note where these sources provide positive support for the coronavirus hypothesis, where they require engagement with competing hypotheses, and where primary source gaps remain to be resolved.

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2. Source Material and Methodology

2.1 Archive and Document Types

We draw upon documents from the National Archive of Uzbekistan (O‘zR MA) and the Archive of the Foreign Policy of the Russian Empire (AVPRI). The primary source base comprises Russian-language reports from the Russian Political Agency in Bukhara, combined with Persian-language internal Bukharan administrative records.

Late nineteenth-century medical data presents inherent challenges to modern health specialists, regardless of geographic origin. Bukhara was a unique case even within the Russian Empire: it was an indirectly ruled protectorate, meaning Russian authorities relied on cooperation from Bukharan Amirate officials, and the two administrative traditions used separate languages (Russian and Persian) within increasingly separate frameworks for understanding disease and medicine: although germ theory began displacing miasma in the popular understanding of Russian physicians, and many Bukharan sources remained rooted in the Avicennan paradigm, in practice medical paradigms were split between old and new on both sides of the colonial divide. Comparing documents across these registers is imprecise—but, we argue, not altogether impossible.

Documents included in this analysis:

- Internal Bukharan reports about disease symptoms and mortality
- Correspondence between regional governors and the capital describing labor shortages, population losses, and prevailing illness descriptions
- Mortuary and burial records from multiple villages, with mortality dates and sometimes explicit cause-of-death notes
- Trade and caravan records documenting illness-related disruptions to commercial movement

It is worth emphasizing that —even though to modern eyes the data may seem sparse— the depth of documentation about this episode is remarkable, suggesting an extraordinary event. There are very few suriviving government records (as distinct from the manuscript tradition, which is much more plentiful) from the Bukharan side prior to 1890 *at all*. Meanwhile, the Russian Political Agency (a permanent colonial resident) had been established just a few years prior to these events, making the amount of attention paid to the pandemic similarly remarkable.

2.2 Translation and Terminology Validation

A core methodological challenge is distinguishing between genuine clinical descriptions and administrative formulae or scribal templates. We address this through systematic comparison of

terminology across independent sources.

Key symptom terms:[7]

Original Term (Persian)	Original Term (Russian)	Modern Clinical Correlate	Date Range	Confidence
tab (fever)	*likhoradka*, glossed as "malarial," "swamp," or "local"	Fever	1883–1889	High — primary term in both traditions
farāshā, *larza* (trembling/shivering)	*oznob*	Rigors/chills	1886, 1888–9	High — document explicitly equates these terms
—	*paroksizm*	Paroxysm (febrile or convulsive; ambiguous)	1886, 1889	Medium — Russian only; referent unclear
lāḥaż (weakness)	—	Asthenia/weakness	mid-1880s	Medium — Persian only; etymology ambiguous
wazmini / *wazbini* (heaviness)	—	Malaise or myalgia (inferred)	1885–6	Medium — Persian only; literal meaning "heaviness"
zardī (yellowness)	Descriptive only: "dark, sickly yellow" skin — *zheltukha* not used	Jaundice or skin discoloration (inferred)	1884–6	Medium — clinical identification is editorial inference; neither source uses a diagnostic term
kabud (blue, of fingernails and toenails)	Descriptive only — *tsianoz* not used	Peripheral cyanosis (inferred)	1884–6	Medium — clinical identification is editorial inference; both fingernails and toenails specified in Persian
—	*opukhal'* / *opukhol'*	Ascending edema	1886	High — Russian only, but described in detail

Original Term (Persian)	Original Term (Russian)	Modern Clinical Correlate	Date Range	Confidence
qay (vomiting), *ashāl* (diarrhea)	*zheludochnye kattary*	Gastrointestinal symptoms	mid-1880s (Persian); 1889 (Russian)	High — attested in both traditions
—	*porazhenie pecheni, selezenki*	Hepatosplenomegaly (advanced cases)	Sept. 1889	Medium — Russian only; Jizakh report only
—	*porazhenie mozga i mozgovykh obolochek*	Neurological involvement (advanced cases)	Sept. 1889	Low-Medium — Russian only; Jizakh report only; vaguely described

Validation method: Where multiple independent sources describe the same outbreak (e.g., village tax records, burial registers, provincial correspondence), we compare the terminology used. Consistency across independent, non-overlapping documents, and (in some cases) absence of overlap between contemporary documents and Avicennan terminology in medical manuals, suggests these were observations of discrete phenomena rather than scribal templates. For example, the mid-1880s outbreak in villages near Qarshi and Kermina is described independently in numerous documents with consistent terminology for 'yellow skin' and 'blue fingers,' suggesting these were salient, observable phenomena.[8]

Limitations explicitly acknowledged:

- These records were not written by physicians; they reflect layperson observation and administrative convenience.
- Terminology may conflate different conditions.
- Absence of a symptom in the records does not mean it did not occur—only that it was not recorded as noteworthy or administratively relevant.
- Mortality data may undercount rural deaths; burial practices varied and not all deaths necessarily reached official record.
- The sample size of surviving indigenous (Persian-language) documents is limited by modern standards, numbering in the dozens.

