Hypothesis

Medical Hypothesis: Respiratory Epidemics and Pandemics Without Viral Transmission

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Summary

The spatiotemporal all-cause mortality (weekly time resolution, >100 jurisdictions) during the Covid period (the period of the declared pandemic, 2020-2023) disproves that the excess deaths could have been caused by the spreading contagion of any novel virus or its postulated variants (Rancourt et al., 2024).

What then caused the estimated 31 million excess deaths worldwide (Rancourt et al., 2024)?

In this paper, I systematically present many facets (based on the existing scientific literature) of my overall hypothesis whereby the Covid-period pandemic of mortality was a pandemic of transmissionless self-infection bacterial pneumonias induced by biological stress (in the sense of medical researcher Hans Selye, which includes psychological stress) arising from the coordinated and largescale mandates, measures, so-called responses, and medical assaults including testing, diagnostic bias, isolation, denial of treatment (especially antibiotics for pneumonia), mechanical ventilation, sedation, experimental and improper treatments, and vaccination.

Transmissionless self-infection bacterial pneumonias are normally prevalent in the geriatric context, where they are known as aspiration pneumonia. Transmissionless pneumonias do not rely on person-to-person transmission or contagion but rather are associated with the stressed or assaulted respiratory tract microbiome.

My contribution is to advance that the likelihood of fatal transmissionless pneumonias in the elderly and persons with comorbidities increases significantly with environmental changes or assaults that cause biological stress, and to describe several mechanisms. My hypothesis is that this proposed phenomenon is amply sufficient to cause epidemics, pandemics and seasonal mortality, always targeting the frail and sick, and that Covid was exactly such a case, completely caused by institutions and governments.

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1. Introduction

The dominant (industry-promoted) paradigm of a pandemic is that a novel virulent pathogen emerges randomly or by design and spreads from person to person to many places, causing death. The latest twist is that the said novel pathogen immediately spawns genetic variants of itself, causing cascading pandemics in succession over the same territories visited by the parent pathogen.

The long-term so-called solution advanced by the industry and governments is to constantly vaccinate entire populations, repeatedly, to boost immunity and to address the new variants.

The end result of permanent national COVID-19 vaccine-campaign dependence resembles a protection racket. The yearly cost of these continuing vaccinations from public funds during the Covid period (2020-2023) was comparable to USA arms sales to its so-called allies.

In fact, the novel-viral spread theory of the declared Covid pandemic is disproved by a large amount of spatiotemporal all-cause mortality data, with weekly resolution in hundreds of jurisdictions (0).

What then caused the large excess all-cause mortality worldwide during the Covid period (31 million excess deaths worldwide, Rancourt et al., 2024), and what generally causes pandemics?

Here I show that current and growing scientific knowledge published in leading journals recognizes the prevalent phenomenon of transmissionless spontaneous self-infection bacterial pneumonia. Self-infection bacterial pneumonia does not rely on person-to-person transmission but rather is associated with the stressed or assaulted respiratory tract microbiome. This is related to the area of medicine known as aspiration pneumonia, and such self-infection is consistent with the epidemiology of tuberculosis.

The somewhat geriatric-medicine-siloed phenomenon of prevalent aspiration pneumonia is confirmed by classic microbial methods and is augmented by genomic studies of the respiratory tract microbiome. A picture emerges in which stress and assaults can cause a rapid self-organized and deleterious imbalance of the respiratory tract microbiome that is fatal in the elderly or in persons with comorbidities.

This does not mean that transmission of bacterial pneumonia is not a real phenomenon; only that it is not necessary to produce epidemics or pandemics occurring in response to imposed biological stress and widespread environmental or medical assaults.

My hypothesis in this paper is to develop many facets whereby it is reasonable to conclude that the 2020-2023 Covid-period pandemic of mortality was misdiagnosed and was a pandemic of transmissionless bacterial pneumonias induced by the biological stress (which includes psychological stress) from the coordinated and largescale mandates, measures, so-called responses, and medical assaults including testing, diagnostic bias, isolation, denial of treatment (especially antibiotics for pneumonia), mechanical ventilation, sedation, experimental and improper treatments, and vaccination. Compelling arguments in this direction were previously made by Rancourt et al. (2021a) based on USA data.

I argue that the same framework is consistent with what is known of all pandemics in history, always involving tremendous episodes of hardship and environmental and societal assaults, generalized to include skin and digestive tract microbiomes and parasites. The crux of my hypothesis also explains the synchronous seasonal patterns of large winter mortality, quantitatively observed for more than a century.

The idea that epidemics, pandemics and seasonal mortality are due to environmental and health-status factors (often discussed in terms of so-called terrain theory vs germ theory) rather than contagion is not new. In fact, it is as old as epidemiology itself ("epidemic constitutions of the atmosphere", Sydenham, 1676). My contribution here is to postulate about the disease-producing mechanisms at the interface between the individual's total environment (or total field, in the sense of behavioral psychology, Lewin, 1951) and the body, mechanisms that involve a person's microbiomes and parasites.

Section 0 describes empirical data that constrains the interpretations and hypotheses. Section 0 presents the many facets of my hypothesis. Section 0 is a conclusion.

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Researchers and I at CORRELATION and collaborators continue to be engaged in a broad research program of all-cause mortality and its associations with various factors: https://correlation-canada.org/research/

2. An epidemiological phenomenon in need of explanation

The following is a description of a particular phenomenon that motivates me to advance hypotheses about one or several of its contributing causes, and to eliminate other potential causes:

I. A peak in excess all-cause mortality surges synchronously with the World Health Organization (WHO) 11 March 2020 declaration of a pandemic, in many national and sub-national jurisdictions (especially in the Northern Hemisphere).
II. The said peak in excess all-cause mortality (nominally March-May 2020) does not occur in many (most) jurisdictions, including in the Northern Hemisphere.
III. There are no similar peaks occurring prior to 11 March 2020, and there is essentially no detected excess all-cause mortality prior to approximately 11 March 2020 (in many years of data, in >100 countries).
IV. The magnitude of the said peak in excess all-cause mortality, normalized by jurisdictional population, is highly heterogeneous across jurisdictions in which it occurs, and it is often zero (i.e., undetected).
V. There is contrary evidence of spatiotemporal spread of excess mortality in these peaks. All the said peaks occur essentially at the same time, irrespective of their differing magnitudes (normalized by jurisdictional population). The said peaks in excess all-cause mortality do not cross jurisdictional borders, do not grow into new geographic regions, and do not systematically have rising edges delayed in time as one moves away from the strongest centers of large-magnitude said peaks.
VI. High heterogeneity across jurisdictions of the magnitude of the said peak also occurs when the said peak is normalized by expected or historic baseline mortality under the peak (i.e., when it is expressed as a P-score), instead of by jurisdictional population. Since P-score excess mortality is measured in comparison to the intrinsic baseline mortality rate (by time period) in the jurisdiction, peaks expressed as P-scores include all the factors normally affecting mortality, such as age structure and prior health status. P-score mortality is thereby age and health-status adjusted.
VII. Normal mortality rates are generally exponential with age. Likewise, the mortality in the said peak is predominantly from the elderly population.
VIII. The most intense said peaks, where they occur, occur in urban regions with high poverty and high population density. However, for example, the said peak is essentially absent from Eastern European countries.
IX. The excess all-cause mortality in the said peak, in well-documented cases, is quantitatively equal to or closely matched by the number of tabulated respiratory deaths, assigned as COVID-19 deaths, in the same time period, in the given jurisdiction.

The above-described phenomenon is well documented in many scientific reports, such as in the extensive work done by me and my collaborators (Hickey et al., in preparation; Johnson and Rancourt, 2022; Rancourt, 2020, 2022; Rancourt et al., 2020, 2021a, 2021b, 2022a, 2022b, 2022c, 2023a, 2023b, 2024; Rancourt and Hickey, 2023) (and in references therein).

3. What are possible causes and what are causes that can be ruled out?

In this section, I describe my present understanding of the causes of death to explain excess mortality during the Covid period, by presenting hypotheses that are either disproved (0) or consistent with (0, 0, 0, 0, 0, 0, 0) the observed excess all-cause mortality (such as that described in Section 0).

In so doing, I adopt, adapt and augment the 18-page discussion about Covid-period all-cause mortality from section 5.12 of Rancourt et al. (2024). Much of the text is taken directly from Rancourt et al. (2024), and repeated because of its relevance and importance, and to create a coherent picture.

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In particular, following Rancourt et al. (2024), I make the important distinction (Section 0) between:

- a primary cause of death, and
- an associated or proximal cause of death.

Without this fundamental distinction between primary and proximal causes of death, the subject, in my view, is too imprecise to produce any useful insights.

