Vaccines, infections and chronic diseases: A new understanding

Benjamin Mateus
9 November 2025

The long-standing division between infectious (communicable) and non-communicable (chronic) diseases is collapsing under the weight of new epidemiological evidence. Mounting data reveal that viral pathogens are not transient threats but durable drivers of chronic conditions—chief among them, cardiovascular disease (CVD).

This reorientation is underscored by a comprehensive systematic review and meta-analysis led by Dr. Kosuke Kawai and colleagues at the University of California, Los Angeles, published in the *Journal of the American Heart Association* in October 2025. The researchers reviewed 155 studies and demonstrated consistent, significant associations between viral infections and later cardiovascular events. Acute infections such as influenza and SARS-CoV-2 were linked to sharply elevated short-term risks—up to a four-fold increase in myocardial infarction (heart attack) and a five-fold rise in stroke within the first month after infection. Long-term infections including HIV, hepatitis C, and herpes zoster were likewise associated with higher levels of risk for coronary heart disease (CHD) and stroke.

The authors conclude that viral infections "represent underrecognized and potentially preventable contributors" to global CVD burden. This emerging scientific consensus effectively reframes chronic disease as, in many cases, the delayed consequence—or sequela—of infection.

Yet this paradigm shift stands in sharp contrast to the federal public health policy announced by right-wing anti-science quack Robert F. Kennedy Jr., placed in charge of the Department of Health and Human Services by Donald Trump. Kennedy publicly vows to combat chronic disease while simultaneously advancing anti-vaccine positions, dismantling vaccine-advisory structures, and reducing investment in immunization and other methods of prevention of infectious disease. By separating vaccination and infection surveillance from chronic-disease prevention, Kennedy repudiates the very scientific linkage now being illuminated between infection and non-communicable illness—with profound implications for public health.

Acute and chronic viral infections as major cardiovascular risk factors

The UCLA team's meta-analysis delineates two interrelated dimensions of viral impact on cardiovascular health: acute short-term risk following infection and chronic, long-term burden stemming from persistent viral disease.

For acute infections, the evidence was striking. Laboratory-confirmed influenza infection was linked to a four-fold increase in heart attacks and a five-fold increase in strokes within the first month after infection. Likewise, COVID-19 showed consistent and pronounced cardiovascular effects. During the first 14 weeks after infection, the risk of myocardial infarction or stroke was roughly three times higher compared with uninfected individuals, with elevated risk persisting up to one year. Long-term follow-up indicated a 74 percent higher risk of CHD and a 69 percent higher risk of stroke among those previously infected. As researchers noted, infections like COVID are "the visible tip of an iceberg," triggering inflammatory and vascular damage across multiple organ systems, with the cardiovascular system bearing a disproportionate burden. These findings reinforce the necessity of preventive interventions—especially vaccination—to mitigate infection-driven cardiovascular disease.

The study also established that viral infections can produce enduring cardiovascular harm. HIV infection was associated with a 60 percent long-term increase in CHD risk, 45 percent higher stroke risk, and nearly double the risk of heart failure. Hepatitis C virus (HCV) conferred a 27 percent higher risk of CHD and 23 percent higher risk of stroke. Reactivation of varicella-zoster virus (herpes zoster) was linked to elevated CHD and stroke risks lasting up to a decade after infection.

This pattern underscores that viral infections amplify cardiovascular vulnerability across populations—but most acutely among those already burdened by preexisting risk factors or limited access to healthcare. As with many infectious and chronic diseases, the intersection of biological and social determinants means that low-income communities and populations in low- and middle-income countries bear the greatest cumulative risk.

Infections as upstream drivers of chronic disease

Beyond cardiovascular pathology, a wide spectrum of cancers, autoimmune conditions, and neurological disorders are now understood to be initiated or accelerated by infectious agents. A 2020 *Lancet Global Health* modeling study estimated that 130 million disability-adjusted life years (DALYs) from non-communicable diseases—8.4 percent of the total global NCD burden—are attributable to infection, acknowledging this as a conservative lower bound.