3. Environmental and Epidemiological Context: The Bukharan Oasis

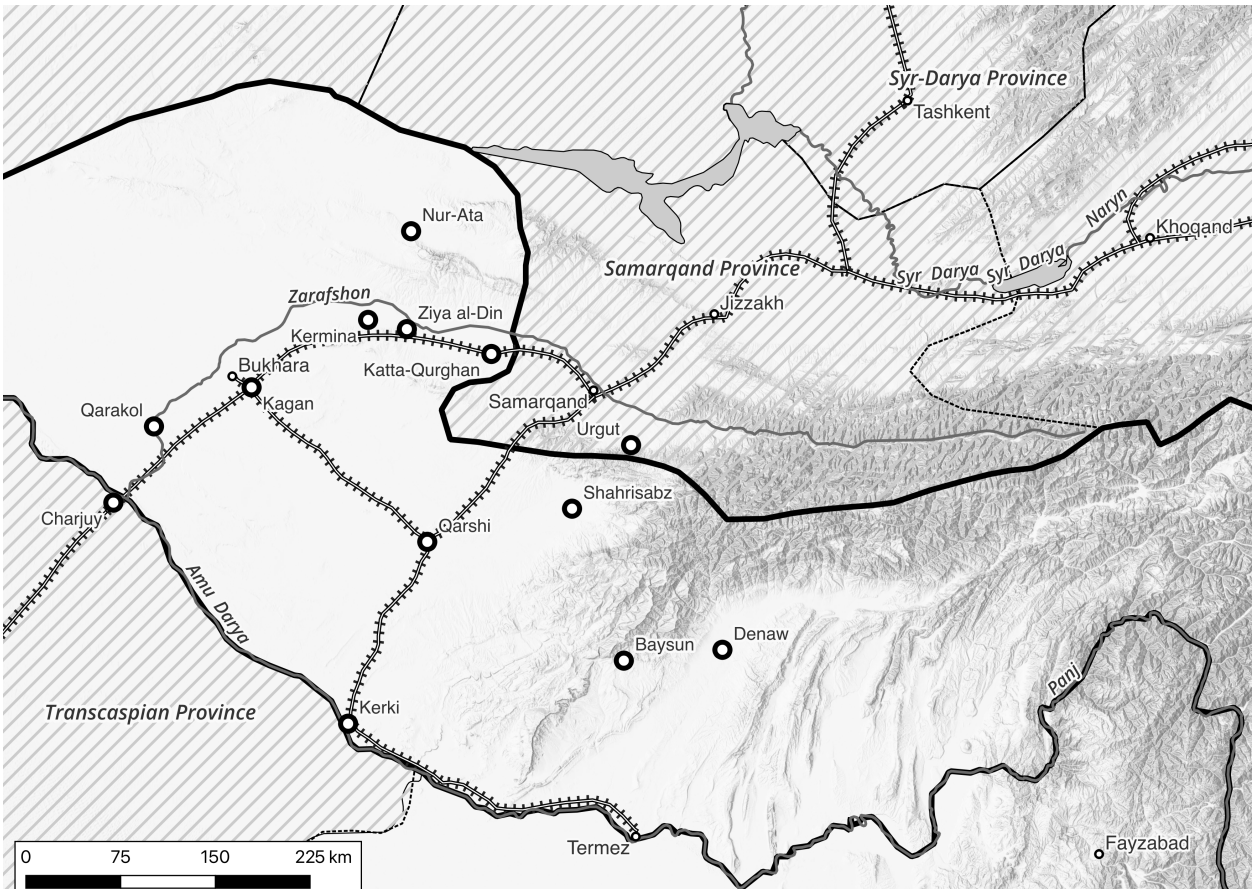

Figure 1: Bukharan Oasis

3.1 Ecological Setting and Earlier Fevers

The Bukharan oasis in the nineteenth century was characterized by intensive agriculture and a distinctive livestock economy centered on karakul (Qaraqol) sheep herding. Karakul sheep are prized for their astrakhan pelts and were widely bred in Central Asia, particularly in the oasis regions surrounding Bukhara and in the Kyzylkum desert, where their ability to forage on sparse vegetation made them the dominant livestock species. This animal husbandry practice is epidemiologically significant for understanding pathogen spillover.

The ecological context was not without earlier precedent for unusual fevers. Russian administrative records from the early years after the conquest of Central Asia document reports of severe fevers in the Zeravshan valley as early as 1871, when the head of the Zeravshan province wrote to the Governor-General of Turkestan that "entire villages" had fallen ill in the summer heat, rendering villagers unable to harvest grain and native officials too sick to collect taxes—and calling for quinine to be sent from Petersburg.[9] The recurrent presence of quinine as the requested remedy across multiple decades is epidemiologically relevant (see Section 5.1). We cannot determine whether these

earlier fevers represent the same pathogen, a distinct endemic illness, or malaria; their significance is primarily to establish that the Bukharan oasis was an ecologically complex disease environment whose inhabitants were familiar with multiple pathogens, which makes the consistent description of the mid-1880s Qarshi illness as distinctly novel all the more meaningful (see Section 4.1).

3.2 The Karakul Sheep Ecological Niche

Unlike the waterfowl-agriculture interface implicated in influenza emergence, the intensive ruminant herding and lambskin fur trade in Bukhara create a distinct ecological niche, with the prevalence of sheep husbandry emphasized by contemporary travellers.[10] This animal-commerce interface is precisely the kind of high-risk spillover setting that contemporary frameworks for pandemic risk identify as warranting targeted surveillance (Carlson et al., 2025) — and, we argue, as warranting retrospective historical investigation for the same reason.[11] Related coronaviruses are documented in sheep and other ruminants: bovine coronavirus (BCoV) infects cattle and sheep, while camel coronavirus (HKU23) is endemic in camel populations. [12] Transmission of disease from Bukharan herds to those in Turkestan was an active concern of colonial authorities, who established veterinary inspection points along the border specifically to intercept infected livestock.[13] The ecological argument thus runs in both directions: the Bukharan oasis presents a plausible setting for a coronavirus spillover event and presents a meaningful obstacle to the influenza hypothesis.

The evolutionary trajectory from animal coronavirus to human transmissibility shows selection pressure toward respiratory tropism—a dynamic that we develop formally in Section 6.1 and argue is visible in the clinical and epidemiological data from Bukhara.

Additionally, the Bukharan oasis supported complex human-animal contact through:

- Shared water sources between herds and human settlements
- Milk and milk product consumption (a documented transmission route for some coronaviruses)
- Close proximity during animal processing and slaughter, including large-scale slaughter of sheep for Eid al-Fitr
- Overlapping foraging areas between sheep herds, bats, and rodents—relevant to the rodent-origin hypothesis for the OC43 ancestor
- High density of human settlement in the oasis relative to surrounding desert, facilitating secondary human-to-human transmission once respiratory adaptation had occurred

The timing of Eid al-Fitr in 1889 (late May–early June) is potentially significant: large-scale human gathering combined with intensive human-animal interface may have provided superspreading conditions favorable for further population-level adaptation for human-to-human transmission, just prior to the documented urban outbreak in Bukhara in June–July 1889.[14] We flag this as speculation warranting further attention.