3.1. Primary cause of death versus associated diseases or conditions

> "Some diseases have specific causes, the direct actions of certain particular, disease-producing agents, such as microbes, poisons, or physical injuries. Many more diseases are not caused by any one thing in particular; they result from the body's own response to some unusual situation."
>
> — Hans Selye (Selye, 1956; p. 179)

Given the difficulty identified by Selye, in our analysis, I (with Rancourt et al., 2024) distinguish between a "primary cause of death" (hierarchical top-level circumstances or stressors or assaults that cause an early or accelerated death, irrespective of the mechanism) and "associated diseases or conditions reported at death", for example, on a clinical death certificate (proximal or clinical causes of death in the final moments, involving organ and organ system failures or stoppages).

For example, hypothetically, infection by a specific deadly pathogen could be a primary cause of death, where the deaths, in different individuals, might be concomitantly associated with generic conditions such as "respiratory infection" and "heart failure", and sub-categories of such conditions.

In other examples:

- A poison leads to heart failure. Then the primary cause of death is the poison.
- A poison weakens the body's defenses, leading to a massive intestinal infection and eventual respiratory and heart co-failures. Then the primary cause of death is the poison.
- Chronic biological stress causes exhaustion-phase collapse of the body's resistance to the biological stress, and death follows (Selye, 1956). Then the primary cause of death is what caused the said chronic biological stress, which can be specific.
- Chronic psychological stress causes immunosuppression, enabling a severe respiratory infection from ambient microbes and air pollution, followed by death. Then the primary cause of death is what caused the said chronic psychological stress, which can be specific.
- A person is seriously ill for some reason, even dying. A medical intervention accelerates the death, and the person dies prematurely. Then the primary cause of death is the medical intervention.

This distinction between primary and proximal causes of death aligns completely with the important conclusion of Link and Phelan (1995) who state:

> "we argue that social factors such as socioeconomic status and social support are likely 'fundamental causes' of disease that, because they embody access to important resources, affect multiple disease outcomes through multiple mechanisms, and consequently maintain an association with disease even when intervening mechanisms change."

3.2. Inferred respiratory medical conditions in the deaths contributing to Covid-period excess all-cause mortality

Prior to suggesting likely or possible primary causes of the deaths contributing to excess all-cause mortality, it is relevant to consider whether a dominant syndrome, disease or condition, based on reported symptoms and conditions prior to death, can be inferred to be associated with all or most excess all-cause mortality during the Covid period.

In the high-quality databases for the USA, there is a close match between the weekly reported COVID-19 mortality and weekly excess all-cause mortality, in the Covid period (2020-2022), including prior to and during the vaccine rollouts (CDC, 2023).

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To the degree that COVID-19 death assignation represents a serious respiratory condition at death, and given the intricate weekly temporal matching of the reported COVID-19 mortality and excess all-cause mortality for up to 3 years in the USA data, this represents strong evidence that respiratory infections were dominantly (virtually entirely) associated with the excess all-cause mortality.

Rancourt et al. (2021a) showed this in detail, into 2021 (their Figures 34a through 34i). They also pointed out that more than half of the deaths assigned as COVID-19 deaths could include life-threatening co-occurring bacterial pneumonia, according to CDC tabulations of death certificates, and that prescriptions of antibiotics were significantly reduced in the same period. Rancourt et al. (2021a) concluded:

> "Finally, our examination of plausible mechanisms for the exceptionally large COVID-era mortality in the USA, given all our empirical observations, leads us to postulate that COVID-19 may largely be misdiagnosed bacterial pneumonia (using a faulty PCR test: Borger et al., 2021; and see Ginsburg and Klugman, 2020), that correctly assigned bacterial pneumonia itself largely goes untreated, while antibiotics (and Ivermectin) are withdrawn, in circumstances where large populations of vulnerable and susceptible residents have suppressed immune systems from chronic psychological stress induced by ("COVID response") large-scale socio-economic disruption, and that the USA has, in the COVID-era, thus recreated the conditions that produced the horrendous bacterial pneumonia epidemic of 1918 (Morens et al., 2008) (Chien et al., 2009) (Sheng et al., 2011)."

For the present purposes, these results (CDC, 2023; Rancourt et al., 2021a) imply that essentially all excess all-cause mortality during the Covid period is associated with life-threatening respiratory infections or conditions, rather than mainly associated with other conditions such as cancer, suicide, drug overdoses, homicide, delayed medical interventions, and accidents.

As emphasized above (Section 0), a dominant association to a disease or condition is separate from the question of "primary cause of death".

Note that the proposed and apparent dominant respiratory disease or condition during the Covid period relates to excess all-cause mortality, and does not preclude specific primary causes of death in individuals leading to main associated conditions at death other than respiratory infections or conditions. Such cases could produce significant mortality too small to be detected as a discernable contribution to all-cause mortality. An example might be heart failure due to COVID-19-vaccine-induced myocarditis, in which the primary cause is the vaccine. Another example could be induced immunodeficiency enabling cancer. In both examples, the increased associations at deaths could be confirmed by clinical observation or autopsy, while being too small to be recognized features in all-cause mortality data.

The proposal that respiratory infections or conditions are the dominant (or a dominant) association with (not primary cause of) excess all-cause mortality during the Covid period is further supported by additional lines of evidence.

- (*Already outlined above:* Intricate weekly temporal matching of the reported COVID-19 mortality and excess all-cause mortality for up to 3 years during the Covid period in the USA data.)
- Intricate weekly temporal matching of the reported COVID-19 mortality and excess all-cause mortality for the March-May 2020 peak (the said peak of Section 0) in Canada and Canadian provinces (Rancourt et al., in preparation).
- Respiratory infections are a major recognized cause of death for all ages, both historically and presently, which is consistent with the intrinsic vulnerability of the lungs and the unavoidable rate of constant breathing (and aspiration, see Section 0).
- The median ratio of excess all-cause mortality to reported COVID-19 mortality in the Covid period (2020-2022) for some 100 countries is 1.55 (Rancourt et al., 2024), which is not too different from 1.
- Many deaths occurred from early aggressive hospital treatments for respiratory conditions, such as mechanical ventilators, toxic experimental doses of drugs, and lethal palliative drug cocktails (March-May 2020 said peak of Section 0) (e.g., Bailey and Köhnlein, 2020; Chaillot, 2024, their

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Chapter 6; Menage, 2021; Rancourt, 2020, 2023a; Richardson et al., 2020; Roedl et al., 2021; Torjesen, 2021; Watts et al., 2021).

- The VAERS system of adverse effect reporting contains many post-vaccination nominally COVID-19 infections (e.g., Hickey and Rancourt, 2022, their Figures S3(a) and S4(a)).
- Post-vaccination nominally COVID-19 infections are common, and are generally more frequent and more serious following multiple COVID-19 vaccine doses (1st dose, 2nd dose, booster) (Amer et al., 2024, their Table 6).

Furthermore, from a mechanistic point of view, all forms of bacterial pneumonia have unreasonably been overlooked and left untreated during the Covid period (Rancourt et al., 2021a).

In particular, as one example, tuberculosis (TB) is a category of ancient bacterial pneumonias, which is highly prevalent and deadly, which is a leading proximal cause of death in the world, and which is clearly linked to social and economic factors, especially household or residential living conditions.

In their section 5.4, Rancourt et al. (2024) linked a mortality discontinuity between Eastern and Western European countries during the Covid period to the post-1990s pre-Covid-period prevalence of active TB, as an indicator of or proxy for large socio-economic differences.

Apparent transmission of TB, from infectious persons to household members, is well established (e.g., Chapman and Dyerly, 1964), such that transmission in hospital, care-home and prison settings is expected and high prevalence and incidence are demonstrated (Baussano et al., 2010; Joshi et al., 2006). Turner et al. (2017) provide a recent review of potential transmission mechanisms.

Airborne transmission of TB has been definitively shown, from infected humans to laboratory guinea pigs and mice in close indoor quarters designed to share the same air (Dharmadhikari et al., 2011; Plumlee et al., 2021; Riley et al., 1959). The animals were presumably biologically stressed by their laboratory conditions.

One-third of the world population is estimated to be infected with the TB pathogen, 9–14 million confirmed active cases occur every year, and there are approximately 1.2 million confirmed TB deaths per year in regular recent times (Bagcchi, 2023; Dattani et al., 2023; Dye et al., 1999; O'Garra et al., 2013).

It is estimated that there may be up to 10 times more active pathological TB cases than the above reviewed numbers, not confirmed by bacteriological tests (e.g., Houben et al., 2022), which would represent approximately 100 million active cases in any year.

Therefore, it appears that humanity's evolutionary and pervasive coexistence with TB represents a large reservoir of potential respiratory ailments ready to enter the world stage whenever mass events suppress immunity (Sections 0 and 0), not counting the multitude of other prevalent bacterial, fungal and co-infection pneumonias (Dietert et al., 2017; Jacobs et al., 2015; Liu et al., 2023; Paquette et al., 2024; Torres et al., 2021).