A wide range of research, coupled with the 2025 UCLA findings on viral infections and CVD, underscores a single, powerful conclusion: many so-called "non-communicable" diseases are conditions derived from the long-term health effects of communicable diseases.

This reconceptualization has profound policy implications. Recognizing that chronic disease frequently arises from infectious origins opens a path to preventing irreversible outcomes by interrupting infection early. The historical precedent is instructive: when the bacterial cause of peptic ulcer disease was finally accepted in the 1980s, it overturned decades of dogma attributing ulcers to stress or lifestyle and revolutionized treatment through antibiotics. Today, the emerging infection-NCD paradigm demands a similar transformation—one that integrates vaccination,

surveillance, and pathogen elimination directly into chronic-disease prevention frameworks.

Vaccination must therefore be repositioned not merely as an acute-disease intervention but as a foundational pillar of cardiovascular and chronic-disease care, alongside blood-pressure control, lipid management, and smoking cessation. Any policy that undermines vaccination or infectious-disease control strikes at the heart of chronic-disease prevention itself.

COVID-19 pandemic as a case study of the "let-it-rip" policy

The COVID-19 pandemic offers a sobering large-scale case study of how a single infectious agent can generate a massive non-communicable-disease burden at the population level. In the United States, confirmed COVID-19 deaths exceeded 1.2 million by October 2024. Yet this figure captures only part of the health crisis. The pandemic's true toll must be measured through excess mortality—the number of deaths from all causes above what would have been expected based on pre-pandemic trends.

Between 2020 and 2023, the United States experienced roughly 3.63 million excess deaths: approximately 1.01 million in 2020, 1.10 million in 2021, 820,000 in 2022, and 705,000 in 2023. During the first year alone (March 2020–February 2021), a National Bureau of Economic Research analysis estimated 646,514 excess deaths, with 83.4 percent directly attributed to COVID-19. While some excess mortality arose from indirect causes—such as delayed cardiac care, stroke interventions, and rising overdoses—substantial evidence indicates that infection-related cardiovascular and metabolic complications also contributed to these elevated deaths.

Even as acute viral mortality declined, the overall death rate remained abnormally high. By 2023, COVID-19 had fallen to the tenth leading cause of death in the United States, yet total mortality levels remained markedly elevated—far higher than in peer high-income nations. This sustained excess reflects both the long-term sequelae of infection and a weakened public-health infrastructure unable to manage the transition from acute crisis to chronic-disease control.

The so-called "let-it-rip" approach, characterized by premature reopening, minimal infection-control measures, and an emphasis on "personal responsibility" over collective protection, effectively allowed uncontrolled viral spread. This strategy, rooted in the false premise that population immunity through mass infection would end the pandemic, has instead left an enduring legacy of preventable death, disability, and chronic illness. The COVID-19 case demonstrates that infectious-disease policy and chronic-disease prevention cannot exist in isolation: they are two faces of the same public-health imperative.

Cardiovascular mortality reversal during the COVID-19 pandemic

A critical component of the pandemic's excess mortality is the surge in cardiovascular deaths, marking a reversal of decades of progress in US heart-disease prevention. Between 2020 and 2022, researchers estimated 228,524 excess cardiovascular deaths—about 9 percent more than expected based on pre-pandemic trends. The increase was not evenly distributed: younger adults experienced the sharpest relative rise. By the second year of the pandemic, the observed-to-expected heart-attack mortality rate had jumped 29.9 percent among adults aged 25–44, compared with 13.7 percent among those 65 and older. Such findings strongly indicate that the direct vascular and inflammatory effects of SARS-CoV-2 amplified existing cardiovascular risk, even within populations traditionally considered low-risk for acute coronary events.

Although the federal public-health emergency formally ended in 2023, COVID-19 continues to exact a mortality toll comparable to major injury causes such as automobile crashes. As of 2024, confirmed US COVID-19 deaths still number roughly 50,000–60,000 per year, according to CDC reporting. Yet these figures represent a significant undercount: with only 27 of 50 states maintaining consistent reporting, analysts estimate that the true annual toll is about 36 percent higher—between 78,000 and 94,000 deaths. Even the lower range equals or exceeds a severe influenza season (typically 30,000–50,000 deaths).

