

# Environmental Synchronization Between Background Galactic Cosmic Radiation and the Non-Random Timing of Viral Outbreak Emergence

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## Research Article

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# Abstract

**Background:** The temporal emergence of major viral outbreaks has traditionally been regarded as largely stochastic, with limited capacity for long-range anticipation. Increasing evidence from heliophysics and atmospheric science suggests that Galactic Cosmic Ray (GCRs) flux—modulated by solar magnetic activity and Earth–atmosphere coupling—constitutes a persistent background source of ionizing radiation and may represent an overlooked environmental driver capable of organizing biological phenomena across multiple timescales. As a ubiquitous component of the planetary radiation environment, GCRs continuously interact with biological systems, providing a plausible environmental context for large-scale temporal modulation without implying direct deterministic causation.

**Methods:** A total of 514 viral outbreak events (EM-DAT, 1964 to 2025) and GCRs intensity data (Oulu Cosmic Ray Station) were analyzed using SARIMA forecasting, PELT change-point detection, Granger causality, cross-spectral and Wavelet Transform Coherence (WTC), and a Vector Error Correction Model with exogenous harmonic components (VECMX). These methods were applied within an environmental–ecological time-series framework to evaluate whether large-scale GCRS variability aligns with population-level viral outbreak dynamics. Harmonic structures corresponding to the Hale (~22-year), Schwabe (~11-year), annual/semi-annual, and Quasi-Biennial Oscillation (QBO) cycles were incorporated to assess phase synchronization, ecological timing cues, and long-range temporal alignment between cosmic radiation variability and viral emergence patterns.

**Results:** GCRs variability significantly Granger-caused viral outbreak occurrence across all tested lags up to 12 months ( $p < 0.05$ ), with maximal significance within the first four months ( $p < 0.0001$ ), while no reverse causality was detected. Spectral coherence revealed robust phase-locked coupling at the Hale and Schwabe solar cycles (coherence = 0.91 and 0.85, respectively), indicating long-term synchronization. Additional statistically significant coherence was identified at quasi-biennial, annual, and semi-annual timescales, consistent with GCRs secondary particles modulation by the Quasi-Biennial Oscillation and seasonal atmospheric shielding. WTC demonstrated sustained coherence at the ~11-year Schwabe periodicity. Burst detection analysis further showed clustering of viral outbreak onsets during periods of low solar activity, notably around the 2009 and 2019 solar minima. Conditional harmonic VECMX and SARIMA projections indicate a renewed increase in GCRs intensity toward ~2030, coinciding with the anticipated solar cycle A<0, 25/26 minimum and a corresponding phase-aligned rise in viral outbreak activity.

**Conclusions:** Global viral outbreak dynamics exhibit statistically robust, multi-scale synchronization with GCRs variability. While GCRs are unlikely to act as direct causal agents, they may function as environmental timing cues or permissive triggers that modulate viral emergence or ecological susceptibility windows. Incorporation of heliophysical indicators as contextual environmental risk modifiers may enhance early-warning systems and global outbreak preparedness when integrated with conventional epidemiological surveillance frameworks.

# Introduction

The ability to forecast the sudden emergence of viral pandemics remains one of the most challenging real-world problems. While traditional models focus mainly on the human–animal–environment interface, these frameworks often struggle to account for the abrupt, synchronized shifts in viral outbreak observed globally. The impact of COVID-19 alone caused trillions of dollars of economic loss with millions of lives lost [1–3]. The unpredictable magnitude of these viral pandemics indicate that our existing models may miss critical feedbacks that can aid in early warning [4]. Conventional terrestrial parameters such as population density, zoonotic spillover events, and global travel patterns may be insufficient to account for sudden, coordinated surges in viral activity or the accelerated genetic shifts observed in emerging viral pandemics (Fig. 1A) [5–7]. Also, as the world becomes increasingly complex and interconnected, the threat posed by such pandemics grows even more. The speed and scale of global interactions will amplify the impact of localized outbreaks to escalate into worldwide crises, placing humanity at greater risk in the future [8].

The inability of traditional factors alone to predict the abrupt changes in viral outbreak emergence suggests the influence of external factors [1, 5]. In this context, Galactic Cosmic Rays (GCRs) emerge as a possibly critical modulators, interacting with the biosphere. The solar cycle intensity and magnetic field, regulates the flux of GCRs reaching Earth, with periods of solar minimum allowing increased GCRs radiation to penetrate the atmosphere [9, 10]. This interaction may contribute to global-scale shifts in viral mutation rates and transmissibility, providing a potential explanation for the synchronized, abrupt changes observed during major pandemics. This study aims to study the longitudinal relationship between GCRs flux and viral outbreaks events through advanced time-series analysis. The primary objectives are to evaluate variable synchronicity through cross-spectral coherence, cross-wavelet coherence (WTC), and Granger causality, with a project long-term phase-aligned dynamics (2026–2046) via a conditional harmonic Vector Error Correction Model with exogenous components (VECMX).

## Galactic Cosmic Rays and Solar Weather

The solar heliosphere is a vast, complex tailed bubble-like region of space dominated by the Sun's magnetic field and the outward flow of the solar winds. It acts as the solar system's primary defense against the interstellar medium and GCRs. The GCRs are high-energy particles originating from outside the solar system, primarily accelerated by supernova shockwaves within the Milky Way [11]. The GCRs consist mostly of ionized positively charged protons which can drift across solar magnetic field [11]. Variations GCRs flux arise from complex interactions between solar activity through heliospheric magnetic field (HMF), solar wind intensity and Earth atmospheric processes that influence GCRs propagation and its secondary particle production (Fig. 1B). The 11-year solar cycle modulate the intensity of the solar winds, here during solar maximum, the Sun's stronger magnetic field and denser solar wind deflect GCRs, leading to lower intensities on Earth [12]. Conversely, solar minimum sees a

significant increase in GCRs exposure, these particles, along with their secondary particle cascades, represent a potential source of genotoxic stress capable of damaging DNA and RNA [13].