In conclusion, it is plausible that virtually all or most excess all-cause mortality during the Covid period is associated with respiratory infections or conditions. This is likely to be the case in the USA, which is a large country with diverse populations of vulnerable individuals, living in diverse socio-economic, institutional, climatic and environmental conditions. It is also true for the March-May 2020 peak (said peak of Section 0) in Canada (Rancourt et al., in preparation). There is no counter evidence that this would not also be the case in other countries (Rancourt et al., 2024).

Therefore, primary causes of death in excess all-cause mortality during the Covid period (Section 0) should be consistent with this finding of respiratory condition prevalence, as a proximal or associated cause.

Regarding the particular microscopic pathogen to blame for the proximal respiratory conditions: You can only see what you look for (if the test is reliable), and you can't see what you don't look for. The medical hypotheses of the instant paper (Sections 0 and 0) provide more possible bacterial culprits than only those referred to above (in this Section 0).

A similar situation appears to apply to the 1918 pandemic, in which virtually all excess mortality was associated with respiratory conditions or infections (autopsy-confirmed as bacterial pneumonia: Chien et al., 2009; Morens et al., 2008; Sheng et al., 2011), whereas the primary cause of death in excess all-cause mortality would have been the conditions imposed by the First World War (Bailey et al.,

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2024), in the post-war socio-economic adjustment circumstances, with a strong association to poverty (Mamelund, 2006; Mamelund et al., 2021), in the context of a massive illicit drug trade in opium, morphine and heroin (literally tons of drugs) into the USA and European countries after the First World War, run by leading pharmaceutical companies such as Hoffman-La Roche at great profit (Braithwaite, 1984; Gøtzsche, 2013).

3.3. Primary causes of death in excess all-cause mortality during the Covid period

3.3.1. Ruled out: SARS-CoV-2 as a primary cause of death

The hypothesis of a specific spreading viral respiratory pathogen causing excess all-cause mortality during the Covid period (2020-2022) is contrary to and disproved by two main direct observations:

i. It is incompatible with the large country-to-country heterogeneity of age and frailty adjusted (P-score) excess all-cause mortality rate (e.g., Rancourt et al., 2024, their sections 5.5 and 5.7; and see COVID-19 Forecasting Team, 2022, re heterogeneity of IFR).
ii. It is incompatible with the country-to-country spatiotemporal pattern of deaths, including the phenomenon of not crossing national borders. There is no evidence of spread, only local (by country) and time-specific assaults that do not geo-temporally evolve (e.g., Rancourt et al., 2024, their sections 4.11, 4.12 and 5.6).

These basic incompatibilities have been previously reported (Rancourt, 2020, 2022; Rancourt et al., 2020, 2021a, 2021b, 2022a, 2022b, 2023a, 2023b, 2024). For example, in India there was no detectable excess all-cause mortality until the vaccine was first rolled out starting in March 2021 (Rancourt, 2022).

The said basic incompatibilities, while already proven, are corroborated in a high spatial resolution spatiotemporal hemispheric-scale study by Hickey et al. (in preparation), and for Canada (Rancourt et al., in preparation).

It is highly unlikely also that the frequently observed (in all countries, and repeatedly) peaks in excess all-cause mortality following the start or completion of COVID-19 vaccination rollouts, including boosters, occur from new variants of pandemic potency, for which no cross-immunity has been achieved (after more than one or two years of declared pandemic and typically more than one year of universal vaccination). For example, specific hypotheses of new variants of concern — corresponding to extraordinary coincidences following rapid vaccine rollouts — are argued to having been fabricated in the cases of India (Rancourt, 2022) and Australia (Rancourt et al., 2022a).

In addition to being strictly incompatible with definitive and repeated observations, the SARS-CoV-2 hypothesis (or the hypothesis of any specific pandemic-causing respiratory virus) as a primary (Section 0) cause of death during the Covid period is not needed to explain any feature of the all-cause mortality in any of the countries studied.

The more immediate hypotheses of biological stress, medical interventions, and vaccination largely suffice:

- There was biological (including psychological) stress, induced by aggressive and life-changing mandates.
- There were deadly medical interventions (including denial of treatment) and overreaction directed by institutional messaging and propelled by managerial and professional self-interest.
- There was a coordinated international so-called response and global vaccination campaign with multiple rollouts, driven and protected by aggressive establishment and industry forces (Bergman, 2024; Homburg, 2024; Kennedy, 2021; Von, 2022).
- All-cause mortality is a record of the consequences.

3.3.2. Hypothesis: Sudden and extraordinary stress from mandates and measures

It would be difficult to overestimate the importance of biological stress (which includes psychological stress) in causing death, irrespective of the mechanism.

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Selye (1956) defined biological stress and made an encyclopedic and systematic review of the many diverse stressors studied in the scientific literature up to 1976 (Selye, 1976a). Fatal physiological consequences of chronic biological stress have been known and extensively studied since their initial discovery (Selye, 1936, 1956, 1976a; Szabo et al., 2017).

While being foundational, the Selye line of research did not (Selye, 1976b) and has not generally (Szabo et al., 2017) included two paramount factors (see Rancourt, 2023b):

i. The role of social dominance hierarchy, in both human and animal societies, as a structural and leading source of complex, situational and time-dependent stressors (dominance aggression) that primarily determine an individual's (social-status-dependent) health and longevity.
ii. The dependence of biological adaptation to stress and failure (collapse) of biological adaption to stress on, not solely whether the biological stress is acute (episodic) or chronic (constant), and not solely on how the stress is experienced by the particular individual, but critically on the time sequence of the acting stressors of varying intensities; that is, on their time-type-intensity spectrum, which can have both regular and chaotic components.

More generally, as is now known, the health and survival of individuals among social animals, and primates in particular, is predominantly determined by the individual's position and role in the dominance hierarchy, in relation to the physiology and biochemistry of dominance aggression (Sapolsky, 2005), including via respiratory infections (Cohen et al., 1997a). Irrationality or randomness of the acts of dominance aggression plays an important role, via stress response mechanisms, and amplifies the harm to subordinate individuals (Silk, 2002).

Together, these studies (e.g., Selye, Sapolsky, Cohen, Silk) show that socially and environmentally mediated biological stress is a major determinant of death and survival. This is easily admitted for non-human animals. However, the current medical scientific literature generally shies away from admitting the prevalence of this brutality in human societies, and instead tends to focus on sanitized questions of immunology and individual psychological stress.

From the perspective of the immune system, ordinary psychological stress alone significantly impacts immune response, and psychoneuroimmunology is a large field of research (Ader and Cohen, 1993; Dhabhar, 2009, 2014). Psychological stress alone probably causes many varied and common diseases (Cohen et al., 2007; Morey et al., 2015). Psychological stress and social isolation have strong associations with respiratory infections, including the common cold, acting to increase both frequency and severity of the infection (Cohen et al., 1991, 1997a, 1997b). Meanwhile, the impact of age increases vulnerability to stress (Morey et al., 2015; Prenderville et al., 2015). "Immunosuppressive activity increases with aging", as the immune system is "remodeled" (Salminen, 2022). Early adversity, clinical diagnoses, and other factors also impact vulnerability to stress (Morey et al., 2015; Rancourt et al., 2024, their sections 5.4 and 5.10). Some aspects of psychological stress in the Covid period context have been reviewed by Peters et al. (2021).

Regarding stressors acting in the USA during the Covid period, and their social-status dependence, Rancourt et al. (2021a) summarized this way:

> "Therefore, it is not difficult to imagine that the massive socio-economic disruptions of the COVID-era would have caused undue chronic psychological stress and amplified dominance-hierarchy stress predominantly against those who are already at the bottom of the societal dominance hierarchy, and have the least means to adjust to dramatically new circumstances. The new circumstances include: loss of sources of income, both legitimate and illegal, increased social isolation, increased hierarchical impositions, constant fear propaganda, severe mobility restrictions, closing of public and corporate-public spaces previously used, enforcement and intimidation against private or informal gatherings, mobbing against those who do not cheerfully accept the 'new reality', and increased aggressions from equally stressed individuals. The missing means to adjust would include: undisturbed salary and ability to work from home, means to stay connected by Zoom (by video conferencing applications), large comfortable air-conditioned homes, means to home-school children in an adapted environment, nearby facilities for outside exercise, private

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facilities for physical exercise, undisturbed shopping by home delivery, undisturbed self-medication, continued access to health care, and so on."

Finally, a recent study of young mice is strikingly apropos. Li et al. (2023) found that mice subjected to a single episode of restraint and immobilization stress (single 5–20 h stress sessions of confinement) experienced severe immunosuppression, more so and differently than with other common laboratory stressors (cold, biochemical). The behaviourally stressed mice could not efficiently defend against intravenous challenge with bacteria, and also showed significant spleen macrophage cell death, among several other corroborating biochemical observations.

