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Perspective

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Long COVID and Cancer Biology: Distinguishing Mechanistic Overlap from Evidence Across Cancer-Related Outcomes

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Abstract

Long COVID is characterized by persistent immune, inflammatory, vascular, and metabolic abnormalities that overlap with cancer-relevant pathways. These observations have prompted hypotheses regarding cancer, but de novo incidence, recurrence or progression of pre-existing disease, and treatment efficacy, toxicity, and tolerance (including treatment modification or interruption) are distinct outcomes and warrant separate evaluation. This Perspective evaluates these outcomes separately. Current Long COVID-specific evidence remains insufficient to determine whether de novo cancer incidence is increased, unchanged, decreased, or varies across patient subgroups. Experimental and observational studies after respiratory viral infection raise credible questions about dormant or established malignancy, but they are not equivalent to evidence from clinically characterized Long COVID cohorts. Evidence regarding anticancer treatment outcomes is also limited and heterogeneous. Pandemic-era and SARS-CoV-2 infection cohorts provide contextual information but cannot substitute for longitudinal studies comparing clinically defined Long COVID with matched recovered controls. Apparent post-pandemic trends in cancer incidence may reflect disruptions in screening and diagnosis, while the long latency of many cancers limits biological inferences from short follow-up periods. Outcome-specific follow-up should specify the Long COVID definition, baseline cancer risk, latency, and cancer endpoints. Current evidence therefore leaves uncertainty about the direction or magnitude of any Long COVID-specific effect on cancer incidence, recurrence, progression, or treatment efficacy, toxicity, and tolerance.

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Keywords:
long COVID; cancer biology; carcinogenesis; immune dysregulation; chronic inflammation; tumor progression

1. Introduction

Long COVID is a heterogeneous post-acute condition characterized by symptoms and diagnosable conditions arising from combinations of immune, autonomic, vascular, metabolic, and tissue-level abnormalities [1,2]. For this Perspective, Long COVID is defined as a post-SARS-CoV-2 condition present for at least 3 months, consistent with the 2024 National Academies definition [3]. The World Health Organization employs a related definition in which symptoms usually begin within 3 months and last at least 2 months [4]. Long COVID is the term adopted throughout this Perspective. Because study definitions vary, cohorts identified by post-acute diagnoses or sequelae are not necessarily equivalent to cohorts with clinically defined Long COVID.

Primary Long COVID studies have documented persistent inflammatory signaling, immune dysregulation, complement abnormalities, and T-cell activation or exhaustion [5–8]. These findings converge with cancer biology [9]. A peer-reviewed review discusses possible oncogenic or tumor-promoting effects of SARS-CoV-2-related biology [10], whereas a recent review emphasizes

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mechanistic convergence and uncertainty [11]. Neither establishes a clinically defined Long COVID-specific cancer effect.

A pathway may be relevant to cancer without being carcinogenic, and a biological state may influence an existing tumor without initiating a new malignancy. This Perspective therefore evaluates three questions separately: de novo cancer incidence, recurrence or progression of pre-existing malignancy, and anticancer treatment efficacy, toxicity, and tolerance (including treatment modification or interruption).

A targeted PubMed search was updated through 17 September 2026 using combinations of the terms Long COVID or post-COVID condition with cancer, carcinogenesis, incidence, recurrence, metastasis, treatment response, immune dysregulation, inflammation, viral persistence, and tumor dormancy. Primary human, longitudinal, and mechanistic studies were prioritized; reviews were consulted for context and citation chaining. This was a narrative, non-systematic search without protocol registration, formal risk-of-bias assessment, or evidence grading.

2. An Outcome-Specific Evidence Framework

Established oncogenic viruses have been linked to cancer through converging epidemiologic, molecular, and tissue-specific evidence, including persistent infection, tumor associations, effects on oncogenes or tumor-suppressor pathways, genomic instability, immune suppression, and chronic inflammation [12]. For SARS-CoV-2 and Long COVID, cancer-relevant pathway activity is mechanistic context; each clinical outcome necessitates its own direct evidence.

Figure 1 illustrates an outcome-specific framework rather than a single sequential causal pathway. Shared biology and persistence may motivate hypotheses, but de novo incidence, recurrence or progression, and treatment efficacy, toxicity, and tolerance are parallel questions. De novo incidence concerns new primary cancer in people without known malignancy; recurrence or progression begins with malignant cells already present; treatment studies evaluate efficacy, toxicity, and tolerance, including treatment modification or interruption. Evidence in one domain cannot establish another.