The solar dynamo is a highly complex, nonlinear process that generates multiple overlapping cycles of solar magnetic activities [14]. While the 11-year Schwabe cycle governs sunspot variability, it is superimposed by another complex 22-year Hale magnetic cycle, during which the Sun's global magnetic polarity reverses and returns to its original state. This polarity reversal, occurring near solar maximum, introduces differences between successive solar cycles and significantly influences heliospheric conditions and GCRs flux [15]. Solar cycle morphology frequently exhibits a double-peaked maximum, commonly referred to as the Gnevyshev Gap, which is primarily attributed to hemispheric asymmetry and the temporal separation of magnetic flux emergence [16]. In contrast, GCRs modulation displays a well-established dependence on solar cycle intensity and the Hale magnetic cycle which is more apparent in solar minimum, where drift effects dominate. During positive polarity epochs ( $A > 0$ ), GCRs enter primarily through the solar polar regions, producing a flat-topped maximum, whereas during negative polarity epochs ( $A < 0$ ), transport along the heliospheric current sheet (HCS) and leads to a sharper GCRs peak on Earth [15, 16].

## Biological impact of Galactic Cosmic Rays

The biological impact of GCRs is primarily governed by their high linear energy transfer (LET) and high ionizing potential, characterized by dense energy deposition along the tracks of the charged particles as they traverse biological matter [17, 18]. Unlike low-LET radiation such as X-rays or gamma rays, GCRs and particularly heavy ions with high charge and energy (HZE) like carbon atoms (C), oxygen (O), neon (Ne), silicon (Si), calcium (Ca), and iron (Fe) can produce dense ionization columns that induce complex, clustered genetic lesions that are difficult for cellular mechanisms to repair [18, 19]. When primary GCRs interact with the Earth's atmosphere, they undergo nuclear spallation. This process generates secondary particle cascades, including neutrons, protons, pions, and muons [19]. Neutrons, despite being uncharged, possess high Relative Biological Effectiveness (RBE) (Fig. 1B). Because they are highly penetrating, they frequently collide with hydrogen nuclei in biological tissue, producing recoil protons that can cause localized, high-density damage [19–21]. These secondary cascades can penetrate the troposphere, contributing to a continuous background of ionizing radiation that fluctuates with solar activities and Earth atmospheric conditions [22, 23].

The resulting genetic damage may occur via two primary pathways, in which high-energy particles directly strike DNA, causing double-strand breaks (DSBs) and locally multiply damaged sites (LMDS) [19, 24]. The radiation can induce the radiolysis of water molecules, generating reactive oxygen species (ROS) such as hydroxyl radicals ( $\cdot\text{OH}$ ). These radicals cause oxidative base modifications and systemic oxidative stress [19]. In the context of viral dynamics, GCR-induced mutagenic pressure may alter viral quasispecies structure. At moderate levels, such mutational stress could enhance viral diversification, potentially facilitating reactivation or amplification. However, if the mutation rate exceeds the error-threshold, viral populations may enter an error-catastrophe regime [24]. Furthermore, GCRs flux is

modulated by solar cycle and the Hale Cycle. During solar minima, the weakened HMF allows for increased GCRs penetration into the inner solar system [16]. These periods of elevated GCRs flux, particularly during specific solar magnetic polarities ( $A < 0$  or  $A > 0$ ), have been hypothesized to act as external mutagenic drivers that may promote abrupt viral genetic shifts, potentially enhancing viral virulence or facilitating zoonotic spillover events [25, 26]. In host populations, chronic exposure to GCR-induced secondary radiation can theoretically lead to altered immune responsiveness, potentially compromising the host's ability to clear emerging viral variants [19, 27–30].

## Materials and Methods

### Data

The cosmic ray data were obtained on 26 July 2025 from the Oulu Cosmic Ray Station, which provides long-term monitoring of GCRs intensity using ground-based neutron monitors (<https://cosmicrays.oulu.fi/>). These measurements represent the flux of high-energy charged particles originating outside the solar system and modulated by heliospheric and atmospheric processes [31]. Viral outbreak data were obtained on the same date from the Emergency Events Database (EM-DAT), a comprehensive global repository maintained by the Centre for Research on the Epidemiology of Disasters (CRED), which systematically records natural disasters, including viral epidemics (<https://www.emdat.be/>) [32, 33]. For this study, viral outbreaks were analyzed using the reported start date of each outbreak event as the temporal reference. The combined dataset spans the period from 1 October 1964 (1964-10-01T00:00:00Z) to 1 March 2025 (2025-03-01T00:00:00Z) and comprises 514 discrete viral outbreak events. Viral outbreaks were treated as a point process, allowing for multiple outbreak occurrences on the same calendar date. When repeated events shared the same start date, they were preserved and counted to quantify event multiplicity. Viral event intensity was quantified by aggregating the number of outbreak events occurring on the same date. These time series were visualized jointly to assess long-term temporal patterns in cosmic radiation intensity alongside viral outbreak frequency.

### Seasonal Autoregressive Integrated Moving Average (SARIMA)

A seasonal autoregressive integrated moving average model [34], SARIMA(1,0,1)×(1,0,1,11), was fitted to the yearly GCRs radiation time series using `statsmodels.tsa.statespace.SARIMAX`, with an 11-year seasonal period corresponding to the solar cycle. Annual GCRs measurements were obtained by resampling the data to yearly resolution, with linear interpolation applied to fill missing years prior to model fitting. Stationarity and invertibility constraints were relaxed to allow flexible parameter estimation. Stationarity of the series was assessed using standard unit-root diagnostics prior to model fitting. Model performance was evaluated using the Akaike Information Criterion (AIC) (AIC = 644.981). The fitted model was used to generate a 10-year GCRs radiation forecasts, with 95% confidence intervals derived from the model's predictive distribution. Local maxima and minima in the forecasted series were

identified using `scipy.signal.find_peaks`. Observed values, forecasts, confidence intervals, and identified peaks and troughs were visualized using `matplotlib`, with the x-axis scaled in 5-year intervals to emphasize long-term behavior.