This study in mice (Li et al., 2023) suggests, on a mechanistic basis, that confinement and isolation in humans may have a significant negative effect on susceptibility to infections, consistent with field observations of college students (Cohen et al., 1991, 1997b), and non-human primates (Cohen et al., 1997a).

For a human society under lockdown, universal enforced confinement to one's home (or room in a care home), for example, will in-effect consist of very different confinement intensities depending on social class and financial means, for obvious reasons.

Regarding the deleterious health effects of immobility and confinement, it is not unrelated to point out that:

- Being connected to a mechanical ventilator or simply held at an ICU (intensive care unit) station are extreme immobilizations not unlike those imposed on the mice in the study of Li et al. (2023), but of longer duration on particularly fragile individuals.
- Contrary to caregiver expectations, medically ordered bedrest is virtually never beneficial to ill and recovering patients. In the words of Allen et al. (1999):

  "In 15 trials investigating bed rest as a primary treatment, no outcomes improved significantly and nine worsened significantly for some conditions (acute low back pain, labour, proteinuric hypertension during pregnancy, myocardial infarction, and acute infectious hepatitis)."
- Likewise, imposed low mobility and medically recommended bedrest is always harmful to elderly patients. In the words of Brown et al. (2004):

  "Conclusion: Low mobility and bedrest are common in hospitalized older patients and are important predictors of adverse outcomes. This study demonstrated that the adverse outcomes associated with low mobility and bedrest may be viewed as iatrogenic events leading to complications, such as functional decline."

I (with Rancourt et al., 2024) conclude this sub-section with the hypothesis that the Covid-period mandates and measures were a primary cause (Section 0) of death significantly contributing to the measured excess all-cause mortality. See: (Rancourt et al., 2021a, 2021b) (Rancourt et al., 2022b, their boxed figure in their Conclusion).

3.3.3. Hypothesis: Collectively amplified individual biological stress

The above (Section 0) considerations of Covid-context causes of biological stress include solely the unidirectional influence "stressor(s) → individual stress". However, in the circumstances of the Covid period, there must certainly also be a non-linear social transmission and amplification of biological stress (which includes psychological stress) and fear in the individual, presumably mediated (and mostly not attenuated) by mass media and government and health professional messaging.

I would argue that in the declared pandemic and massive pandemic so-called response context of the Covid period we must admit the likely importance of social transmission and social positive-feedback amplification of biological stress (which includes psychological stress) and fear. The known hormonal and brain circuitry responses to stress would mechanistically enable the said social transmission and social positive-feedback amplification (Rodrigues et al., 2009). The social transmission of physiologically detected stress in animal collectives is scientifically established

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(Brandl et al., 2022) and it is compatible with the hypothesis of social positive-feedback amplification of biological stress and fear.

When biological stress of the individual can result from or be increased by mass-communication-mediated non-linear social transmission and positive-feedback amplification, then epidemiological consequences including excess mortality can be sudden in any given population and synchronous over dispersed population centres.

3.3.4. Hypothesis: Assaults from extraordinary medical interventions other than COVID-19 vaccination

There is much evidence that medical interventions (including denial of treatment) other than COVID-19 vaccination caused premature deaths during the Covid period, which would not have occurred otherwise until later (e.g., Rancourt, 2020, 2023a; Rancourt et al., 2021a, 2022b, 2023a, 2024, and references therein).

In this regard, we must have the evidence-based perspective that, even in ordinary times, medicine itself is a leading cause of harm and death (Gøtzsche, 2012, 2016; Makary and Daniel, 2016; Panagioti et al., 2019; and references therein).

The said medical treatments would include:

- coordinated denial of antibiotics or Ivermectin against bacterial pneumonia
- systematic use of mechanical ventilators and their associated medications
- experimental treatment protocols (large-dose hydroxychloroquine, HCQ)
- new palliative and psychological medication protocols, overdoses (e.g., midazolam) (Marliot et al., 2020; Sy, 2024)
- isolation of vulnerable individuals in medical or institutional facilities
- denial of intensive care and disease management facilities
- denial of home and community care medical services
- aggressive-testing accidents
- accidents and infections from reduced attending staff in care homes
- encouraged voluntary or involuntary assisted dying (Marliot et al., 2020; Menage, 2021; Sy, 2024)
- increased prevalence of medical errors due to declared-pandemic conditions

The March-May 2020 peak often occurring in all-cause mortality (Section 0) (20-3 feature, section 4.2 in Rancourt et al., 2024), in particular, is difficult to explain absent medical interventions (Rancourt, 2020, 2023a). See also: Bailey and Köhnlein, 2020; Chaillot, 2024 (their Chapter 6); Richardson et al., 2020; Roedl et al., 2021; Torjesen, 2021; Watts et al., 2021.

I (with Rancourt et al.) conclude this sub-section (0) with the hypothesis that medical interventions (other than COVID-19 vaccination, including denial of treatment) were a primary (Section 0) cause of death significantly contributing to the measured excess all-cause mortality.

This medical intervention primary cause of death hypothesis is not invalidated by the background of biological stress that would have characterized the Covid period, since the relevant medical interventions accelerated the deaths that would not have otherwise occurred until later.

3.3.5. Hypothesis: Assaults by COVID-19 vaccination

Rancourt (2022) and Rancourt et al. (2022a, 2023a, 2023b, 2024) have shown many examples of strong temporal associations between rapid COVID-19 vaccine rollouts and peaks in excess all-cause mortality, in more than 100 countries and states, including in age stratified data.

Worldwide evidence showing that COVID-19 vaccination is associated with increased excess all-cause mortality, rather than reduced mortality, is highlighted by Rancourt et al. (2024, their section 5.11).

In that study of 125 countries, Rancourt et al. (2024, their Appendix B) observed the following specific associations between COVID-19 vaccine rollouts and peaks or increases in excess all-cause mortality:

i. *30% of countries (37 of 124) have no detected excess all-cause mortality in all of 2020, only later when vaccines are rolled out*

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- 124 countries had sufficient all-cause mortality data (the data for Cabo Verde was too noisy) to determine whether excess all-cause mortality started after the end of 2020, after the vaccines were introduced.
- Of these 124 countries, 37 countries (30% of countries) had no detectable excess all-cause mortality in 2020. For at least the first nine months of the declared pandemic (declared on 11 March 2020) these 37 countries had virtually no measurable excess all-cause mortality: Antigua and Barbuda, Australia, Barbados, Bermuda, Brunei, Cuba, Faroe Islands, Finland, French Guiana, French Polynesia, Gibraltar, Greenland, Hong Kong, Iceland, Jamaica, Japan, Macao, Malaysia, Martinique, Mauritius, Monaco, Mongolia, Namibia, New Caledonia, New Zealand, Norway, Philippines, Réunion, Saint Kitts and Nevis, Saint Vincent and the Grenadines, Seychelles, Singapore, South Korea, Suriname, Taiwan, Thailand, and Uruguay.
- To this list of 37 countries (Rancourt et al., 2024), we can add India (Rancourt, 2022).
- All these 37 + 1 countries have first peaks or increases in excess all-cause mortality (if present) occurring only after vaccination is initiated, or later when the bulk of injections have been administered and additional (booster) doses are rolled out (Rancourt et al., 2024; Rancourt, 2022).

ii. *100% of countries (110 countries with sufficient vaccination data) show varied associations between vaccine rollouts and excess mortality*

- 110 countries of the 125 countries in the Rancourt et al. (2024) study had sufficient data (both vaccination and mortality data, which is not too noisy) to allow determinations of temporal associations.
- There were significant associations between COVID-19 vaccine rollouts and peaks or increases in excess all-cause mortality in all 110 of these countries (100% of countries) having sufficient data (Rancourt et al., 2024, their Appendix B): Albania, Argentina, Armenia, Aruba, Australia, Austria, Azerbaijan, Bahamas, Barbados, Belgium, Belize, Bermuda, Bolivia, Bosnia, Brazil, Brunei, Bulgaria, Canada, Chile, Colombia, Costa Rica, Croatia, Cuba, Cyprus, Czechia, Denmark, Dominican Republic, Ecuador, Egypt, Estonia, Faroe Islands, Finland, France, French Guiana, French Polynesia, Georgia, Germany, Gibraltar, Greece, Guadeloupe, Guatemala, Hong Kong, Hungary, Iceland, Iran, Ireland, Israel, Italy, Jamaica, Japan, Jordan, Kazakhstan, Kuwait, Latvia, Lebanon, Liechtenstein, Lithuania, Luxembourg, Macao, Malaysia, Maldives, Malta, Mauritius, Mexico, Moldova, Monaco, Mongolia, Montenegro, Namibia, Netherlands, New Caledonia, New Zealand, Nicaragua, North Macedonia, Norway, Oman, Palestine, Paraguay, Peru, Philippines, Poland, Portugal, Puerto Rico, Qatar, Romania, Russia, Saint Kitts and Nevis, Saint Vincent and the Grenadines, Serbia, Seychelles, Singapore, Slovakia, Slovenia, South Africa, South Korea, Spain, Suriname, Sweden, Switzerland, Taiwan, Tajikistan, Thailand, Tunisia, Turkey, Ukraine, United Arab Emirates, United Kingdom, USA, Uruguay, and Uzbekistan.