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4. Outbreak Patterns: Clinical and Epidemiological Description

4.1 The Qarshi Outbreak (1884–1888): Initial Spillover Phase

Russian sources explicitly report that a fever (*likhoradka*) 'raged' (*svirepstovala*) in Qarshi beginning in 1884 or perhaps 1886; dating in the sources is sometimes ambiguous.[15] Crucially, contemporaneous Russian accounts emphasize that this illness seemed *inherently new and different*—distinct from the endemic fevers, malaria, and cholera that the local population would have recognized. The sources imply alarming novelty in clinical presentation, as opposed to simply an increased incidence of a known endemic pathogen—a distinction that is more significant precisely because these observers had a wide baseline of endemic illness against which to compare.

The most important Russian archival source is a brief 1886 report written in Katta Qurghan, based on intelligence from Central Asian intermediaries.[16] According to this account:

- The stricken individual initially felt thirsty and hot, often with shivering (*oznob*) beginning within 1.5 hours
- Fever would follow 3–4 hours after initial onset, with paroxysm (a term that appeared frequently in the 1889 reports) and full-fledged fever after 1–3 days[17]
- The sick individual became emaciated and pale; skin turned dark yellow
- After 2–3 months, swelling (*opukhal'*) appeared, usually beginning at the feet and working upward to the waist
- Progression of swelling to the midsection (whether ascending or descending, over 3 months to 1 year) typically resulted in death; the swelling took on a "vitreous" (*steklovidnoi*) appearance during high temperature and a dark yellow color during shivering (*oznob*).[18]
- The swelling took on a 'vitreous' (*steklovidnoi*) appearance during periods of high temperature, and a dark yellow color during periods of shivering

The Russian report emphasized that only people living in Qarshi itself were affected; nearby villages were entirely spared unless residents traveled to the city.[19] However, a notable anecdote is recorded: when 80 people from one particular village visited Qarshi for 5–6 days, all but one died from the illness after varying intervals following exposure. A retrospective report from 1889 also claims that the Amir of Bukhara himself, Muzaffar (d. 1885), succumbed to this illness. [20]

Bukharan sources from roughly the same period (1884–1888, though dating is approximate due to administrative practice) describe persistent fevers (*tab-i daraja-i muqarrari*) in Karmina (modern-day Navoi) and Qarshi.[21] Bukharan records document an especially severe outbreak in a specific village in Ziya al-Din also mentioned in Russian reporting, confirming that the separate paper trails refer to the same illness.[22]

Symptoms emphasized in Bukharan sources include: fever (*tab*), shivering (*farasha* [specifically trembling from a fever, likely equivalent to the Russian *oznob*], *larza*),[23] overbearing pressure or heaviness (*wazmini*),[24] and enervation (*lahaz*, which in the original Arabic meaning referred to an illness that brought the victim to the verge of death).[25] More severe outcomes were marked by

distinctive signs: if the victim's fingernails and toenails began to turn dark blue (*kabud*) shortly after weakness set in, death came within 3–4 hours. Conversely, if the victim turned yellow, they generally recovered after experiencing vomiting and diarrhea.[26]

On notable absences: In addition to the details described explicitly, there are also several noteworthy omissions. While absence of evidence should not be equated with evidence of absence, it seems unlikely that either rashes or bleeding were essential features of this illness. Respiratory symptoms are somewhat more ambiguous, as 'paroxysm' (*paroksizm*) seems equally likely to refer to a coughing fit as to a neurological convulsion.[27] Similarly, the names for well-known illnesses such as malaria and cholera are rarely invoked with specificity.

The clinical pattern here is notable: an initial period of constitutional symptoms and fever, followed by visible signs of either hepatic involvement (jaundice) or severe hypoxia (cyanosis), with distinct prognostic trajectories. The prolonged course (months to a year), progressive edema beginning at the feet and ascending, and hepatic involvement require explicit engagement with a significant differential diagnosis, discussed in Section 5.1.

4.2 The Bukhara Outbreak (Summer 1889): Respiratory Dominance

Most surviving Bukharan documents are provincial reports sent to the center, meaning accounts of the epidemic in Bukhara city itself are predominantly from the Russian side. In summer 1889, rumors swirled that 300–400 people were dying per day—so many that body-washers were forced to forego traditional sacraments.[28] This panic prompted the Russian Political Agent, V.O. von Klemm, to pursue more realistic statistics.

When Klemm explicitly requested numbers from the Bukharan government, a representative replied they were 'in no position to provide precise information.'[29] Undeterred, Klemm devised an indirect calculation: he sent agents to survey the guards at Bukhara's city gates, asking how many bodies they observed being carried to burial grounds. At the height of the epidemic (late July 1889), approximately 7 corpses passed through each of the city's main gates daily—roughly 49 bodies total. Assuming the same for cemeteries within the city, Klemm estimated 2,200 deaths over a two-week period, yielding a total death count for the summer of approximately 7,000—roughly 5% of Bukhara's population.[30]

Klemm's figures were deliberately conservative, intended as an antidote to what he considered fearmongering. A military commander reported that from two battalions of 2,000 soldiers each, 10 died in July and 6 in August. An intelligence report from 12 July 1889 stated that 600 out of 1,000 Bukharan soldiers stationed in Karmina had fallen ill.[31] In one neighborhood where illness was especially severe, 30 out of 500 people died (5 from chronic illness, 15 minors).[32] In other neighborhoods, death rates remained normal. The outbreak was serious by June; by late August, it had abated in the city but remained strong in the countryside. By late September 1889, it was reported that the illness had fully disappeared in the city of Bukhara, and was beginning to drop off in the surrounding villages as well.[33] Intelligence from mid-June suggests that the illness was

mostly raging outside of Bukhara in the first two weeks following the end of Ramadan (which ended May 30, 1889), before the city's death count became serious by the end of June—consistent with an initial rural phase followed by urban amplification. A terse intelligence report from 13 June suggests that as of that date the illness was mostly raging outside of the city of Bukhara, but by the end of the month that had changed.[34]

Age distribution: The epidemic did not spare the powerful and wealthy. Two of the Amir's sons (ages 3 and 8) succumbed in late September, as did high officials such as the Chief Judge and Diwanbegi.[35] Conversely, evidence for disproportionate elderly mortality is present but requires further documentation: Klemm's neighborhood-level data notes 15 minors among the dead but does not systematically disaggregate by age.[36] Indeed, Klemm specifically states that healthy adults were not spared: two military batalions of 2000 men lost sixteen individuals to the illness, and in one neighborhood in which 30 out of 500 people had died, around 15 were minors.[37] This matches mortality levels when the disease spread to Jizakh, where a Russian doctor reported children as constituting around 6% of the deaths.[38] The age distribution of mortality—whether it concentrated in the elderly as predicted by the coronavirus hypothesis, or showed the W-shaped or U-shaped curve more typical of influenza—remains a priority for further source analysis.