## Cross-Spectral Coherence, Burst Detection, and Granger Causality

Monthly radiation and viral event counts were aggregated into a continuous monthly time series. Months without recorded events were zero-filled, representing absence of the documented viral outbreak events rather than confirmed zero incidence; however, this assumption may bias short-term variability. The abrupt changes in viral event frequency were identified using the Pruned Exact Linear Time (PELT) change-point detection algorithm implemented in the `ruptures` package [35]. Cross-spectral coherence [36] between GCRs radiation and viral outbreak events counts was computed using `scipy.signal.coherence`, with frequencies converted to periods expressed in months. Statistical significance was assessed using a permutation-based surrogate approach. Viral outbreak events counts were randomly shuffled 1,000 times to generate a null distribution of coherence values, and empirical p-values were calculated as the fraction of surrogate coherences exceeding the observed value. Periods exceeding a coherence threshold of 0.8 and the 95th percentile of the surrogate distribution were considered significant. Also, Granger causality analysis [37] was performed on the monthly series using lags up to 12 months. Both forward (radiation to events) and reverse (events to radiation) directions were tested to assess directional predictability, with p-values below 0.05 considered indicative of significant Granger causality.

## Cross-Wavelet Coherence and Phase Analysis

To resolve time-varying periodic synchronization between GCRs radiation and viral event frequency, the author performed WTC using the `pycwt` library [38]. Both monthly time series were standardized (z-score normalization) to ensure the wavelet power reflected relative oscillations rather than absolute magnitudes. The analysis utilized a Morlet mother wavelet (dimensionless frequency  $\omega_0 = 6$ ) with a sub-octave resolution of  $dj = 1/12$  and a starting scale ( $s_0$ ) of two months. Statistical significance was established at the 95% confidence level against a red-noise background. To prevent misinterpretation of edge effects arising from the finite length of the time series, a Cone of Influence (COI) was calculated; data outside this boundary were excluded from periodic interpretation. Phase relationships were visualized via quiver plots on the coherence spectrum. The orientation of the phase arrows indicates the lead/lag relationship: right-pointing arrows signify in-phase synchronization, while left-pointing arrows signify anti-phase synchronization.

## Conditional Harmonic VECMX Projection

Prior to Vector Error Correction Model (VECM) estimation, the Johansen cointegration test was [39] applied to the monthly GCRs and viral outbreak event series using

statsmodels.tsa.vector\_ar.vecm.coint\_johansen. Thirteen lagged differences ( $k_{ar\_diff} = 13$ ), corresponding approximately to one annual cycle, and no deterministic trend ( $det\_order = 0$ ) were specified. Both trace and maximum-eigenvalue statistics were evaluated against 90%, 95%, and 99% critical values. A cointegration rank of one was identified and used in subsequent VECM estimation. To capture long-term phase relationships between GCRs and viral outbreak event counts, a conditional harmonic with exogenous variables (VECMX) [40] was implemented. Monthly series were standardized and augmented with exogenous harmonic components representing Hale (21.33-year), Schwabe (10.67-year), Quasi-Biennial Oscillation (QBO) (1.94-year), annual, semiannual cycles, informed by heliophysical theory and prior spectral coherence analysis. Each cycle was expressed using sine and cosine terms to encode phase information. The VECMX employed 13 lagged differences, a cointegration rank of one, and a constant term in the cointegration relation. Projections were generated for a 20-year horizon (2026–2046), with a 12-month rolling mean applied to emphasize dominant cyclical behavior. Predicted GCRs and viral event series were normalized using MinMaxScaler to facilitate comparison. Phase alignment between projected GCRs intensity and viral event frequency was further assessed using cross-correlation analysis of the normalized, smoothed projection series. Cross-correlation functions were computed over the full range of possible lags, and the lag corresponding to the maximum correlation was interpreted as the dominant phase offset between the two signals at monthly resolution. All analyses were conducted on a Linux system using Python 3.12 and standard scientific libraries, including pandas, numpy, matplotlib, scipy, ruptures, and statsmodels.

## Result and Discussion

Although the timing of major viral outbreaks has long been an enigma in modern medicine [4-7], this study provides a possible statistical framework for outbreak prediction. By synthesizing spectral coherence, Granger causality, and cross-wavelet coherence analyses, the result shows that global viral crises are significantly coupled with fluctuations in GCRs flux. This relationship—driven primarily by solar cycle and Sun magnetic cycle harmonic components—suggests that what was once viewed as stochastic unpredictability can possibly be a predictable temporal pattern. Here, (Figure 2) visually demonstrates a temporal alignment between peaks in the start of viral outbreak activity and intervals of elevated GCRs intensity. The (Figure 3 and Table 1) highlight a predicted surge in GCRs intensity around 2030, likely coinciding with the transition into the next 25/26 solar minimum [41,42].

In this context, viral outbreak emergence may not represent random epidemiological noise, but rather a phenomenon that exhibits synchronization with external GCRs and its solar and atmospheric modulators. As shown in the analysis, GCRs activity significantly "Granger-causes" viral events across all 12 tested lags ( $p < 0.05$ ), with the strongest significance ( $p < 0.0001$ ) occurring in the first four months. Conversely, there is no statistical significance to suggest that viral events influence GCRs flux ( $p > 0.21$ ). This lack of feedback may indicate that the relationship is unidirectional (Table 2). The GCRs may contain predictive information that precedes the viral outbreaks. The persistent significance up to a 12-month lag may suggest a priming period, where solar and Earth atmospheric conditions may influence the terrestrial environment or viral outbreak well before they reaches a detectable threshold. Also, burst

detection mapping of viral outbreak events against GCRs intensity identifies a clustering of burst occurrences during periods of low solar activity, most notably around the solar minima of approximately 2009 and 2019 (Figure 4A). Moreover, the spectral coherence analysis identifies statistically robust associations at multiple characteristic timescales. Prominent long-term periodicities corresponding to the Hale magnetic cycle (21.33 years) and the Schwabe cycle (10.67 years) exhibit high coherence values of 0.91 and 0.85, respectively, indicating a possible phase-locked behavior between solar variability and the analyzed viral outbreak signal. At shorter timescales, a high coherence is observed at the semi-annual cycle (0.50 years), with a coherence value of 0.95 ( $p = 0.000$ ), consistent with a possibly seasonal modulation processes (Figure 4B, Table 3). In addition, a coherent spectral band spanning 1.94–2.37 years aligns closely with the known periodicity of the QBO [43].