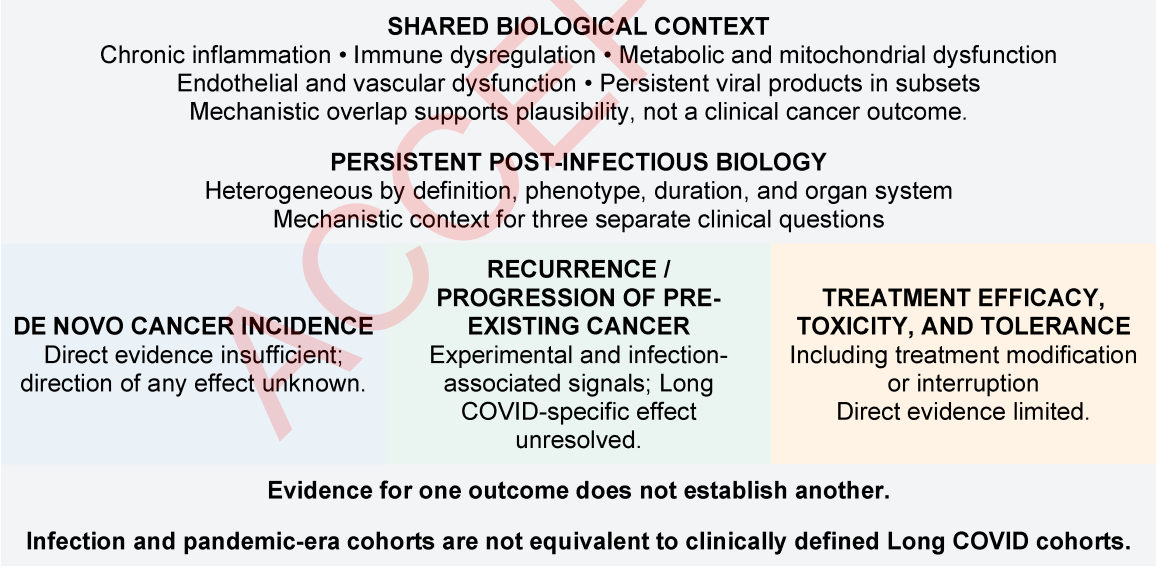


Figure 1: Outcome-specific evidence framework for Long COVID and cancer biology. Shared cancer-relevant mechanisms and heterogeneous persistent post-infectious biology provide mechanistic context for three separate clinical questions: de novo cancer incidence, recurrence or progression of pre-existing malignancy, and treatment efficacy, toxicity, and tolerance (including treatment modification or interruption). Evidence in one outcome domain does not establish another. Pandemic-era or SARS-CoV-2 infection cohorts provide contextual information but are not equivalent to clinically defined Long COVID cohorts.

Persistent viral products illustrate the importance of distinguishing direct from indirect evidence. In a blinded, two-year study, circulating SARS-CoV-2 antigen was more frequently detected in participants with Long COVID than in recovered participants at 6–12 months. However, at 18–24 months, antigen positivity was 3.5% in participants with Long COVID, 0% in recovered participants, and 1.5% in uninfected controls [13]. Late antigen positivity was low across all three groups, including a small proportion of uninfected controls. Addi-

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tional studies detected circulating spike in some participants with persistent post-acute symptoms, as defined in the study [14], as well as gut antigen persistence after acute COVID-19 [15]. These findings support persistence in subsets but do not establish a cancer outcome.

3. Cancer-Relevant Biology in Long COVID: Mechanistic Evidence and Limits

Primary Long COVID studies have documented sustained immune activation, T-cell dysregulation or exhaustion, inflammatory abnormalities, and complement-associated thromboinflammatory signals [5–8]. In cancer, inflammatory and immune states may affect tumor growth, immune escape, and the microenvironment [9]. Whether a Long COVID immune phenotype is clinically relevant to cancer therefore depends on tissue, duration, cell type, tumor context, and the outcome studied.

Metabolic and mitochondrial dysfunction provides another area of overlap. A skeletal-muscle study in Long COVID documented reduced exercise capacity and post-exertional worsening with local and systemic metabolic abnormalities [16]. Cancer also alters cellular energetics, but reduced oxidative capacity in symptomatic muscle does not reflect the metabolic adaptations that support malignant-cell survival and proliferation.