Virtually all deaths were confined to the native population. Over the entire summer, only 2 Europeans died from the illness, both minors, out of a Russian population of perhaps a few thousand.[39] Many more Europeans fell ill, but Klemm noted anecdotally that 'out of twenty Cossacks stationed at the Political Agency, only a few suffered separate bouts of fever, which ceased after two or three doses of quinine.'[40]

4.2.1 Clinical Description in Bukhara, 1889 The dominant identification in 1889 Bukhara was 'fever' (*likhoradka*), sometimes glossed as 'malarial,' 'swamp,' or 'a local variety' of fever.[41] Russian physicians reported fever and neurological effects, emphasizing diverse manifestations.[42] Descriptions of precise gastric symptoms are somewhat vague: 'The severe form of this fever—which is accompanied by gastric and neurological phenomena and, following paroxysm, complete exhaustion (*pol'nyi upadok sil*)—has instilled panic in the afflicted natives.'[43]

Russian physicians identified comorbidities: guinea worm (*rishta*),[44] spotted typhus (*piatnistyi tif*), cholera, consumption (*chakhotka*),[45] and dropsy (*vodniaka*).[46] Of note, malaria was often understood not as a comorbidity but as the cause itself.[47] Russian doctors insisted the fever 'does not have an epidemic character,'[48] citing poor hygiene and traditional medical practices (bloodletting) as responsible for mortality ("…at least 25% of sick individuals die from improper treatment and ignorance of the most elementary requirements of hygiene"),[49] combined with environmental factors (a severe preceding winter followed by a scorching summer) and the fact that entire families fell sick simultaneously, leaving no one to care for the household or livestock. In the context of households being unable to feed themselves, Klemm writes: "Then sharp (*ostrye*), gastric catarrh (*zheludochnye kattary*) predominate, which constitute a common phenomenon in the summertime, affecting primarily children and the elderly." [50]

4.3 Geographic Spread: Samarqand, Jizakh, and the Ferghana Valley

By fall 1889, the number of infected Russians in Samarqand had surpassed that in New Bukhara (Kagan).[51] Jizakh, located roughly halfway between Samarqand and Tashkent, reported spiking mortality in September 1889, with rates dropping by month's end.[52] At peak, approximately 5–15 people died per day.[53]

A Jizakh physician reported: "By observing the sick in person, and from questioning the surrounding population, I became convinced that advanced cases involved liver failure (*porazhenie*), spleen failure, and sometimes even damage to the brain and brain lining. I met people who remained sick for 15, 20, even 40 days, suffering fever every day if they did not receive treatment. Weakened, emaciated subjects, who did not receive any treatment for significant time, through repeated malarial fevers would often become victims of one of the more serious symptoms."[54] Like Klemm, this physician emphasized quinine as critical for treatment.[55] A local district official, however, reported that following the death or recovery of a sick person, "none of the neighbors, or even the victim's own family members, fell ill"[56]—evidence of limited or absent intra-household transmission in this phase and location, consistent with the still-limited human-to-human transmission efficiency predicted at Stage 2 of the spillover model (see Section 6.1).

Similarly, the Russian garrison in Ferghana Valley (approximately 500 miles to the east of Bukhara, directly ruled Russian territory) recorded an expected spike in the incidence of "malarial fever" in 1889 (2,043.5 per 1,000 roster strength, up from 1,534.0 in 1888, then declining again to 1,528.3 in 1890); however, *mortality* had already nearly tripled the preceding year (from 1.48 per 1,000 in 1887 to 4.61 in 1888, before falling back to 2.20 in 1889 and 2.21 in 1890) — a pattern which aligns with acquired immunity to a novel pathogen over time, and that sits uneasily with malaria, a disease not typically associated with such variable year-on-year mortality rates.[57] Archival sources notably describe the 1890 outbreak using similar language: "malarial fever" caused by "fever bacteria" (*likhoradochnye bakterii*) residing in filthy water.[58]

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5. Assessing Continuity: Did Qarshi and Bukhara Represent the Same Illness?

Two intertwined questions arise: (1) Was the Qarshi fever of the mid-1880s the same illness as the 1889 Bukhara outbreak? (2) Do the respective Bukharan and Russian reports describe the same phenomenon?

Russian officials certainly believed so, stating this explicitly in correspondence that the disease originated in Qarshi, spread to surrounding territories, and then ultimately to Bukhara city itself in 1889.[59] Many (though not all) reported symptoms align between mid-1880s Qarshi and 1889 Bukhara: fever, shivering, paroxysm, emaciation, and jaundice appear in both.

However, some differences are worth noting. In 1889 Bukhara, physicians consistently reported (though somewhat vaguely) gastric symptoms, whereas the 1886 Qarshi report does not explicitly

emphasize GI involvement. Additionally, there is no mention in 1889 Russian reports of the specific swelling term (*opukhol'*) used in 1886.[60] These gaps may reflect documentation practices rather than genuine clinical differences—and as we argue in Section 6.1, a *shift* in the dominant clinical presentation is precisely what a coronavirus host-adaptation model would predict.