In addition, the WTC analysis provides a dynamic, time-resolved evidence for the synchronization between GCRs variability and viral outbreak events. Unlike stationary spectral methods, the wavelet framework explicitly captures how shared periodicities emerge, persist, and intermittently strengthen or weaken across the full duration of the study period. The predominance of phase-locked regions, represented by contiguous dark-red coherence domains, indicates extended intervals during which both signals evolve in a tightly coupled manner. Notably, a persistent high-coherence band centered around the 11-year periodicity extends across the temporal axis (Figure 5), indicating that the Schwabe solar cycle may act as a stable and recurrent modulator of the viral outbreak events rather than a transient or episodic influence. Also, the 20-year conditional harmonic projection (2026–2046) demonstrates a persistent and coherent phase alignment between the projected GCRs radiation signal and the projected viral outbreak trajectories. Across the forecast horizon, peaks and troughs in the projected viral outbreak event consistently track the phase evolution of the GCRs radiation harmonics. Rather than predicting the exact events, this model shows that future viral trends continue to mirror historical radiation cycles. These findings may suggest that global viral outbreaks can be organized around predictable GCRs rhythms instead of occurring at random (Figure 6A, Figure 6B). As shown in the SARIMA forecast (Table 1), GCRs flux intensity is predicted to rise steadily toward a peak around 2030, a corresponding increase in the frequency of viral outbreaks can be anticipated based on established periodic rhythms.

Independent experimental and theoretical studies provide external support for the biological plausibility of the statistical associations observed in the present analysis. Mehta et al. demonstrated that exposure to simulated GCRs, including high-energy protons and heavy ions, that can directly induce reactivation of latent human cytomegalovirus (CMV) *in vitro*, leading to a significant increase in viral copy number without requiring major genomic sequence alterations [44]. Instead, radiation exposure triggered transcriptional activation of lytic-phase viral genes, indicating that ionizing radiation can act as a functional switch from latency to active replication. These findings support the hypothesis that fluctuations in GCRs intensity may influence viral activity through reactivation or amplification mechanisms rather than *de novo* viral emergence, consistent with the lead–lag structure identified in the Granger causality and wavelet analyses. Complementary theoretical work proposed that variations in cosmic ray flux and solar activity represent underappreciated environmental drivers of emerging viral infectious diseases and argued for their integration into global early-warning surveillance systems [45].

Their study highlights that periods of solar minima coincide with several major viral emergence events. This framework aligns with this finding that viral outbreak timing may be phase-locked to dominant solar and heliomagnetic cycles, particularly the Schwabe cycle and Hale cycle. Together, these studies provide convergent biological and conceptual support for the hypothesis that GCRs variability may contain predictive information relevant to viral outbreak dynamics [44-46].

Beyond the dominant decadal solar-cycle harmonics, this analysis identifies statistically significant coherence between viral outbreak and GCRs variability within the quasi-biennial ( $\approx 1.9\text{--}2.4$  years) and annual/semi-annual cycles. The quasi-biennial periodicity aligns with the QBO, a primary mode of equatorial stratospheric variability. The QBO is known to modulate the vertical coupling between the stratosphere and troposphere, influencing global circulation and the transport of trace gases and aerosols [47-50]. The QBO modulates the dynamical coupling between the tropical stratosphere and the extratropics through the Holton–Tan effect. During the easterly QBO phase, enhanced upward propagation of planetary waves weakens the polar vortex, whereas the westerly phase is associated with a stronger, more stable vortex [49-55]. Numerous studies have shown that the atmospheric response to solar variability is phase-dependent on the QBO, with statistically significant signals often emerging when stratified by QBO phase [50-55]. While the QBO does not alter the incoming flux of GCRs, it can regulate how solar-driven perturbations are dynamically expressed within the middle and lower atmosphere [56]. Similarly, the high coherence at annual and semi-annual scales may reflect the seasonal modulation of GCRs flux. This flux is governed by the Earth's orbital position within the HCS and seasonal changes in Earth atmospheric shielding [54-57]. However, such coherence may also arise from climatologically favorable viral outbreak seasons, during which intrinsic seasonal drivers dominate transmission dynamics, potentially producing apparent synchronization independent of heliophysical forcing [58,59]. By integrating these results, a multilevel coupling framework emerges; while long-term solar variability establishes the the long term risk, shorter-term oscillations like the QBO or seasonal cycles may regulate the specific timing of viral outbreak emergence. The increased GCRs flux and its secondary particle production has been linked to changes in the polar vortex strength which can impacted by QBO phases [60-63].

Moreover, recent research indicates that the Sun is entering a period of prolonged low solar activity, potentially marking the onset of a modern Grand Solar Minimum. The Sun has completed solar cycle 24, which was the weakest cycle observed in over a century, and solar cycle 25, which began in 2020, has exhibited a notably slow start in generating active regions and flares [64]. During 2020, the Sun experienced 115 spotless days (78% of the year), exceeding previous space-age records and indicating a continued suppression of sunspot formation. If this trend persists, the extended periods of minimal solar activity will correspond to conditions analogous to historical grand solar minima, such as the Spörer (ca. 1440–1460), Maunder (ca. 1687–1703), and Dalton (ca. 1809–1821) minima [64-67]. Prolonged low solar activity, increases GCRs flux, which may partly explain the occurrence of the two major 21st-century pandemics—Swine Flu (2009) and COVID-19 (2019)—both coinciding with solar minima and separated by approximately 10 years.