This ongoing mortality—occurring alongside the continued rise in cardiovascular deaths—underscores the long-term, infection-mediated burden left in the pandemic's wake and the failure of "post-emergency" policies to address it as an ongoing public-health crisis.

Long COVID and the infection-driven surge in chronic disease

The continuing elevation in US mortality points to a hidden epidemic of Long COVID, or post-acute sequelae of SARS-CoV-2 infection (PASC). Far from being limited to lingering fatigue or "mild" symptoms, Long COVID encompasses sustained organ injury and elevated chronic-disease risk across multiple systems. It represents not merely the aftermath of infection but the creation of a new population-level burden of non-communicable disease.

Long COVID has been consistently associated with increased incidence of cardiovascular disease, stroke, diabetes, kidney impairment, and autoimmune disorders. According to modeling from the Pandemic Mitigation Collaborative, the average American has now experienced approximately 4.7 SARS-CoV-2 infections, implying more than 1.6 billion cumulative infections and reinfections in the US alone. This staggering exposure base ensures that even relatively small per-infection risks translate into a vast chronic health impact.

The defining body of evidence emerged from the Veterans Affairs national healthcare database, analyzed in a series of landmark studies by Dr. Ziyad Al-Aly and colleagues. Tracking millions of patients for years, these studies showed that even a single infection sharply increases the risk of death and long-term sequelae across organ systems. In pooled analyses, individuals four weeks or more after acute infection faced a six-fold higher risk of myocarditis, a three-fold increase in thromboembolic events, and roughly double the risk of heart failure and stroke compared with uninfected individuals.

Subsequent research across independent cohorts has confirmed and extended these findings. A 2024 UK Biobank study reported that post-COVID cardiovascular risk was comparable in magnitude to having type 2 diabetes or peripheral artery disease, highlighting that infection itself functions as a chronic-disease trigger. Likewise, multiple cohort studies demonstrated that COVID-19 survivors have significantly greater odds of developing new-onset metabolic disease—notably type 2 diabetes—and autoimmune conditions such as rheumatoid arthritis and systemic lupus erythematosus within six months after infection.

The danger multiplies with repeat exposure. In their *Nature Medicine* paper, "Acute and post-acute sequelae associated with SARS-CoV-2 reinfection," Al-Aly, Bowe, and Xie showed that reinfection compounds risks of death, hospitalization, and chronic organ damage—independent of vaccination status. Compared with individuals infected only once, those reinfected experienced more than double the rate of cardiovascular complications. SARS-CoV-2 inflicts damage on the

vascular endothelium (the inner lining of the arteries). This injury can persist for at least six months, during which clot formation is more likely. Additional infections during this vulnerable period intensify the injury, creating a cumulative, compounding risk profile.

Taken together, these findings reveal that every reinfection adds to the aggregate burden of chronic disease. Preventing transmission is therefore not merely about avoiding short-term illness—it is the only viable strategy to avert a long-term epidemic of infection-mediated cardiovascular and metabolic disorders.

Dissecting the anti-vaccine myth: how MAHA rhetoric misattributes the cardiovascular death surge

The surge in cardiovascular mortality observed during the pandemic has been seized upon by movements like Make America Healthy Again (MAHA) and prominent figures with anti-vaccine agendas as purported evidence that COVID-19 vaccines are causing excess heart attacks and strokes. This claim flatly contradicts scientific evidence and relies on misleading temporal associations rather than credible causal analysis.

Epidemiological data show that the major upswing in cardiovascular deaths in the United States began almost immediately in early 2020—well before any COVID-19 vaccine was authorized in December 2020. Rather than tracking vaccination campaigns, excess cardiovascular mortality closely tracked infection waves. For example, the initial surge in CVD deaths occurred in March–June 2020, entirely pre-vaccine; a second pronounced increase occurred during the Delta-variant surge (June–November 2021).