Only documents written after 1892 use the term 'cholera' (*waba*) to describe the epidemic, which implies the Bukharan administration maintained continuity of understanding through 1890 but may have reclassified the illness afterward. (In works rooted in the Greco-Islamic tradition of Avicenna, *wabāʾ* is an umbrella term for epidemic diseases caused by corruption of the air. However, in Central Asia the term referred more specifically to cholera, likely acquiring that connotation from Tatar usage.)[61]

On balance, the weight of evidence—official Russian assertion of continuity, shared symptom clusters across independent documents, geographic proximity, and timing—suggests these represent the same epidemic process spanning 1884–1890. The alternative—that two distinct novel pathogens with similar clinical manifestations emerged in the same small region within a few years of one another—is possible but requires a higher evidentiary bar.

5.1 The Quinine Problem and Differential Diagnoses

Bukharan and Russian sources alike emphasized the efficacy of quinine for treating this illness. In mid-July 1889, the Political Agency began distributing free quinine, distributing over 11,000 grams over the summer.[62] Klemm reported "very satisfactory results."[63] Bukharan documentation supports this, recording that citizens accosted Russian officials in the streets seeking quinine.[64] This finding is not trivial: quinine is an antimalarial. At first pass, its apparent efficacy cuts directly against both the coronavirus and influenza hypotheses and in favor of a quinine-sensitive pathogen.

However, the interpretive picture is more nuanced than this. First, the interference of quinine-class compounds with sialic acid biosynthesis in sufficiently high doses may be a plausible antiviral mechanism specifically targeted at exactly the class of virus this paper proposes as the 1889 pandemic pathogen.[65] Second, and more likely: quinine was used empirically as a general remedy for febrile illness throughout this era, and was routinely prescribed for respiratory illness even when malaria was not the suspected cause—including as one of the standard-of-care treatments recommended during the 1918 influenza pandemic, where it was administered without evidence of efficacy against influenza itself.[66] In such cases perceived recovery of milder cases simply represented regression to the mean.

Third, quinine and its derivatives have documented immunomodulatory properties that constitute the basis of their continued use in autoimmune conditions including lupus nephritis and rheumatoid arthritis. If the yellow skin discoloration and liver involvement observed in some cases represent an immune-mediated sequela of novel coronavirus infection rather than direct viral hepatotoxicity, quinine's immunomodulatory effects could plausibly have contributed to recovery in such cases.

Finally, malaria was endemic and documented as a comorbidity (the the disease itself was sometimes understood as a "malarial fever" [*maliariinaia likhoradka*]);[67] in such cases quinine may have improved symptoms by treating concurrent infection rather than the primary (novel) diagnosis.

Nevertheless, malaria remains a possibility as the identity of the disease itself: a recent historical study by Ryazantsev & Smirnov, which contends colonial officials were right the first time when they characterized the disease as a "malarial fever" in early reports, and that the seeming continuity between the outbreak in Bukhara and the subsequent global pandemic is coincidence.[68] They propose cattle spillover in the Tomsk region of Siberia as the actual origin of the pandemic, rather than Bukhara.[69] Our understanding of the illness's clinical presentation (based on partially overlapping sources on the Russian side) matches theirs, and this alternative explanation deserves to be taken seriously. However, this scenario would require the connection between the mid-1880s Qarshi outbreak and the 1889 pandemic asserted in the primary sources to be erroneous; for the trajectory of the illness out of Bukhara along the newly-constructed railroad to be unrelated; and for the particular strain of malaria (presumed to be P. Falciparum) to be uncharacteristically virulent in adults.

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6. Discussion: Implications for Pathogen Attribution

6.1 A Predictive Framework: What a Pre-Pandemic Coronavirus Spillover Would Look Like

Before assessing the Bukharan evidence, we propose an explicit predictive framework. A coronavirus jumping from an animal reservoir into humans does not arrive pandemic-ready; it passes through recognizable stages of host adaptation. The closest real-world template is MERS-CoV: a betacoronavirus with a camelid reservoir, approximately 35% case fatality rate in humans, multisystem involvement including documented jaundice and acute hepatic injury alongside respiratory disease, and—critically—still essentially stuck in the early stages of this adaptation process today, with no sustained human-to-human transmission. The evolutionary trajectory for coronaviruses more broadly runs from broad multi-organ tropism in the animal host toward increasingly efficient airway replication in a new host.[70]

A spillover event at Stage 1 would present in the historical record as: high-mortality illness among individuals with direct animal contact; geographic clustering reflecting the patchy distribution of the animal reservoir rather than human-to-human transmission chains; broad organ tropism including hepatic involvement; and limited secondary spread. Stage 2 would show: occasional secondary cases from close contact; small clusters that burn out; declining mortality as viral doses in human-to-human transmission are lower than direct animal-contact doses; and continued hepatic features attenuating as respiratory tropism strengthens. Stage 3, the pandemic-capable strain, would show: efficient respiratory transmission; substantially reduced CFR; and largely shed hepatic features, though residual multisystem involvement might remain as a clinical distinguishing mark.

The prediction this framework generates for the historical record is precise: if this was a coronavirus spillover evolving toward respiratory pandemic efficiency, we would expect to find, in the pre-pandemic record, *high-mortality hepatic-respiratory illness among people with dense ruminant contact in a pastoral Central Asian setting, geographically clustering around the animal reservoir, with the eventual pandemic strain showing notably attenuated hepatic involvement relative to the pre-pandemic cases.*

The Bukharan archival evidence, which we present without having constructed from this framework, maps onto this prediction with some fidelity. The 1884–1888 Qarshi phase: high mortality, narrow geographic clustering (only Qarshi city, villages unaffected unless residents traveled there), prominent hepatic and cyanotic signs, and high case fatality in the documented village anecdote (79 of 80 visitors died). The 1889 Bukharan phase: broader spread, lower proportional mortality (5% CFR, still high by comparison to the eventual global pandemic average, but dramatically lower than the Qarshi anecdote), onset of the respiratory/neurological complex that eventually became the global pandemic's dominant signature. The limited household transmission reported in Jizakh is consistent with Stage 2—still insufficient for efficient spread, but no longer purely zoonotic.

We present this framework not as established fact but as a structured hypothesis that the archival evidence is consistent with, and that motivates the specific predictions we ask future archival and phylogenetic work to test.