Nevertheless, while the combined SARIMA, spectral coherence, and conditional harmonic VECMX framework offers a mechanistically informed approach to modeling GCR–viral event dynamics, several limitations must be acknowledged. Both SARIMA and VECMX are fundamentally linear and rely on stationarity of differenced series; consequently, unmodeled nonlinear interactions, regime shifts, or structural breaks—arising from abrupt solar variability, climatic transitions, or epidemiological changes—may bias parameter estimates and inferred associations. The conditional harmonic VECMX further constrains system dynamics by imposing phase alignment with predefined solar and seasonal cycles, which, although physically motivated, may fail to capture transient, stochastic, or non-canonical solar influences and complex heliospheric–atmospheric couplings. Interpolation of missing data in both GCRs and outbreak time series may introduce artificial smoothness, induce spurious trends, or attenuate short-term variability, thereby affecting time- and frequency-domain analyses. Spectral coherence and Granger causality inferences are sensitive to surrogate construction, lag selection, windowing, and significance thresholding, and extensive testing across scales and frequencies increases the risk of inflated nominal significance despite correction efforts. Long-horizon projections, particularly those extending to 20 years, remain highly sensitive to unmodeled exogenous changes, including shifts in surveillance practices, public health interventions, viral evolution, climate dynamics, and future solar behavior, and should therefore be interpreted as phase-informed scenario trajectories rather than deterministic predictions. Also, viral outbreak data derived from EM-DAT—especially in the pre-2000 period—are subject to substantial reporting biases which may distort long-term trends and further bias estimated relationships. Finally, to substantiate any causal interpretation, targeted laboratory or experimental research is highly required to assess whether GCR-associated atmospheric ionization or perturbations can meaningfully influence viral persistence, activation, or host susceptibility.

## **Conclusion**

This study shows that global viral outbreak frequency exhibits statistically significant phase coherence with GCRs flux, implying a possible structured temporal organization rather than purely stochastic behavior. This coherence corresponds to dominant heliophysical periodicities, including the solar cycle and Hale magnetic cycle. While these findings may suggest that viral activity may be conditioned by interacting heliophysical and atmospheric rhythms acting as background modulators alongside established ecological and epidemiological drivers, further mechanistic, experimental, and epidemiological studies are required to substantiate and interpret these associations.

## **Declarations**

### **Conflict of Interest**

The author declares that there are no conflicts of interest.

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### **Ethical Approval and Institutional Review Board (IRB)**

Ethical approval was not required for this study. The research involves the secondary analysis of publicly available, aggregated, and de-identified environmental and epidemiological data. No human participants were involved, and no identifiable personal data were accessed or processed.

### **Author Contributions**

Alamin Mustafa solely contributed to the conceptualization, methodology, investigation, data curation, formal analysis, resources, software, validation, visualization, project administration, supervision, and writing (original draft and review & editing).

### **Data Availability:**

The raw data supporting the findings of this study are available from the Oulu Cosmic Ray Station and the EM-DAT database. The specific datasets processed and analyzed for this study are available from the corresponding author upon reasonable request.

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## Tables

**Table 1: Decade-Ahead SARIMA Forecast (2026–2035) of Predicted GCRs Flux Intensity with 95% Confidence Intervals**

Year	Predicted Value [cts/min]	Lower 95% CI	Upper 95% CI	Feature
2026	5703.49	5404.72	6002.25	
2027	5779.74	5273.77	6285.71	
2028	5828.32	5180.48	6476.16	
2029	5870.19	5108.36	6632.03	
<b>2030</b>	<b>5876.80</b>	<b>5017.65</b>	<b>6735.95</b>	<b>Peak</b>
2031	5868.73	4923.76	6813.70	
2032	5789.41	4767.28	6811.55	
2033	5657.03	4564.44	6749.63	
2034	5509.97	4352.37	6667.57	
2035	5426.33	4208.29	6644.36	

**Table 2: Summary of Granger Causality Tests for GCRs Levels and Viral Outbreak Events across 12 Lags**

Lag	Radiation → Events (p-value)	Events → Radiation (p-value)
1	0.0000***	0.8422
2	0.0000***	0.8007
3	0.0000***	0.2750
4	0.0000***	0.3552
5	0.0003***	0.3473
6	0.0008***	0.4236
7	0.0014**	0.4635
8	0.0010**	0.5079
9	0.0032**	0.3634
10	0.0030**	0.4197
11	0.0059**	0.5030
12	0.0375*	0.2140

**Table 3: Spectral Coherence Analysis Identifying Significant Periodicities and Possibly Associated with Solar/Atmospheric Cycles**

Period (Years)	Coherence	P-Value	Possible Cycle Description
21.33	0.91	0.001	Hale Cycle (Solar Magnetic)
10.67	0.85	0.007	Schwabe Cycle (11-Year Solar)
2.37	0.81	0.010	Quasi-Biennial Oscillation (QBO)
2.13	0.83	0.003	Quasi-Biennial Oscillation (QBO)
1.94	0.89	0.003	Quasi-Biennial Oscillation (QBO)
1.02	0.84	0.004	Annual Cycle
0.97	0.81	0.001	Annual Variation
0.65	0.81	0.007	Intra-annual signal
0.51	0.86	0.005	Semi-annual signal
0.50	0.95	0.000	Semi-annual Cycle
0.37	0.82	0.003	seasonal harmonic
0.30	0.83	0.003	seasonal harmonic
0.30	0.91	0.001	seasonal harmonic
0.30	0.92	0.002	seasonal harmoni
0.29	0.91	0.002	High-frequency variation
0.24	0.89	0.000	Quarterly signal (~3 months)
0.21	0.84	0.009	High-frequency jitter
0.21	0.80	0.009	High-frequency jitter