Endothelial and microvascular abnormalities, oxidative stress, senescence-associated pathways, and viral persistence have also been reported or proposed in Long COVID [2,8,13–15]. These observations extend mechanistic plausibility but remain upstream of cancer-specific clinical endpoints.

Long COVID heterogeneity further confounds inference. Dominant phenotypes and definitions vary across studies [1–4]. Combining symptom-defined Long COVID, diagnosis-code cohorts of post-acute sequelae, and post-hospital sequelae may mix clinically and biologically different populations. Cancer studies should therefore clearly document the operational definition of Long COVID, acute infection severity, vaccination and reinfection history, symptom duration, and relevant phenotypic characteristics.

4. Clinical Evidence by Cancer-Related Outcome

4.1. De Novo Cancer Incidence: Long COVID-Specific Evidence Is Insufficient

In this targeted, nonsystematic search, no directly relevant study was located comparing site-specific incident cancer in a clinically defined Long COVID cohort with matched SARS-CoV-2 survivors who recovered without Long COVID. The appropriate inference is uncertainty: current evidence cannot determine whether Long COVID-specific cancer incidence is increased, unchanged, decreased, or subgroup-specific.

The population and infection studies below provide contextual rather than direct evidence regarding Long COVID. A US SEER-22 analysis found that overall cancer incidence in 2021 was lower than in 2018–2019; increases observed for nine rare cancer types were likely explained by changes in cancer reporting standards [17]. A veteran cohort documented a lower observed hazard of cancer diagnosis after a positive COVID-19 test, a result vulnerable to survivor, healthcare-utilization, testing, and ascertainment biases [18]. An Italian cohort reported increased diagnoses during the pandemic while noting delayed diagnosis and screening disruption [19]. None of these studies clinically phenotyped Long COVID as the exposure.

Qian and colleagues reported an association between hospitalization for severe COVID-19 and subsequent lung cancer, and demonstrated accelerated lung tumor growth after respiratory viral infection in murine models [20]. This finding represents an important post-infectious signal; however, the human cohort comprised patients hospitalized with severe COVID-19 rather than patients with clinically characterized Long COVID, and the mechanistic investigation focused specifically on lung tissue. Therefore, it does not provide a Long COVID-specific estimate of cancer incidence.

These studies demonstrate why infection history, acute severity, screening behavior, healthcare utilization, and Long COVID status must be separated analytically. They do not resolve the Long COVID-specific incidence question, and not identifying a direct study in this search is not evidence for either increased or unchanged risk.

4.2. Effects on Pre-Existing or Dormant Malignancy Are a Distinct Question

The strongest experimental evidence linking respiratory viral infection with cancer biology pertains to malignant cells that already exist. In mice, influenza and SARS-CoV-2 triggered interleukin-6 (IL-6)-dependent awakening and expansion of dormant disseminated breast cancer cells in the lung. In human analyses from the same study, breast-cancer patients who developed COVID-19 had an adjusted hazard ratio of 1.44 (95% CI, 1.01–2.05) for subsequent lung metastasis; after additional adjustment, the estimate was 1.41 (95% CI, 0.97–2.04) and was no longer statistically significant [21].

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These findings support the possibility that respiratory inflammation may influence dormant or established malignant cells. They do not demonstrate transformation of normal cells, and the human exposure was infection after cancer diagnosis rather than Long COVID. Long COVID-specific recurrence studies therefore necessitate cancer-survivor cohorts with explicit tumor type, residual-disease risk, infection severity, Long COVID definition, and follow-up.

Recurrence or metastatic progression and de novo incidence should not be conflated. The former begins with known or presumed malignant cells; the latter begins without known cancer. Their cohorts, confounders, endpoints, and latency assumptions differ.

4.3. Treatment Efficacy, Toxicity, and Tolerance (Including Treatment Modification or Interruption)

Post-acute morbidity in patients with cancer does not, by itself, establish effects on treatment efficacy, toxicity, or tolerability. A 2026 cohort study involving 76,807 patients with cancer evaluated post-acute diagnoses and symptoms following Omicron infection [22]. These study-defined post-acute outcomes should not be assumed to represent clinically defined Long COVID. Rather, they reflect the burden of post-acute illness and do not establish effects on anticancer treatment efficacy, toxicity, or tolerability.