For influenza: H3N8 has constitutive respiratory tropism. Initial spillover events do not typically present as GI-or hepatic-predominant, even in naïve populations. The pattern—jaundice and cyanosis as salient prognostic signs in early cases, respiratory symptoms becoming dominant later—runs opposite to how influenza typically behaves. Furthermore, small ruminants including sheep are not established influenza A reservoir hosts, and the intensive karakul sheep herding context of the Bukharan oasis does not map onto the waterfowl-agriculture interface typically associated with influenza emergence.

On the CFR gap: The roughly 5% case fatality in Bukhara stands in stark contrast to the global pandemic CFR of 0.1–0.28%—a 20–50-fold difference. This gap is most parsimoniously explained within the spillover framework by high initial virulence in an immunologically naïve population at the origin site, attenuating as the virus adapted to human-to-human transmission and spread to populations who may have had partial cross-reactive immunity. The alternative explanations—Klemm's denominator undercounting exposures, or concurrent illness in an already-compromised population—are possible but do not require discarding the spillover model.

6.2 Limitations

These records do not definitively identify the pathogen. We lack virological confirmation so can only note that the clinical phenotype is less typical for influenza than for coronavirus, and that none of the contemporaneous Russian records invoke the former. The seroarchaeological evidence advanced by Worobey and colleagues—that birth-cohort mortality patterns in 1918 are consistent

with H3 antigenic imprinting from childhood exposure in the early 1890s—is genuinely compelling evidence for widespread H3 exposure in that era. However, it is difficult to disentangle the theorized effects of imprinting from the equally plausible persistent effects of a novel coronavirus infection on cardiopulmonary development in utero or infancy (e.g., potentially further excacerbated by toxic gas exposure during World War I). More fundamentally, the seroarchaeological evidence demonstrates H3 *exposure* in birth cohorts of the early 1890s; it does not establish that an H3 *pandemic* caused the 1889 outbreak. Concurrent seasonal H3 influenza circulation alongside a pandemic coronavirus is consistent with this evidence.

That said, documentation bias is a serious competing explanation for the GI-to-respiratory shift: scribes may have recorded dramatic GI signs early on (unusual, noteworthy) and respiratory signs later (more 'typical,' less noteworthy). We address this as follows: (1) cyanosis and jaundice are described as prognostic markers across multiple independent reporters, suggesting they were clinically salient features, not incidental observations; (2) the shift toward respiratory dominance tracks geographic spread rather than time alone (more prominent in Samarqand and Jizakh than in Bukhara), which is more consistent with viral evolution than a uniform documentation artifact; (3) the Jizakh physician's prolonged-course description (15–40 days of daily fever) is not consistent with the acute respiratory presentation typical of influenza, even in a later outbreak phase. We acknowledge that the documentation-bias explanation cannot be excluded on the basis of these sources alone.

Crucially, geographic spread (different populations with different illness patterns) versus temporal viral evolution remains difficult to disentangle. Finally, we have no individual-level patient data, only aggregate outbreak descriptions at village and city level, with temporal resolution in weeks to months, not days.

6.3 The Molecular Dating Controversy and Implications for These Data

The coronavirus hypothesis has been supported in part by phylogenetic molecular dating, which suggested the most recent common ancestor of HCoV-OC43 and BCoV diverged in approximately the 1880s–1890s. However, recent reanalysis by Shaw and Gatherer (2023) has contested these dates, finding the most probable MRCA falls in approximately 1898–1902—a decade too late for direct compatibility with the 1889 pandemic—while noting that 1884–1889 remains within the 95% credible intervals of most datasets analyzed.[71]

To the best of our knowledge, though, virologists have not conducted similar studies on sheep or other predominant regional livestock that were plausible intermediate host reservoirs leading to the 1889 pandemic. Moreover, it is well established is that BCoV infects sheep and that the broader embecorvirus family shows considerable host promiscuity.[72] As we have seen from SARS-CoV-2, tracing local animal husbandry practices and trade markets is often the key to understanding pandemic emergence.[73] Similarly, repeated spillovers of SARS-CoV-2 from humans into deer populations, followed by sustained adaptation in that host, demonstrate that anthroponotic transmission

— from humans to animals — can occur and leave lasting phylogenetic traces. This possibility has not been adequately considered in the BCoV/HCoV-OC43 divergence literature: if an early human OC43 lineage seeded bovine populations rather than vice versa, the conventional interpretation of molecular dating would require revision.[74]

6.4 Why These Data Matter

Despite these limitations, the Bukharan sources fill a documented gap in the historical record. The 1889 pandemic's clinical signatures from the initial outbreak regions are poorly characterized in readily accessible sources. These records provide:

- Contemporaneous clinical observations from the probable geographic origin point
- Specific, observable clinical signs (jaundice, cyanosis) documented independently across Russian and Bukharan sources
- Evidence that the illness appeared novel to a population familiar with endemic fevers, malaria, and cholera
- A temporal arc from initial spillover (high mortality, geographic clustering, hepatic and cyanotic signs) to increasingly efficient human-to-human transmission (broader spread, lower relative CFR, emergent respiratory dominance) that is consistent with the three-stage model in Section 6.1
- Documentation of the animal husbandry context (karakul sheep herding) that is both epidemiologically distinct from the waterfowl narrative and specifically inconsistent with the influenza reservoir ecology

For researchers integrating historical and phylogenetic evidence to constrain hypotheses about 1889 origins, these sources offer a dataset that can inform epidemiological modeling and hypothesis refinement.

The temporal claim also has broader implications. If the spillover began as early as 1884, this undermines the hope that coronavirus epidemics quickly resolve into endemic equilibrium—suggesting instead a multi-year adaptation period with sustained high mortality before the virus achieved pandemic-capable respiratory transmission. In that era, immunosuppression was largely limited to the modulating effects of helminthic coinfection, which complicates the emerging narrative that antigenic leaps occur solely from chronic infection in immunocompromised hosts. The pattern suggests iterative spillback between humans and ruminants, with potentially multiple transmission pathways (respiratory, oral-fecal, and milk products), and a lengthy pre-pandemic phase during which the virus was geographically constrained before a superspreading event enabled efficient respiratory transmission and global spread.