## Figures

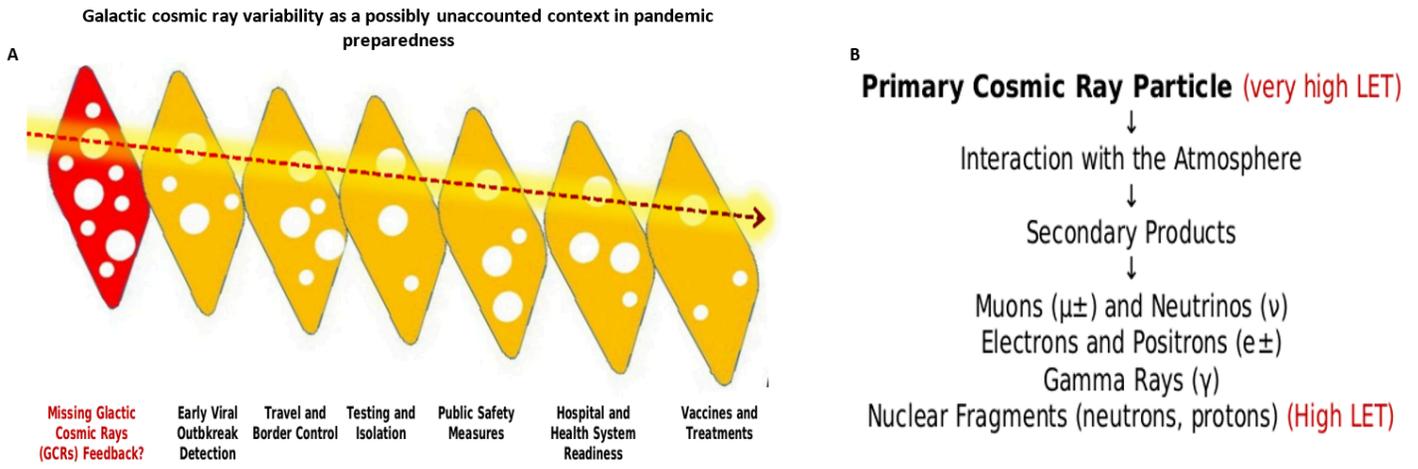


Figure 1

A: Missing GCRs Inputs in Pandemic Response, B: Linear Energy Transfer Characteristics of Primary and Secondary Cosmic-Ray Particles

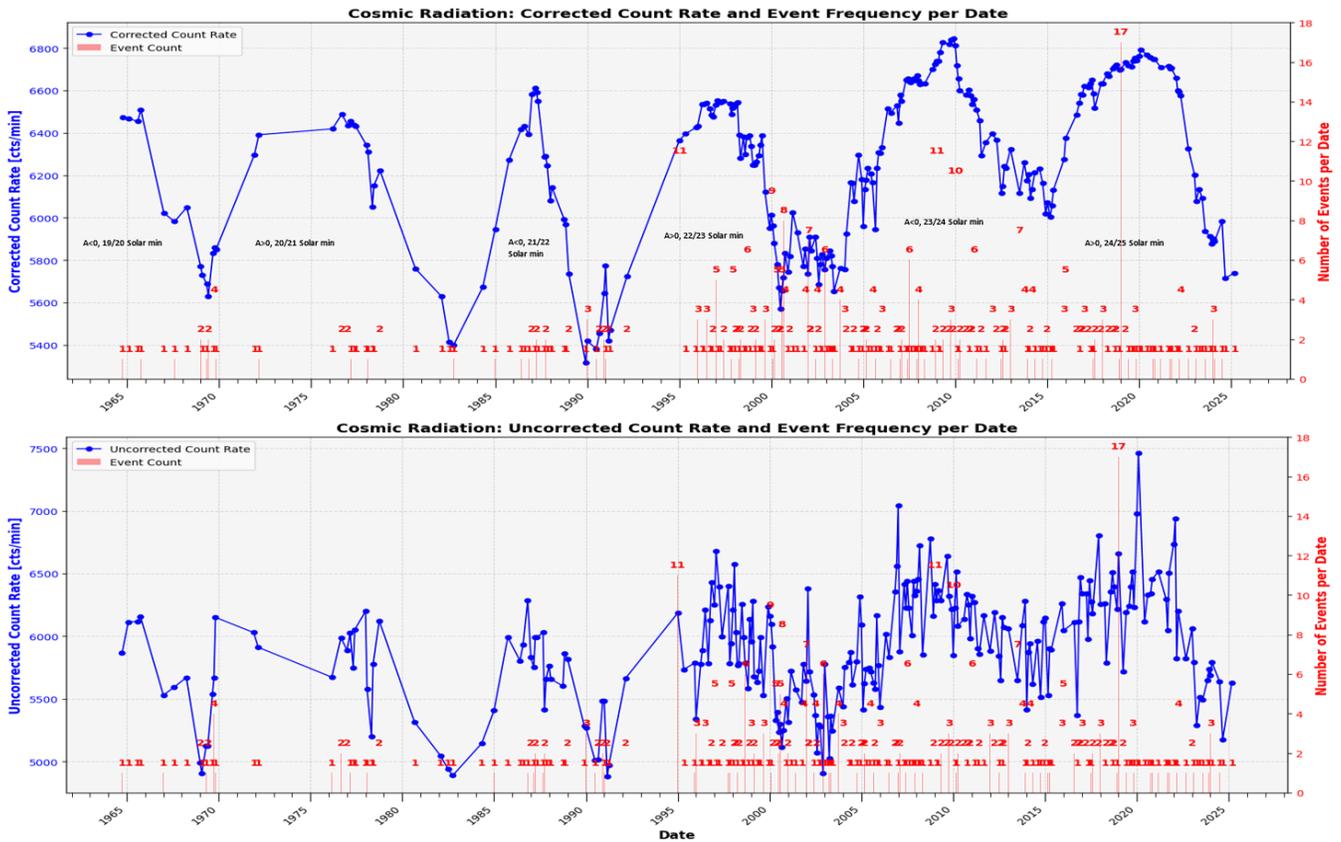


Figure 2

Temporal Co-variation Between Cosmic Rays Radiation Intensity and Viral Outbreak Frequency