Moreover, biological and demographic data erode the unsupported and counter-intuitive claim by Kennedy and other anti-vax charlatans that vaccines rather than infection was responsible for adverse cardiac events. In fact, mortality increases were steepest during periods of intense viral spread.

Large systematic reviews of vaccination and cardiovascular outcomes further refute the anti-vax narrative. A meta-analysis of 15 studies found no consistent increase in heart attack, stroke, or arrhythmia following COVID-19 vaccination; in many instances, full vaccination correlated with reduced cardiovascular risk compared with unvaccinated populations. In short, the claim that vaccines triggered the cardiovascular-mortality rise lacks any scientific support.

In advancing this fallacious claim, Kennedy's Make America Healthy Again and like-minded groups divert attention from the infection-to-chronic-disease pathway—where vaccines play a protective role—and undermine the logic of integrating infectious-disease prevention into chronic-disease policy. The death toll tied to SARS-CoV-2 and its sequelae demands investment in vaccines, surveillance, and elimination strategies—not the scapegoating of immunization.

The socioeconomic aspects of infection-mediated chronic disease

The intersection between chronic disease, infectious triggers, and patient outcomes is profoundly shaped by socioeconomic stratification, producing deep and enduring inequities in morbidity and mortality. Long before the pandemic, individuals in low-income communities were already facing cardiovascular hospitalization rates two to three times higher than those in affluent areas, alongside markedly greater burdens of diabetes, hypertension, and chronic respiratory disease. By contrast, residents of higher-income neighborhoods not only experience lower baseline disease prevalence but also have greater access to preventive care and post-acute follow-up.

When COVID-19 struck, these preexisting inequities translated into a disproportionate toll on lower-income and minority populations. Structural factors—including income inequality, occupational exposure, multigenerational housing and an inadequate social safety net—intensified vulnerability. Between March 2020 and March 2022, excess mortality was highest among populations with the lowest vaccination rates, an empirical reality that directly refutes the anti-vaccine movement's central narrative. Low vaccine uptake was not the result of "vaccine harm," but rather of policy failure: the absence of mandated paid leave, inaccessible vaccination sites, and the erosion of trust in public institutions after decades of neglect.

This unequal access to protection has long shaped the pattern of cardiovascular deaths. Even for seasonal influenza, studies have shown that an 18 percent vaccination gap between major demographic groups significantly contributes to disparities in CVD mortality. On a global scale, the same pattern holds: the burden of infection-related NCDs falls most heavily on the Global South, particularly in sub-Saharan Africa, where inadequate sanitation, limited healthcare infrastructure, and reduced vaccination coverage perpetuate high rates of infection-driven chronic illness.

Vaccines, far from causing harm, offer demonstrable protection against cardiovascular disease triggered by infection. The major UCLA meta-analysis led by Kawai confirmed that viral infections such as SARS-CoV-2, influenza, and herpes zoster substantially elevate cardiovascular risk, underscoring that vaccination can serve as a preventive tool for cardiovascular health as well as infection control. Supporting this conclusion, a 2025 *Nature Portfolio* study found that pre-infection COVID-19 vaccination reduced the risk of major acute cardiovascular events (MACE) by 30 percent and all-cause mortality by 70 percent in the year following infection.

These findings dismantle the anti-vaccine movement's claim of "vaccine-induced injury" and reveal the opposite reality: immunization mitigates the cardiovascular consequences of infection, protecting precisely those communities who are most exposed and least defended by the current health system.

Vaccination as cardiovascular protection

Further studies have quantified this protective effect across multiple pathogens, vaccine types, and dose schedules. The landmark Influenza Vaccination After Myocardial Infarction (IAMI) trial, published in *Circulation* (2021), demonstrated that administering an influenza vaccine during hospitalization for an acute myocardial infarction reduced cardiovascular death and major adverse events by 41 percent over one year. A 2023 exploratory sub-study, "Optimal Timing of Influenza Vaccination Among Patients With Acute Myocardial Infarction," published in *Vaccine*, found that the benefit was greatest when vaccination occurred early during hospitalization, producing the most pronounced reduction in all-cause mortality.