At a minimum, the Kyzylkum desert and Bukharan oasis merit closer ecological and viral surveillance.[75] Specific targets might include ruminant coronavirus serology to assess current diversity in karakul and related sheep breeds, and phylogenetic sampling of contemporary ovine and camelid coronaviruses in Uzbekistan and adjacent areas to characterize their relationship to known em-

becorvirus lineages. Soviet land management and livestock practices since the 1920s may limit contemporary inference, but they do not eliminate the ecological rationale for surveillance. Computational modeling of geographic spillover risk has advanced considerably for bat-associated sarbecoviruses, but no analogous framework yet exists for embecoviruses, whose rodent reservoir and ruminant amplification ecology occupies a distinct niche — and likely a distinct spatial distribution — that the evidence presented here may help motivate.[76]

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7. Conclusion

We present newly translated and systematized primary sources from the Bukharan administrative archive documenting an outbreak beginning in 1884 and continuing through 1889–1890. We interpret this evidence through a three-stage model of coronavirus host adaptation, the closest real-world template for which is MERS-CoV: a betacoronavirus with a camelid reservoir, multisystem tropism including hepatic involvement, high initial case fatality, and limited human-to-human transmission. This model generates a precise prediction for what a pre-pandemic spillover would look like in the historical record, and the Bukharan evidence is consistent with that prediction: high-mortality hepatic-respiratory illness geographically clustering around a ruminant herding center, transitioning toward broader spread and respiratory dominance as the virus adapted to human transmission.

The clinical pattern—GI and hepatic predominance in initial village outbreaks, transitioning toward respiratory dominance in urban transmission—is more consistent with coronavirus spillover dynamics than with typical influenza presentation. The animal husbandry context (karakul sheep herding and fur trade) provides an epidemiologically distinct ecological niche from the waterfowl-agriculture narrative typically associated with influenza emergence.

We have engaged directly with the principal competing evidence and hypotheses: the quinine efficacy data (interpretable as treating comorbid malaria, but also consistent with empirical general febrile illness practice of the era); the documentation-bias alternative for the GI-to-respiratory clinical shift; the contested OC43 molecular dating; and the H3N8 seroarchaeological evidence (compelling as evidence of H3 exposure, but not sufficient to establish pandemic causation). None of these, individually or collectively, constitutes a refutation of the coronavirus hypothesis; each represents an evidentiary caveat that the field should assess.

We deliberately avoid claiming to settle the 1889 identity question. Instead, we offer this material as a curated historical dataset for researchers integrating evidence across disciplines—virology, phylogenetics, epidemiology, and history. We welcome integration with genomic, phylogenetic, and comparative epidemiological analyses by those with expertise in these domains.

For now, these archival sources stand as evidence of a pandemic moment when transmission efficiency was evolving, tissue tropism was shifting, and a human population—documented in remark-

able detail by administrators and physicians on two sides of a colonial boundary—experienced a disease of uncertain but increasingly respiratory character.

Notes

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[2]Alain-Jacques Valleron et al., "Transmissibility and Geographic Spread of the 1889 Influenza Pandemic," *Proceedings of the National Academy of Sciences* 107, no. 19 (2010): 8778.

[3]Valleron et al., "Transmissibility," 8779.

[4]Michael Worobey et al., "Genesis and Pathogenesis of the 1918 Pandemic H1N1 Influenza A Virus," *Proceedings of the National Academy of Sciences* 111, no. 22 (2014): 8107–8112.

[5]Harald Brüssow and Lutz Brüssow, "Clinical Evidence That the Pandemic from 1889 to 1891 Commonly Called the Russian Flu Might Have Been an Earlier Coronavirus Pandemic," *Microbial Biotechnology* 14, no. 5 (2021): 1860. https://doi.org/10.1111/1751-7915.13889.

[6]For a broader discussion of the historical implications of the 1889 pandemic, see James Pickett, *Russian Subject, Muslim Monarch: Sovereignty and Scribal Culture in Colonial Bukhara* (Cornell University Press, 2026), ch. 3.

[7]O‘zR MA i1-29-897, ff. 3a, 41a; i22-1-404, ff. 1a–b; i21-1-69, ff. 6a–b; i126-1-1896, ff. 3a–4a (fever); i22-1-404, ff. 1a–b; i126-1-1896 (1888–9), ff. 8, 10 (chills); i22-1-404, ff. 1a–b; i1-29-897, f. 41b (paroxysm); i126-1-1896, f. 5 (weakness); i126-1-1896 (1885–6), f. 7 (heaviness); i22-1-404, f. 2a; i126-1-1896, ff. 5, 10 (skin discoloration and cyanosis); i22-1-404, ff. 2a–b (edema); i126-1-1896, f. 5; i1-29-897, f. 41b (gastrointestinal symptoms); i21-1-69, ff. 6a–b (liver, spleen, and neurological involvement).

[8]For instance: O‘zR MA i126-1-1896, ff. 8, 10; O‘zR MA i126-1-1896, f. 5.

[9]"Po raportu nachal'nika Zeravshanskogo okruga o svirepstvueshchei v doline Zeravshana likhoradke," O‘zR MA i1-16-436, ff. 1a–b.

[10]Armenius Vambery, *Travels in Central Asia; Being the Account of a Journey from Teheran Across the Turkoman Desert on the Eastern Shore of the Caspian to Khiva, Bokhara, and Samarcand* (Harper & Brothers, 1865), 263. See also Jürgen Paul, "Nomads and Bukhara. A Study in Nomad Migrations, Pasture, and Climate Change (11th Century CE)," *Der Islam* 93, no. 2 (2016): 495–531, https://doi.org/10.1515/islam-2016-0039; C. C. Young, "Origin of Karakul Sheep," *Journal of Heredity* 5, no. 10 (1914): 445–447, https://doi.org/10.1093/oxfordjournals.jhered.a107768.

[11]Colin J. Carlson et al., "Pathogens and Planetary Change," *Nature Reviews Biodiversity* 1 (2025): 32–49. https://doi.org/10.1038/s44358-024-00005-w.

[12]Anastasia N. Vlasova and Linda J. Saif, "Bovine Coronavirus and the Associated Diseases," *Frontiers in Veterinary Science* 8 (2021): 643220. https://doi.org/10.3389/fvets.2021.643220.