iii. *97% of countries (113 of 116) show a late-2021 early-2022 peak in excess all-cause mortality temporally associated with booster rollouts*

- There were 116 of the 125 countries in the Rancourt et al. (2024) study that had sufficient and sufficient-quality data to ascertain the presence of the "22-0 feature", a prominent or statistically evident excess mortality peak that occurs within one month or so of 1 January 2022 (their section 4.5). Of these 116 countries, 113 countries had the 22-0 feature in their excess all-cause mortality data (their section 4.5). The other 3 countries did not measurably exhibit the 22-0 feature (Italy, Macao, Taiwan).
- Therefore, 113 of 116 countries (97% of countries) exhibit a peak in excess all-cause mortality within one month or so of 1 January 2022 (the 22-0 feature, Rancourt et al., 2024, their section 4.5) coincident with (immediately following) the time at which many booster doses were synchronously rolled out globally. The booster rollouts are recognized as peaks in overall (all doses) COVID-19 vaccine administration (e.g., Rancourt et al., 2023a).
- The 113 countries having discernable 22-0 features in excess all-cause mortality were (Rancourt et al., 2024, their Appendix B): Albania, Andorra, Argentina, Armenia, Australia, Austria, Azerbaijan, Bahamas, Barbados, Belgium, Belize, Bermuda, Bolivia, Bosnia, Brazil,

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Brunei, Bulgaria, Cabo Verde, Canada, Chile, Colombia, Costa Rica, Croatia, Cuba, Cyprus, Czechia, Denmark, Dominican Republic, Ecuador, Egypt, Estonia, Faroe Islands, Finland, France, French Guiana, French Polynesia, Georgia, Germany, Greece, Guadeloupe, Guatemala, Hong Kong, Hungary, Iceland, Iran, Ireland, Israel, Jamaica, Japan, Jordan, Kazakhstan, Kosovo, Kuwait, Kyrgyzstan, Latvia, Lebanon, Liechtenstein, Lithuania, Luxembourg, Malaysia, Maldives, Malta, Martinique, Mauritius, Mayotte, Mexico, Moldova, Monaco, Mongolia, Montenegro, Namibia, Netherlands, New Caledonia, New Zealand, Nicaragua, North Macedonia, Norway, Oman, Palestine, Panama, Paraguay, Peru, Philippines, Poland, Portugal, Puerto Rico, Qatar, Réunion, Romania, Russia, Saint Kitts and Nevis, Saint Vincent and the Grenadines, San Marino, Serbia, Seychelles, Singapore, Slovakia, Slovenia, South Africa, South Korea, Spain, Suriname, Sweden, Switzerland, Tajikistan, Thailand, Transnistria, Tunisia, Turkey, Ukraine, United Kingdom, USA, and Uruguay.

- Among these 113 countries having discernable 22-0 features in excess all-cause mortality, some of the most striking associations between a peak in vaccine rollout and the 20-2 feature occur for the 12 countries (Rancourt et al., 2024): Australia (and see: Rancourt et al., 2022a, 2023a, 2023b), Austria, Czechia, Hong Kong, Hungary, Poland, Qatar, Romania, Russia, Saint Vincent and the Grenadines, Slovakia, and Ukraine.

iv. *64% of countries (50 of 78) show a late-2022 early-2023 sharp peak in excess all-cause mortality temporally associated with booster rollouts*

- There were 78 of the 125 countries in the Rancourt et al. (2024) study that had sufficient and sufficient-quality data to ascertain the presence of the "23-0 feature", a prominent or statistically evident excess mortality peak that occurs within one month or so of 1 January 2023, less than 5 months prior to the declaration of 5 May 2023 of the end of the declared pandemic (their section 4.4).
- Of these 78 countries with sufficient data, 50 countries had the 23-0 feature (their section 4.4) in their excess all-cause mortality data. These 50 countries were (Rancourt et al., 2024, their Appendix B): Austria, Belgium, Canada, Chile, Croatia, Cyprus, Czechia, Denmark, Ecuador, Estonia, Finland, France, French Guiana, Germany, Greece, Guatemala, Hong Kong, Hungary, Iceland, Ireland, Italy, Japan, Latvia, Lithuania, Luxembourg, Macao, Moldova, Netherlands, New Zealand, Norway, Paraguay, Poland, Portugal, Puerto Rico, Qatar, Russia, Singapore, Slovakia, Slovenia, South Africa, South Korea, Spain, Sweden, Switzerland, Taiwan, Thailand, Tunisia, Turkey, United Kingdom, and USA.
- The other 28 of the 78 countries with sufficient data did not measurably exhibit the 23-0 feature (Rancourt et al., 2024, their section 4.4). These 28 countries were: Albania, Armenia, Azerbaijan, Bosnia, Colombia, Egypt, Faroe Islands, Georgia, Guadeloupe, Kazakhstan, Kosovo, Kyrgyzstan, Malaysia, Martinique, Mayotte, Mexico, Mongolia, Montenegro, North Macedonia, Oman, Philippines, Réunion, Serbia, Uruguay, Bulgaria, Mauritius, New Caledonia, and Romania.
- Therefore, 50 of 78 countries (64% of countries) exhibited a peak in excess all-cause mortality within one month or so of 1 January 2023 (the 23-0 feature, Rancourt et al., 2024, their section 4.4) coincident with (immediately following) the time at which many booster doses were synchronously rolled out globally, in the last booster rollout prior to the declaration of the end of the declared pandemic. The booster rollouts are recognized as peaks in overall (all doses) COVID-19 vaccine administration (Rancourt et al., 2024; e.g., Rancourt et al., 2023a).
- Although Bulgaria does not have the 23-0 feature (a distinct peak near 1 January 2023), it does have a broader and somewhat earlier peak structure in its excess all-cause mortality, which matches the vaccine rollout at that time (mid to end of 2022). Similar circumstances may be occurring in: Albania, Armenia, Colombia, Egypt, Georgia, Malaysia, North Macedonia, Philippines, Mauritius, and New Caledonia. (Rancourt et al., 2024, their Appendix B)

v. *Particularly striking examples of vaccine-mortality associations in several specific countries*

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- Several countries showed striking examples of vaccine-mortality associations in which the vaccine rollout is synchronous with the only exceptionally large excess all-cause mortality feature (Rancourt et al., 2024, their Appendix B): Bahamas, Cuba, French Polynesia, Gibraltar, Jamaica, Japan, Malaysia, New Caledonia, and Suriname. Here, note that Cuba developed its own vaccine.
- Similarly striking examples include (Rancourt et al., 2024, their Appendix B): Guadeloupe, Hong Kong, Maldives, Mauritius, Namibia, Philippines, Qatar, and Tunisia.

Therefore, there are many temporal associations between COVID-19 vaccine rollouts and increases in excess all-cause mortality, in every country with sufficient data.

It is highly unlikely that these many vaccine-mortality temporal associations — occurring at many different times for different vaccine rollouts during the two years of vaccinations and in every country having sufficient mortality and vaccination data in the Rancourt et al. (2024; and references therein) study — are coincidental.

These vaccine-mortality correlations in time occur even with the limitation that the contributions to all-ages excess all-cause mortality by population are exponential with age (Rancourt et al., 2024, their section 5.10) whereas the vaccine administration shown (their Appendix B) is for all ages, and there are many more injections to non-elderly than to elderly, administered in different rollouts. Observed vaccine-mortality correlations in time are generally most distinct with age stratified data and can be masked in all-ages data (Rancourt et al., 2023a, 2023b).

Possible mechanisms whereby COVID-19 vaccination could be a primary (Section 0) cause of death include:

a. The injection causes death by direct toxicity. Cationic lipids are candidates for toxic components.
b. The injection causes death by inducing an immune overreaction. The resulting immune assault, analogous to an allergic reaction, is enough to accelerate and cause the death.
c. The injection and repeated injections (2nd dose and boosters) cause immunosuppression making the patient generally more susceptible to infections and less able to defend against existing infections, including respiratory infections, in turn causing death. (See Section 0 regarding Amer et al., 2024, their Table 6.)
d. A frail patient is infected or additionally infected by a person who was made more infectious by injection-induced immunosuppression — either another patient or a caregiver, for example. Their death is accelerated where it otherwise would not have been.
e. As an accident of the physical injection itself, the vaccine product can be introduced directly into a large blood vessel, rather than into the muscle tissue as intended. The resulting bolus then potentially leads to fatal consequences by high-concentration rapid mass bulk delivery to sensitive organs, systems or tissues (Girardot, 2024).