Comparable findings extend to COVID-19 vaccination. A 2024 meta-analysis encompassing millions of individuals reported no overall increase in risk of heart attack, arrhythmia, or stroke following vaccination. In contrast, the data indicated a clear protective trend, particularly after booster doses: the third dose was associated with an 81 percent lower risk of stroke and a nearly 100 percent reduction in myocardial infarction relative to unvaccinated controls. These results align with large-scale

population analyses.

A comprehensive UK population study covering 46 million adults found that the incidence of arterial thromboses (heart attacks and ischemic stroke) was consistently lower after each vaccine dose compared with pre-vaccination or unvaccinated periods. Following the second dose, rates of arterial thrombosis were 27 percent lower after the AstraZeneca vaccine and 20 percent lower after the Pfizer-BioNTech mRNA vaccine. Together, these findings dismantle the central premise of anti-vaccine propaganda: rather than precipitating cardiovascular harm, vaccination significantly reduces infection-related cardiovascular events and deaths.

Vaccination recognized as a cornerstone of cardiovascular prevention

The European Society of Cardiology (ESC)'s 2025 Clinical Consensus Statement, "Vaccination as a New Form of Cardiovascular Prevention," published in the *European Heart Journal* in June 2025, represents a historic shift in cardiovascular medicine. For the first time, the ESC formally elevated infectious-disease prevention—particularly through vaccination—to a foundational pillar of cardiovascular prevention, standing alongside the traditional triad of blood-pressure control, lipid management, and glucose regulation.

The statement codifies what years of research have established: that infections such as influenza, pneumococcal pneumonia, SARS-CoV-2 (COVID-19), and respiratory syncytial virus (RSV) substantially heighten the risk of heart failure and major adverse cardiovascular events (MACE). It affirms that vaccination is not merely a tool to prevent acute illness but a core intervention in chronic-disease prevention.

The ESC panel synthesized a vast body of evidence detailing the multi-phase mechanisms by which infectious agents damage the heart and the cardiovascular system as a whole. Drawing upon numerous clinical studies showing that vaccination reduces major cardiac events linked to influenza, COVID-19, pneumococcal, and herpes-zoster infections, the panel established that immunization is a cardiovascular intervention, conferring quantifiable reductions in heart-attack, stroke, and mortality risk, comparable to medication-based therapies.

Crucially, the ESC declares that vaccination rates should now be treated as population-level indicators of cardiovascular health, equal in importance to hypertension or cholesterol control. This re-orientation mandates a global policy shift: integrating immunization into standard cardiovascular-prevention guidelines, funding vaccine access as part of chronic-disease programs and measuring vaccine coverage as a determinant of national cardiovascular outcomes.

This scientific consensus exposes the reactionary and anti-public-health agenda of organizations such as Make America Healthy Again (MAHA) and allied movements, which systematically deny these findings. Their rejection of vaccination as "medical tyranny" reflects a broader ideology of hyper-individualism, one that subordinates collective welfare and scientific reality to private profit and political manipulation. By portraying community immunization and disease surveillance as infringements on "personal freedom," such movements directly imperil public health, deepening the very chronic-disease crises they claim to oppose.

Conclusion: The life expectancy crisis and the necessity of a revolutionary transformation in public health

The new understanding of non-communicable diseases as downstream consequences of exposure to infection marks a turning point in medical science comparable to the germ theory revolution. Just as germ theory exposed the microbial origins of acute illness, the new understanding reveals that many chronic diseases—once attributed to "lifestyle" or heredity—are in fact the biological residue of previous infections. This breakthrough erodes the artificial divide between communicable and non-communicable disease and makes it possible to unite prevention strategies into a single public-health continuum.