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[14]O‘zR MA i1-29-897 (8 September 1889), f. 41b.

[15]AVPRI 147-485-319, f. 12a; O‘zR MA i1-29-897, f. 41a; O‘zR MA i22-1-404, ff. 1a–b.

[16]"O sobranii svedenii o karshinskom bekstve," O‘zR MA i22-1-404, ff. 1a–b.

[17]O‘zR MA i22-1-404, ff. 1a–b; O‘zR MA i1-29-897, f. 41b.

[18]O‘zR MA i22-1-404, ff. 2a–b.

[19]O‘zR MA i22-1-404, f. 3a.

[20]O‘zR MA i22-1-404, f. 3a; O‘zR MA i1-29-897 (8 September 1889), f. 41a.

[21]"Ariza bukharskikh chinovnikov emiru o vspyshkakh epidemii kholery i ospy i ob''iavlenii karantina v khanstve," O‘zR MA i126-1-1896, ff. 3a–4a.

[22]O‘zR MA i1-29-897, f. 41a; O‘zR MA i126-1-1896, f. 5.

[23]O‘zR MA i126-1-1896 (1888–9), ff. 8, 10.

[24]O‘zR MA i126-1-1896 (1885–6), f. 7.

[25]O‘zR MA i126-1-1896, f. 5; Saifiddin Nazarzoda, ed., *Farhang-i Tafsīrī-yi Zabān-i Tājīkī* (Dushanbe: Akadīmīya-yi ᶜIlm ho-yi Jumhūrī-yi Tājīkistān, 2008), 728.

[26]O‘zR MA i126-1-1896, f. 5 (seal dated 1302 h. / 1884–5); O‘zR MA i126-1-1896, f. 10.

[27]O‘zR MA i22-1-404, ff. 1a–b; O‘zR MA i1-29-897, f. 41b; Kucshelevskii, *Materialy*, 37.

[28]"Perepiska s bukharskim polit-agenstvom i drugimi o bor'be s likhoradkoi svirepstvuiushchei v Bukharskom khanstve," O‘zR MA i1-29-897, f. 42a. This report (ff. 41a–48a) is duplicated in "Sanitarnoe sostoianie Bukhary," AVPRI 147-485-319, ff. 8a–19b.

[29]O‘zR MA i1-29-897, f. 42a.

[30]O‘zR MA i1-29-897, ff. 42b–43a. See also Sergey V. Ryazantsev and Alexey V. Smirnov, "Epidemiia v Bukhare – Nachalo Pandemii 'russkogo Grippa' 1889–1890 Gg.? (Sotsial'no-Demograficheskoe Issledovanie)," *Bylye Gody* 18, no. 1 (2023), 356. https://doi.org/10.13187/bg.2023.1.353.

[31]O‘zR MA i1-29-897, f. 43b; O‘zR MA i22-1-404, ff. 1b–2a.

[32]O‘zR MA i1-29-897, ff. 43b–44a.

[33]O‘zR MA i22-1-404, f. 40a; O‘zR MA i22-1-404, f. 49a.

[34]O‘zR MA i1-29-897, ff. 1a, 3a.

[35]O‘zR MA i1-29-897, f. 56a (intelligence report dated 1 October 1889); O‘zR MA i1-29-897 (8 September 1889), f. 44b.

[36]O‘zR MA i1-29-897, ff. 43b–44a; O‘zR MA i1-29-897, f. 44a.

[37]AVPRI 147-485-319, f. 43b.

[38]This percentage comes from one of the few village-level reports that disaggregated between old and young. O‘zR MA i21-1-69, fols. 8a-30a.

[39]O‘zR MA i1-29-897, f. 26a.

[40]O‘zR MA i1-29-897, f. 46a.

[41]O‘zR MA i1-29-897, ff. 3a, 41a.

[42]O‘zR MA i22-1-404, ff. 25a–26a.

[43]O‘zR MA i1-29-897 (8 September 1889), f. 41b.

[44]O‘zR MA i22-1-404 (25 June 1889), f. 1a; O‘zR MA i1-29-897 (8 September 1889), f. 41b.

[45]O‘zR MA i1-29-897, ff. 43b–44a.

[46]O‘zR MA i1-29-897, f. 27a.

[47]O‘zR MA i1-29-897 (8 September 1889), f. 46b.

[48]O‘zR MA i22-1-404 (29 July 1889), f. 4a.

[49]O‘zR MA i22-1-404 (29 July 1889), f. 4b.

[50]O‘zR MA i1-29-897 (8 September 1889), f. 41b.

[51]O‘zR MA i1-29-897 (8 September 1889), f. 46b.

[52]“Delo o smertnosti sredi naseleniia goroda Dzhizaka ot 5 do 15 chelovek v den’, v sviazi zabolevaniem mestnoi likhoradkoi,” O‘zR MA i21-1-69, ff. 6a–7b, 30a.

[53]O‘zR MA i21-1-69, f. 1a.

[54]O‘zR MA i21-1-69, ff. 6a–b.

[55]O‘zR MA i21-1-69, f. 7a; O‘zR MA i21-1-69, ff. 8a–9a.

[56]“Delo o smertnosti sredi naseleniia goroda Dzhizaka ot 5 do 15 chelovek v den’, v sviazi zabolevaniem mestnoi likhoradkoi,” O‘zR MA i21-1-69 (19 September 1889), f. 11b.

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[59]AVPRI 147-485-319, f. 12a.

[60]O‘zR MA i22-1-404, f. 41b.

[61]O’zR MA i126-1-1901, f. 1a; Mustakim Arıcı, “Silent Sources of the History of Epidemics in the Islamic World: Literature on Ṭāᶜūn/Plague Treatises,” trans. Faruk Akyıldız, *Nazariyat* 7, no. 1 (2021): 138. https://doi.org/10.12658/Nazariyat.7.1.M0132en. See also James Pickett, *Russian Subject, Muslim Monarch: Sovereignty and Scribal Culture in Colonial Bukhara* (Cornell University Press, 2026), ch. 3.

[62]O’zR MA i1-29-897 (8 September 1889), f. 45b.

[63]O’zR MA i1-29-897 (8 September 1889), f. 45b.

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