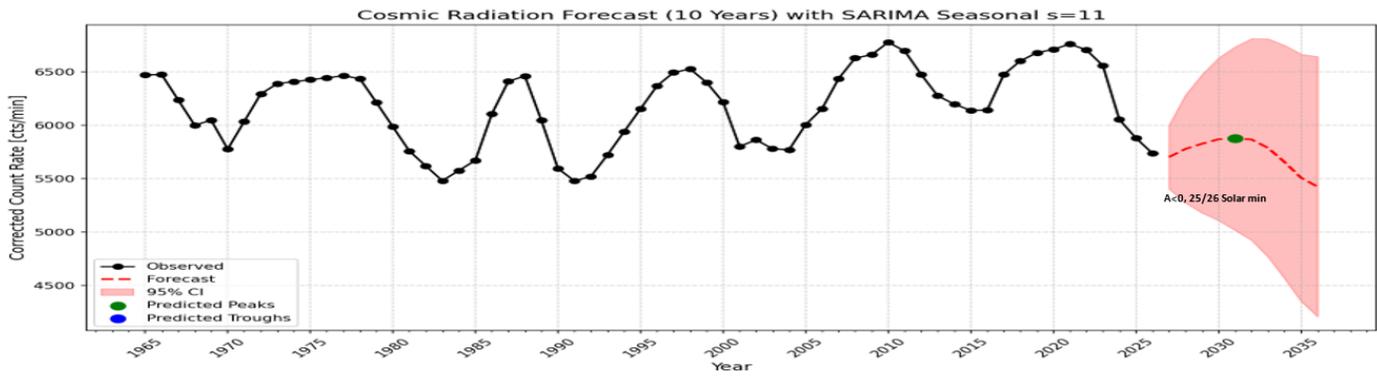


Figure 3

GCRs Forecast Model with 95% Confidence Interval and Peak Predictions

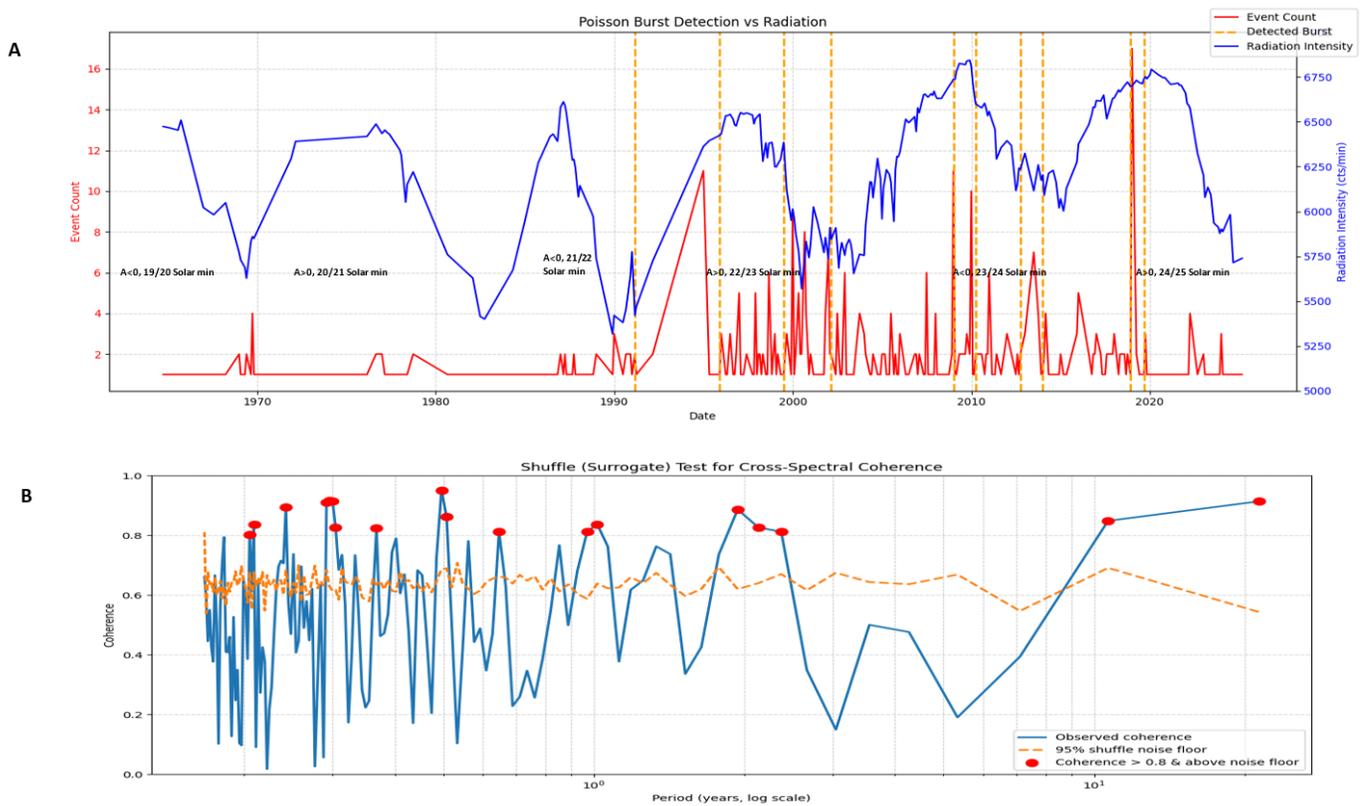


Figure 4

A; Poisson Burst Prediction Mapping of Viral Outbreak Events against GCRs Intensity Flux. B; Cross-Spectral Coherence Analysis with Shuffle Surrogate Noise Floor Testing for Periodic Synchronization Validation.