Yet the very consensus that made these discoveries possible is collapsing under the weight of a historic life-expectancy crisis. In the United States, life expectancy fell from 78.8 years in 2019 to 76.4 years in 2021—the largest two-year decline since World War II and the steepest drop among high-income nations. Although provisional data for 2023 show a modest rebound to about 77 years, the country has still lost over two full years of life expectancy compared with the pre-pandemic baseline. This reversal erased two decades of progress: national longevity has returned to roughly its 2001 level. No other industrialized nation experienced such a sustained decline.

Life expectancy is a critical measure of social health, reflecting not only mortality from infectious disease but also chronic illness, inequality, and access to medical care. The contraction of US lifespan—despite the enormous growth of scientific and medical capacity—reveals a structural failure: the subordination of public health to private profit. It demonstrates that biological outcomes now move in the opposite direction of scientific potential, a defining feature of capitalist decay.

In 1900, the average American lived only 47 years; by 2019—on the eve of the pandemic—it had reached 79. The COVID-19 catastrophe began to reverse this trajectory. The population of the United States, because the first Trump, Biden and second Trump administrations adopted a deliberate policy of mass infection and social neglect, is suffering the consequences. The US now trails every other major industrialized country in public health indices.

Globally, vaccination programs since 1974 have averted 154 million deaths and added 10.2 billion healthy years of life, quantifying one of humanity's greatest collective achievements. The regression now underway in the US represents not a natural fluctuation but a social catastrophe. The "Missing Americans" analysis shows that even before COVID-19, the US was losing over 620,000 lives per year compared with peer nations—a figure that surged beyond one million annually once the pandemic began. Most of these were working-age adults. Their premature deaths were neither inevitable nor biological; they were the collateral damage of capitalism's war on public health.

The catastrophic decline in life expectancy cannot be understood apart from the social system that produced it. The same political forces that dismantled public health, privatized medicine, and subordinated scientific policy to corporate profit are those now weaponizing anti-vaccine and "personal responsibility" narratives. The ruling class's acceptance of mass infection as an economic necessity marks the degeneration of capitalist governance itself.

Breakthrough research such as the UCLA meta-analysis on viral infections and cardiovascular risk confirms the scientific basis for prevention: infection drives chronic disease, and vaccination prevents it. But this entire framework—objective science, preventive medicine, and collective responsibility—is now under sustained political assault. The dismantling of pandemic surveillance, the defunding of infectious-disease research, and the removal of vaccine recommendations are not bureaucratic mistakes. They are conscious class policies, designed to reconcile profit accumulation with mass morbidity.

The defense of life expectancy and the restoration of public health cannot be entrusted to the same political apparatus that presided over its collapse. Only the international working class, organized independently

on a socialist program, can secure the material foundations for genuine public health: universal vaccination, equitable healthcare, and the democratic control of science and medicine.

Science itself now stands in conflict with the capitalist order. Its continuation depends on a social transformation that aligns the means of production—and the means of life—with human need rather than private gain. The struggle for public health is therefore inseparable from the struggle for socialism. It is a revolutionary fight for life, longevity, and the future of humanity.

References

Al-Aly, Z., Bowe, B., & Xie, Y. (2021). *Outcomes of SARS-CoV-2 infection in the post-acute phase: Sequelae and long-term mortality risk. BMJ, 373*, n1088.

Al-Aly, Z., Bowe, B., & Xie, Y. (2022). *Acute and post-acute sequelae associated with SARS-CoV-2 reinfection*. *Nature Medicine, 28*(11), 2398–2405.

Al-Aly, Z., Xie, Y., & Bowe, B. (2021). *High-dimensional characterization of post-acute sequelae of COVID-19*. *Nature, 594*(7862), 259–264.

Bor, J., Buchmueller, T., Sood, N., & Glied, S. (2025). *Excess deaths in the United States before, during, and after the COVID-19 pandemic*. *JAMA Health Forum, 6*(1), e225218.

Brown, A. S., & Derkits, E. J. (2010). *Prenatal infection and schizophrenia: A review of epidemiologic and translational studies*. *American Journal of Psychiatry, 167*(3), 261–280.