Regarding point-c, for example, following Selye (1956), if subjected to widespread (even booster-repeated) presence of antigens in the blood, the body could respond by suppressing its non-specific inflammation response, including in the respiratory tract, thus making the individual more susceptible to varied and deeper respiratory infections (and skin infections: Martora et al., 2023). Also, theoretical mechanisms specifically for COVID-19 vaccine-induced immunosuppression have been suggested (Palmer et al., 2023, their sections 3.3 to 3.5; Seneff et al., 2022).

These possible mechanisms (point-a through point-e) are consistent with the observations based on age-stratified data that the effective "vaccine dose fatality rate (vDFR)" (per injection basis) increases exponentially with age and increases with the dose number (Rancourt et al., 2023a, 2023b).

The COVID-19 vaccine injection primary (Section 0) cause of death hypothesis is not invalidated by the background of biological stress that would have characterized the Covid period, or by the assaults from other medical interventions, since the injections are taken to have accelerated the deaths that would not have otherwise occurred until later.

In conclusion of this sub-section (0), the many directly observed strong temporal associations between vaccine rollouts and sudden increases in excess all-cause mortality, for all ages combined or by age group, in virtually every country with sufficient data (Rancourt et al., 2024; and references therein), would compel one to include the hypothesis that COVID-19 vaccination was a primary (Section 0) cause of death significantly contributing to the measured excess all-cause mortality. In

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addition, there is ample and conclusive evidence, from the clinical and adverse-effects perspectives, that the COVID-19 vaccine injections can cause death in individuals (e.g., as reviewed by Rancourt et al., 2023a, in their section 6.1).

Nonetheless, it is possible, despite the above-described remarkable and pervasive correlations in time between vaccine rollouts and increases in excess all-cause mortality, that the COVID-19 vaccine injections themselves are not a primary (Section 0) cause of death sufficient in strength to be the actual primary cause of the associated increases in excess all-cause mortality. This possibility is discussed in the next Section 0.

3.3.6. Hypothesis: Assaults from campaigns and measures associated in time and place with COVID-19 vaccine rollouts

As mentioned above in Section 0, it is possible that the expected deaths caused by the COVID-19 vaccine do not produce large excess all-cause mortality, despite the vaccine rollouts being known to be strongly temporally associated with observed surges in excess all-cause mortality.

Here, I discuss possible significant primary causes of death other than COVID-19 vaccines, which are associated in time and place with COVID-19 vaccine rollouts.

This possibility was noted by Rancourt et al. (2024, their section 5.1), as:

> "…it is possible that the observed strong correlations occur due to one or several hidden factors, rather than from a direct causal relationship due to challenge via toxicity of the injected substance.
>
> For example, we might postulate that the teams of attendants who walk into the various institutions housing frail people to administer the latest booster during the period of a rapid rollout, are accompanied by or serve the dual function of a team of attendants who test for positive cases of presumed COVID-19. Each positive test or diagnostic determination, in turn, whether real or false, could have significant negative health consequences for the individual, such as isolation, removal to a different location, confinement, and aggressive chemical and mechanical medical treatment."

In other words, the impugned COVID-19 vaccine rollouts may be synchronously accompanied by concomitant aggressive medical and/or health interventions, and the latter interventions would be the relevant primary cause(s) of death.

Examples of such accompanying interventions might include:

- the use of incorrectly stored or handled COVID-19 vaccination products
- incorrect combinations of COVID-19 vaccination products from different manufacturers
- incorrect physical administrations of the COVID-19 vaccine, using rushed or ill-trained staff
- testing for COVID-19, and the associated consequences of positive test results
- more aggressive or extreme immobilisation and isolation enforcement during the vaccine rollout
- the psychological stress of being coerced into re-vaccination, in the institutional environment
- administration of influenza or other vaccinations
- administration of medications intended to facilitate acceptance or to alleviate side effects of the injections
- disrupted patient care schedule, including regular medication, meals and hydration
- transmitted stress of the attendants, or infections from the attendants
- and so on

The thus associated or accompanying assaults can be different in their array and different in magnitude from one country to another, from one institution to another, and from one COVID-19 vaccine rollout to another (with multiple doses, such as boosters). For example, Rancourt (2022) discusses the case of India, compared to the consequences of so-called vaccine-equity campaigns in the USA.

For these rollout-associated concomitant aggressive medical and/or health interventions (the above bulleted list), each can be a relevant primary cause of death if it would have caused the deaths which otherwise would not have occurred (for example, in the relevant short time interval of the sharp mortality peak that follows the vaccine rollout).

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3.3.7. Discerning the COVID-19 vaccination and rollout-associated assault primary causes

In this sub-section, I examine the problem of discerning the COVID-19 vaccination (Section 0) and rollout-associated assaults (Section 0) as primary causes.

Again, as pointed out by Rancourt et al. (2024, their section 5.1):

> "… To constrain whether or not vaccine toxicity directly causes measurable mortality, versus (for example) the fatal impact of other and concomitant large-scale public health interventions, … the researcher should have access to vaccine-status-discriminated all-cause mortality data. Such data will constrain more definitively whether the COVID-19 vaccination rollouts have life-saving benefits, or cause additional mortality, and the degree of these relations. This data is needed for the same countries in which strong temporal associations are present between rapid vaccine rollouts and sharp peaks in excess all-cause mortality."

This is true. Tracy Beth Hoeg vehemently made this and other criticisms of Rancourt et al. (2023a). See Hoeg's criticisms and my response to them: Rancourt (2024, and references therein).

Recently, Rancourt et al. (in preparation) analysed national mortality data in one country in which COVID-19 vaccination status was known at death, including the history of COVID-19 vaccinations, in a case in which the country exhibited rapid vaccine rollouts strongly temporally associated with observed surges in excess all-cause mortality. They found that relevant peaks in excess all-cause mortality associated with booster rollouts could not preferentially be assigned to booster-vaccinated individuals (and also that the vaccine had no detectable survival benefit).

This means that the COVID-19 vaccination primary cause described in Section 0 did not produce a measurable increase in excess all-cause mortality in this country, which in turn means that sharp peaks in excess all-cause mortality which are temporally associated with rapid vaccine rollouts need not imply that COVID-19 vaccination is a primary cause of death. Rather, it seems that (as pervasive as they are) such temporal associations between mortality peaks and rapid vaccine rollouts are due to the primary cause described in Section 0 of campaigns and measures associated in time and place with COVID-19 vaccine rollouts. Although non-conclusive in general, this is consistent with the fact that the vaccine toxicity causing death inferred from population-wide adverse-effect monitoring is usually too small to be detected directly in population-wide (e.g., national) cause-independent all-cause mortality, as per analyses of USA VAERS (Vaccine Adverse Events Reporting System) data (Hickey and Rancourt, 2022).

This would mean that the lethality of medical measures imposed during the Covid period and during vaccine rollouts is much greater than generally acknowledged, and much greater than the known (VAERS, autopsies, etc.) vaccine toxicity itself.

3.3.8. Hypothesis: Increased stressors cause surges in spontaneous microbial respiratory self-infection (aspiration pneumonia)

In this sub-section, I propose a mechanism whereby sudden impositions of extraordinary biological stress (from Covid-period mandates and measures, as per Sections 0 and 0) and/or sudden impositions of extraordinary medical assaults during the Covid period (as per Sections 0, 0 and 0) quickly lead to rapid surges in respiratory deaths, detected as peaks and increases in excess all-cause mortality, without any person-to-person transmission of a respiratory pathogen being required.

The proposed said mechanism in part involves spontaneous development of aggressive bacterial pneumonia in fragile individuals, known as aspiration pneumonia.

"Aspiration is defined as the inhalation of oropharyngeal or gastric contents into the larynx and lower respiratory tract." (Marik, 2001). Aspiration pneumonia is bacterial pneumonia caused by aspiration, including both overt aspiration and silent micro-aspiration during sedation or sleep (Teramoto, 2022). Aspiration pneumonia has a higher mortality rate than non-aspiration pneumonia (Gupte et al., 2022). It is self-generated within the susceptible individual and does not depend on person-to-person transmission of a pathogen.

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Aspiration pneumonia itself is a known and increasingly studied phenomenon (Asai and Isono, 2014; Ebihara and Ebihara, 2011; Gupte et al., 2022; Koivula et al., 1994; Mandell and Niederman, 2019; Marik, 2001; Marik and Kaplan, 2003; Prass et al., 2006; Teramoto, 2022; Zuercher et al., 2019).