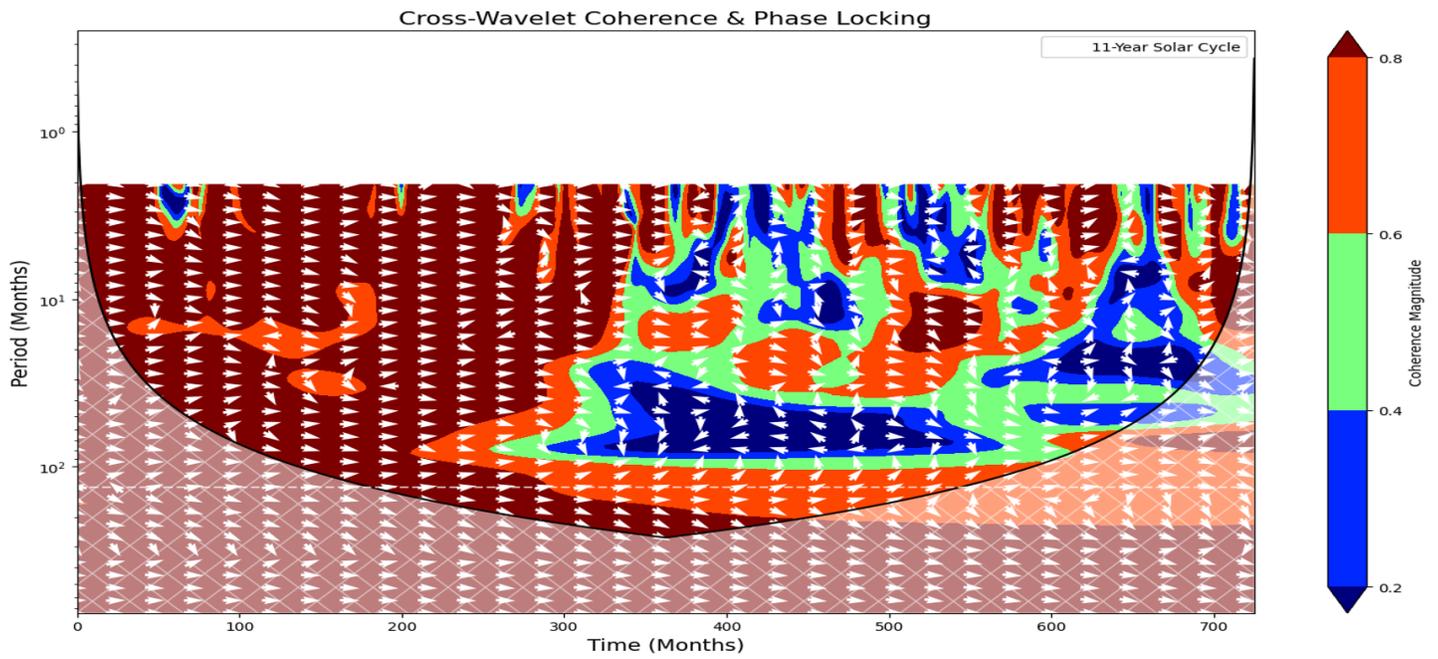


Figure 5

Cross-Wavelet Coherence and Phase Locking between Monthly GCRs and Viral outbreak Event Activity.

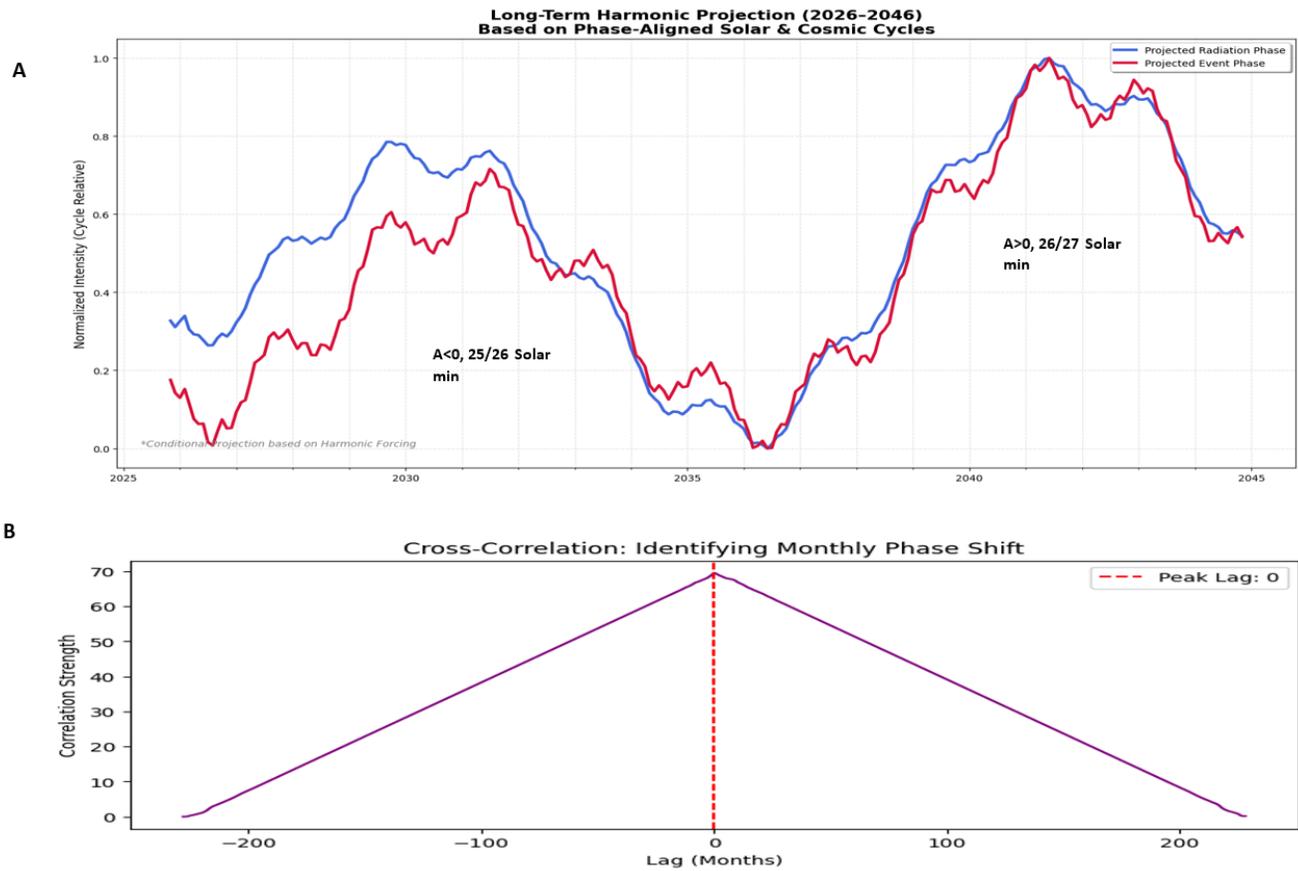


Figure 6

**A; Long-Term Harmonic VECMX Projection (2026–2046) of GCRS and Predicted Viral outbreak Event Phases. B; Cross-Correlation Profile Identifying Zero-Month Phase Lag for Synchronous Variation Between GCRs and Viral Outbreak Frequency**