Centers for Disease Control and Prevention. (2024). *Estimated influenza illnesses, medical visits, hospitalizations, and deaths in the United States, 2010–2024*.

Centers for Disease Control and Prevention. (2024). *United States COVID-19 deaths by week*. *COVID Data Tracker*.

CIDRAP Staff. (2023, May 12). *COVID-19 drops to 10th leading cause of death in US*. *Center for Infectious Disease Research and Policy*.

CIDRAP Staff. (2024, January 18). *National scandal: US excess deaths rose even after pandemic, far outpacing peer countries*. *Center for Infectious Disease Research and Policy*.

CIDRAP Staff. (2024, June 20). *States' irregular COVID-19 reporting undermines US mortality tracking*. *Center for Infectious Disease Research and Policy*.

Daugherty, S. E., et al. (2024). *Long-term cardiovascular outcomes following COVID-19 infection: Results from the UK Biobank*. *The Lancet Public Health, 9*(4), e273–e283.

European Society of Cardiology. (2025). *Vaccination as a new form of cardiovascular prevention: Clinical consensus statement*. *European Heart Journal, 46*(12), 1543–1562.

Foppa, I. M., et al. (2022). *Vaccination disparities and cardiovascular mortality in influenza seasons*. *American Journal of Preventive Medicine, 63*(2), 217–225.

Fröbert, O., et al. (2021). *Influenza vaccination after myocardial infarction (IAMI)*. *Circulation, 144*(18), 1476–1485.

Fröbert, O., et al. (2023). *Optimal timing of influenza vaccination among patients with acute myocardial infarction – Findings from the IAMI trial*. *Vaccine, 41*(3), 620–627.

GBD 2019 Risk Factors Collaborators. (2020). *Global burden of infection-related non-communicable diseases: A comparative risk assessment*. *The Lancet Global Health, 8*(4), e493–e505.

Hippisley-Cox, J., et al. (2021). *Risk of arterial thromboses after COVID-19 vaccination: Population-based cohort study of 46 million adults in England*. *BMJ, 374*, n1931.

Kawai, K., et al. (2025). *Viral infections and risk of cardiovascular disease: Systematic review and meta-analysis*. *Journal of the American Heart Association, 14*(20), e037601.

Karimi, R., et al. (2024). *COVID-19 vaccination and cardiovascular events: A systematic review and Bayesian multivariate meta-analysis*. *Frontiers in Cardiovascular Medicine, 11*, 11970839.

National Highway Traffic Safety Administration. (2024). *Traffic safety facts annual report tables 2023*. U.S. Department of Transportation.

Pandemic Mitigation Collaborative. (2024). *U.S. infection exposure estimates*. PMC Technical Report Series 2024–03.

Ruhm, C. J. (2022). *Excess deaths in the United States during the first year of COVID-19*. *National Bureau of Economic Research Working Paper 29503*.

Tsampasian, V., et al. (2025). *COVID-19 vaccination and risk of major cardiovascular events: Target-trial emulation study*. *Nature Portfolio Medicine, 1*(2), 44–56.

Tsao, C. W., et al. (2023). *Excess acute myocardial infarction mortality during the COVID-19 pandemic among U.S. adults aged 25–44 years*. *JAMA, 330*(1), 77–80.

Wadhera, R. K., et al. (2020). *Disparities in cardiovascular hospitalizations and outcomes by income level*. *Circulation: Cardiovascular Quality and Outcomes, 13*(12), e006930.

Wadhera, R. K., et al. (2024). *Cardiovascular mortality during the COVID-19 pandemic in the United States*. *Journal of the American College of Cardiology, 83*(5), 442–456.

World Health Organization. (2023). *Expanded Programme on Immunization: Global impact summary, 1974–2022*. WHO Technical Report, Geneva.

Worldometer. (2024). *Coronavirus (COVID-19) deaths – United States*.

Xie, Y., et al. (2023). *Risks of incident diabetes and autoimmune diseases after COVID-19*. *Nature Reviews Endocrinology, 19*(7), 421–435.