The study of aspiration pneumonia has provided an entirely new view of pneumonia, as an infection that can be self-generated rather than necessarily transmitted from another person. It has also provided a new view of the lungs themselves. In the words of Mandell and Niederman (2019):

> "Studies of the lung microbiome have challenged our assumptions of lung sterility and of bacterial access to the lungs through aspiration (microaspiration or macroaspiration) and inhalation. Specifically, genomic methods have defined a complex taxonomic landscape of bacteria in the lung and revealed the presence of diverse communities of microbiota.
>
> … The complex adaptive system model suggests that acute bacterial pneumonia results from enhancement of a growth-promoting signal by a positive feedback loop. This may result in a rapid shift from a diverse microbial mixture to dominance by a single species (e.g., *Streptococcus pneumoniae* or *Pseudomonas aeruginosa*).12
>
> One hypothesis linking the airway microbiome with aspiration pneumonia is that illness may result in a change in the lung microbiota (dysbiosis), which may, in turn, interfere with or impair pulmonary defenses. A macroaspiration event, particularly in a patient with risk factors for impaired bacterial elimination, such as reduced consciousness or an impaired cough reflex, could then overwhelm the elimination side of the immigration–elimination balance, further disrupting bacterial homeostasis and triggering an increase in a positive feedback loop leading to acute infection."

This means, among other things, that pneumonia does not require person-to-person transmission. Aspiration pneumonia, in particular, is a phenomenon that does not require or involve person-to-person transmission, as it is presently described in the medical literature.

Aspiration pneumonia is a dominant cause of death in old people in ordinary (non-pandemic-response) circumstances, especially in care facilities and hospitals. It is the leading cause of death among residents of nursing homes (Gupte et al., 2022; Marik and Kaplan, 2003; Teramoto, 2022). The recognized treatment protocol involves correct diagnosis, preventative and treatment measures against dysphagia (difficulty swallowing, see Zuercher et al., 2019), and the administration of antibiotics (Mandell and Niederman, 2019).

My hypothesis is that the pandemic-response circumstances of the Covid period (the mandates, measures and medical assaults, Sections 0, 0, 0, 0 and 0) induced a significantly amplified occurrence and virulence of aspiration pneumonia in elderly and frail populations. This is supported by several known characteristics of aspiration pneumonia, as follows.

i. A dominant risk factor for aspiration pneumonia is immunosuppression, whereas:
   a. biological and psychological stress suppress immune response (Section 0)
   b. including immobilization and isolation (Section 0)

ii. Dominant known risk factors for aspiration pneumonia include several iatrogenic causes, which increased significantly with pandemic so-called response, including:
   a. diagnostic bias and general denial of antibiotic treatments (Mandell and Niederman, 2019; Rancourt et al., 2021a, their section 5)
   b. mechanical ventilation (Pneumatikos et al., 2009)
   c. sedatives, hypnotics, muscle relaxants, sleeping pills, psycho psychotics (Gupte et al., 2022; Teramoto, 2022; their Table 1)
   d. drugs that cause dry mouth (e.g., anticholinergic drugs, tricyclic antidepressants) (Teramoto, 2022; their Table 1)
   e. tube feeding (placing of the nasogastric tube itself disturbs swallowing function) (Teramoto, 2022; their Table 1)
   f. polypharmacy (unexpected side effects of agents) (Teramoto, 2022; their Table 1)
   g. endotracheal intubation tube, tracheotomy tubing (Teramoto, 2022; their Table 1)
   h. depressed consciousness, use of opioids, anesthesia (Asai and Isono, 2014)
   i. depressive disorder (Gupte et al., 2022)
   j. suicide attempt (Teramoto, 2022; their Table 1)

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k. medications for the treatment of gastroesophageal reflux (Gupte et al., 2022)

iii. A dominant risk factor for aspiration pneumonia is suppression of the defenses against aspiration, whereas
   a. sedation significantly increases aspiration (Gupte et al., 2022)
   b. immobilization and being bedridden significantly increases aspiration (Prass et al., 2006)
   c. any obstruction to breathing (which presumably would include face mask wearing) significantly increases aspiration ("impair[ed] pulmonary clearance", Gupte et al., 2022)
   d. medical or other suppression of coughing significantly increases aspiration and aspiration pneumonia (Ebihara and Ebihara, 2011)

iv. Comorbid conditions associated with death from aspiration pneumonia include alcoholism and opioid related disorders (Gupte et al., 2022). The use of alcohol and opioids may have increased in the elderly during the Covid period.

I would add that one should expect pandemic-response-induced increased fear and psychological stress to increase aspiration disorders and the frequency of silent micro-aspirations during sleep, which are dominant causes of aspiration pneumonia in old people.

I would also add that (Covid period) biological and psychological stress and medical assaults can significantly perturb the gut or digestive-tract microbiome, and that this perturbed gastrointestinal microbiome may in turn increase the likelihood and severity of aspiration pneumonia.

The general and systemic denial of antibiotics would have been devastating (Rancourt et al., 2021a, their section 5), given the above-described importance of aspiration pneumonia (Gupte et al., 2022; Mandell and Miederman, 2019; Teramoto, 2022) and its necessary treatment (Mandell and Niederman, 2019), irrespective of any considerations of "spread" and "contagion".

Likewise, the novel and widespread hospital use of mechanical ventilation during the Covid period is difficult to understand given the knowledge that existed in 2020 about ventilator-associated pneumonia and ventilation-induced aspiration pneumonia. Ventilation was a sure way to induce aggressive pneumonias, irrespective of anything else.

3.3.9. Hypothesis: Spontaneous microbial respiratory self-infection (spontaneous pneumonia) without aspiration

Given the above discussion (0), it is immediate to advance the hypothesis that transmissionless spontaneous pneumonia induced by the mandates, measures, so-called responses and medical assaults (0, 0, 0, 0, and 0) need not be aspiration pneumonia (need not require aspiration), but rather can be self-infection pneumonia developed without aspiration, including TB (Section 0), that relies on respiratory tract microbiome diversity, instability and intrinsic susceptibility to runaway imbalance (see the above quoted paragraphs from Mandell and Niederman, 2019).

3.4. Is a pandemic of transmissionless bacterial pneumonia possible?

In view of the above discussion (Section 0), it would seem that the answer to the question in this section's title is "yes", absolutely.

One need only satisfy at least one of the following conditions:

i. A sudden assault on many countries using measures that increase the likelihood and/or lethality of aspiration pneumonia (0) or spontaneous pneumonia (0) among elderly and frail groups, and/or

ii. A sudden socio-economic collapse affecting many countries, of a type that increases the likelihood and/or lethality of aspiration pneumonia (0) or spontaneous pneumonia (0) among elderly and frail groups, and/or

iii. A sudden man-made or natural environmental change affecting many countries, of a type that increases the likelihood and/or lethality of aspiration pneumonia (0) or spontaneous pneumonia (0) among elderly and frail groups

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In hindsight, this appears to already occur every winter, giving rise to the seasonal cycles of respiratory deaths in the Northern and Southern Hemispheres. The winter peaks in all-cause mortality are synchronous across all mid-latitude regions for a given hemisphere and show no detectable shifts that would imply contagious spread. In this case, the yearly environmental change is winter atmospheric conditions (Kuzmenko, 2019):

a. lower temperatures, known to cause significant biological stress (Section 0), with cold homes known to be associated with respiratory emergencies in winter (Rudge and Gilchrist, 2005)
b. lower atmospheric humidity, known to cause dry mouth, in turn known to increase aspiration, also expected to affect respiratory tract tissues and the associated microbiome, known to increase aerosol load and residency time
c. larger atmospheric pressure, and larger atmospheric pressure variability, expected to affect circulation, known to affect severity of pneumonia (during airplane flights)
d. larger partial pressure of oxygen, unknown effect on pneumonia incidence, large potential to affect respiratory tract microbiome and immune response (Park et al., 1992)
e. decreased daylight hours, unknown effect on pneumonia incidence, presumed connection to vitamin D (Chatfield et al., 2007)
f. decreased geomagnetic activity, unknown effect on pneumonia incidence

See the comments about seasonal mortality by Rancourt (2023b), and see published examples of seasonal patterns in many countries (Rancourt, 2020, 2022; Rancourt et al., 2020, 2021a, 2021b, 2022a, 2022b, 2022c, 2023a, 2023b, 2024; and references therein).

Historic pandemics may also be explained by large sudden natural environmental changes, such as large explosive volcanic eruptions. Stothers (1999) found that climatic and epidemiological (pandemic) consequences of the seven largest dry-fog-producing volcanic explosions of the past 21 centuries were closely associated in time, to a degree that was "very unlikely coincidence". Stothers emphasized volcanic cooling and cold proximal winters, but it is also possible that dispersed toxic volcanic emissions played a role.

On the other hand, a competing or complimentary thesis is that the great plagues of the Middle Ages (including the Black Death) were induced by socio-economic collapse, in great waves that repeat the sequence:

growing class exploitation →

money shortage →

rising prices of living commodities →

widespread extreme poverty →

deleterious developmental and health consequences →

plague (concomitant revolution and/or monetary reset)

This thesis is developed in the landmark work of Fischer (1996) entitled "The Great Wave: Price Revolutions and the Rhythm of History". Fischer's observations are consistent with the work of DeWitte and Wood (2008) who showed by forensic analysis of skeletons that those who died in the Black Death were developmentally malnourished and in poor prior health.

Pneumonia epidemics in nursing or care homes may also be transmissionless events. Residents of nursing homes already systemically suffer from malnutrition and dehydration (Kayser-Jones et al., 1999; Volkert et al., 2019). Many care-home residents already suffer from dysphagia, the dominant risk factor for aspiration pneumonia. Therefore any sufficiently impactful sudden change of the conditions in a care home can induce a spontaneous epidemic of aspiration pneumonia (0) or spontaneous pneumonia (0) in the said care home, irrespective of any person-to-person (resident-resident or resident-staff/visitor) transmission of a respiratory pathogen. Examples of sufficiently impactful sudden changes might be as follows, which may occur in combination:

- loss or change of care staff (including increased negligence)

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- change in staff management (including loss of oversight)
- change in diet or its administration (including fluids)
- change in public-health safety protocols and conditions (including isolation)
- loss of access to facilities or services (including washrooms or attendants)
- change in administered medications
- increased immobilization (in-bed or in-room) for any reason
- change in environmental conditions (e.g., temporary loss of indoor heat)
- transferred or transmitted fear or stress from staff
- increased frequent physical displacements (e.g., for treatment or testing)

Similarly, I would expect that gastrointestinal epidemics of diarrhea (for example) could be spontaneously induced by such changes, without necessarily involving person-to-person transmitted pathogens.

I end this section by suggesting that transmissionless mechanisms for pandemics and epidemics have incorrectly been overlooked and underappreciated. Basically, a person's own microbiome is enough to cause virtually anything, under changing conditions. The bias towards pathogen-transmission mechanisms exhibited by clinicians, epidemiologists and public-health managers, as the dominant or driving mechanism of pandemics and epidemics, has evolved historically (Martin and Martin-Granel, 2006), and in my opinion is not justified. In fact, it is disproved by epidemiological all-cause mortality studies (Section 0). The said bias, including the associated and widespread diagnostic bias, relies disproportionately on molecular tests of bodily fluids and blood, which are not as specific as claimed, and are not sufficiently and rigorously validated, despite their comprehensive promotion by the industry.

3.5. Has a pandemic-causing viral respiratory pathogen ever existed?

At this point, it is not unreasonable to ask whether the viral spread theory of respiratory epidemics and pandemics has any relevance whatsoever to the observed phenomena.

The said viral spread theory as a cause of the declared Covid pandemic is disproved by a large amount of empirical mortality data (Section 0), and its supporting evidence is tenuous, appearing largely to rely on a persistent narrative and technological optimism, while being motivated by large industrial and institutional interests.

Is humanity really at risk of catastrophic near-extinction events from mutations (accidental or designed molecular sequences) in relatively small genetic macromolecules that spontaneously enter and mega-replicate in living cells in our healthy bodies? I cannot find the receipts.

What is the relevance of a spreading specific respiratory pathogen if every epidemic and pandemic requires:

- virtually synchronous deaths over large areas, from institutional to global
- large pools of malnourished and unhealthy individuals
- spectra of comorbidities
- immunity from death of the wealthy or well-resourced
- large episodes of socio-economic upheaval
- contexts of aggression and societal conflict (war, class predation, international impositions…)
- large environmental events (toxic substances, volcanos, floods, lengthy droughts…)
- co-infection by more virulent (bacterial) pathogens
- propagandistic feedback and social transmission of fear?

Regarding this question, I have difficulty understanding those scientists who admit the fact of no detectable excess all-cause mortality attributable to a spreading virus during the Covid period yet insist that there are "novel viruses and variants" out there and among us, as though their proposition, true or false, was relevant to public health. They often fall back on "novel symptoms" such as loss of smell. Long-term and temporary loss of smell and taste are commonly associated with seasonal colds and other ailments (de Haro-Licer et al., 2013; Pellegrino et al., 2016). I would additionally make the point that any condition or disease brought on or exacerbated by exceptional biological stress or exceptional medical assaults will have "novel symptoms". In addition, symptoms are always a

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function of the individual's environmental and societal field, convoluted with the individual's particular health status and biology. Symptoms are rarely proof of a specific cause, and all the symptoms in the present context are non-specific.

In contrast, the critics of pandemic-causing viral spread theory bring many significant points that the theory is not actually supported (Bailey, 2022). Their points should be rigorously addressed rather than summarily discounted.

Many well-researched books delve into this matter and cannot easily be disregarded, such as:

- *"The Final Pandemic: An Antidote To Medical Tyranny"* ---- by Mark Bailey and Samantha Bailey (2024)
- *"Can you catch a COLD?"* ---- by Daniel Roytas (2024)
- *"Turtles All The Way Down: Vaccine Science and Myth"* ---- by Anonymous (2022) ---- Mary Holland J.D. (Foreword, Editor), Zoey O'Toole (Editor)
- *"Virus Mania: Corona/COVID-19, Measles, Swine Flu, Cervical Cancer, Avian Flu, SARS, BSE, Hepatitis C, AIDS, Polio, Spanish Flu. How the Medical Industry Continually Invents Epidemics, Making Billion-Dollar Profits At Our Expense"* ---- by Torsten Engelbrecht and Claus Köhnlein and Samantha Bailey (2020) (2021)
- *"The Moth in the Iron Lung: A Biography of Polio"* ---- by Forrest Maready (2018)
- *"Dissolving Illusions: Disease, Vaccines, and the Forgotten History"* ---- by Suzanne Humphries and Roman Bystrianyk (2013) (2015)
- *"Anatomy of an Epidemic: Magic Bullets, Psychiatric Drugs, and the Astonishing Rise of Mental Illness in America"* ---- by Robert Whitaker (2010) (2015)
- *"Fear of the Invisible: How scared should we be of Viruses and vaccines, HIV and AIDS?"* ---- by Janine Roberts (2008)
- *"AIDS Inc.: Scandal of the Century"* ---- by Jon Rappopport (2004)
- *"Inventing the AIDS Virus"* ---- by Peter Duesberg (1996)
- *"The Myth of Heterosexual AIDS"* ---- by Michael Fumento (1993)

In view of all of the above (all sections), it is possible that all recorded pandemics in history have occurred from biological-stress-induced (including environmental exposure and extreme malnutrition) transmissionless spontaneous self-infections. The said self-infections could arise from within the respiratory tract and digestive tract microbiomes (and parasites) of individuals, in the living conditions at the time and place. The said biological-stress-induced transmissionless spontaneous self-infections could also be spontaneous infections of the skin and its injuries, from the skin microbiome (Byrd et al., 2018), skin parasites (Norgan and Pritt, 2018), and environmental toxins.

The same would be true of the synchronous seasonal patterns of large winter mortality, quantitatively and universally observed (except at equatorial latitudes) for more than a century.

4. Conclusion

Within the present state of knowledge, it is possible that the declared Covid pandemic (2020-2023) was entirely caused by the coordinated and largescale mandates, measures, so-called responses, and medical assaults including testing, diagnostic bias, isolation, denial of treatment (especially antibiotics for pneumonia), mechanical ventilation, sedation, experimental and improper treatments, and vaccination.

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In other words, it is possible that the declared Covid pandemic was a pandemic of mistreatment- and biological-stress-induced transmissionless spontaneous bacterial pneumonia (such as aspiration pneumonia).

It appears the impact of biological stress (which includes psychological stress) on individual health vulnerability and mortality is vastly underestimated and largely disregarded by the medical establishment (0).

The viral spread theory of respiratory pandemics as a cause of the declared Covid pandemic is disproved by a large amount of spatiotemporal excess all-cause mortality data worldwide (0), irrespective of whether the presumed virus is postulated to be natural or engineered. In contrast, all the epidemiological data is consistent with my hypothesis.

As a corollary, this would mean that the testing that was implemented is essentially meaningless (e.g., Rancourt, 2021, 2022; Rancourt et al., 2023a, their section 6.6), and that if a pandemic had not been declared and acted upon, then nothing unusual would have occurred in population health.

Furthermore, it is possible that all recorded pandemics in history have occurred in this way, from biological-stress-induced (including environmental exposure and extreme malnutrition) transmissionless spontaneous self-infections. The said self-infections could arise from within the respiratory tract and digestive tract microbiomes (and parasites) of individuals, in the living conditions at the time and place. The said biological-stress-induced transmissionless spontaneous self-infections could also be spontaneous infections of the skin and its injuries, from the skin microbiome and from skin parasites.

Additionally, the synchronous seasonal patterns of large winter mortality, quantitatively observed for more than a century, are likewise explained.

This would mean that the spreading virus hypothesis is unnecessary, and that we always carry on and within our own bodies everything needed to get sick and die when our physical and human environments violently assault us beyond our ability to recover.

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