The OnCovid registry more directly examined cancer care: among 466 patients receiving systemic anticancer therapy before COVID-19, 15.0% permanently discontinued therapy and 38.2% resumed with a dose or regimen modification; permanent discontinuation was associated with higher mortality [23]. This observational cohort from the early pandemic cannot distinguish the biological effects of Long COVID from those of acute illness severity, treatment interruptions, or other confounding factors. A small lung-cancer study identified immune-microenvironment changes after SARS-CoV-2 infection without impaired long-term progression-free survival on checkpoint inhibitors [24]. A review has proposed possible effects of persistent inflammation on immunotherapy response [25]. Prospective cohorts evaluating treatment efficacy, toxicity, and tolerance (including treatment modification or interruption) in clinically defined Long COVID remain necessary.

The principal claims and the evidence needed to support them are summarized in Table 1.

Table 1: Evidence boundaries for proposed Long COVID-cancer relationships.

Question	Direct Long COVID Evidence and Endpoint	Contextual Evidence	Main Uncertainty
Shared cancer-relevant biology	Biological abnormalities: primary studies document immune, inflammatory, metabolic, vascular, complement, and antigen-related abnormalities in subsets of Long COVID. These are not cancer outcomes.	Supports mechanistic overlap and biological plausibility.	Which abnormalities, if any, produce tumor-specific effects, in which tissues and phenotypes.
De novo cancer incidence	Cancer outcome: the targeted, nonsystematic search did not identify a directly relevant study of site-specific incident cancer comparing clinically defined Long COVID with matched recovered survivors.	Pandemic and infection cohorts are mixed; a 2026 severe-COVID study reported a lung-cancer signal with supporting murine mechanisms.	Whether clinically defined Long COVID changes site-specific incidence over adequate follow-up.
Recurrence or progression of pre-existing cancer	Cancer outcome: the targeted, non-systematic search did not identify a directly relevant prospective study comparing recurrence or progression in cancer survivors with clinically defined Long COVID against comparable recovered survivors without Long COVID.	Respiratory viral infection can alter dormant or established malignant-cell behavior in experimental models; human signals are limited.	Whether Long COVID changes recurrence or progression in defined cancer-survivor populations.

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Table 1: *Cont.*

Question	Direct Long COVID Evidence and Endpoint	Contextual Evidence	Main Uncertainty
Treatment efficacy, toxicity, and tolerance (including treatment modification or interruption)	Treatment outcomes: direct evidence for efficacy, toxicity, and tolerance in clinically defined Long COVID remains limited. Post-acute symptoms and biological abnormalities alone do not establish a treatment effect.	Cancer cohorts after SARS-CoV-2 infection show sequelae and treatment changes; a small immunotherapy cohort found immune changes without impaired long-term progression-free survival.	Whether Long COVID independently affects treatment efficacy, toxicity, and tolerance (including treatment modification or interruption).
Pandemic-era cancer trends	No direct Long COVID exposure: calendar-period trends do not measure a clinically defined Long COVID cohort or a Long COVID-specific cancer outcome.	Useful for identifying health-system effects, diagnostic disruption, and hypotheses.	How much temporal change reflects biology versus screening, diagnosis, healthcare utilization, registry effects, or infection severity.

Legend: Direct Long COVID biological evidence documents abnormalities in participants with Long COVID; it does not establish a cancer outcome. Direct cancer-outcome evidence necessitates that the specific outcome be evaluated in a clinically defined Long COVID cohort with an appropriate comparator. Statements that a study was not located refer to the targeted, nonsystematic search updated through 17 September 2026 and do not establish that no such study exists. Contextual pandemic-era, infection, and post-acute sequelae cohorts are not treated as equivalent to clinically defined Long COVID cohorts. The table describes study design and directness without assigning formal certainty grades.

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5. Interpreting Post-Pandemic Cancer Trends and Latency

The pandemic disrupted cancer epidemiology through interrupted screening, delayed procedures, altered diagnostic pathways, healthcare avoidance, and registry changes. These effects varied by cancer type, region, age, and health system. A rise in diagnoses after disruption may therefore reflect delayed detection of prevalent disease rather than new biological initiation [17,19].

Latency also varies by cancer type and evolutionary stage. In pancreatic cancer, genomic reconstruction estimated that at least a decade elapsed between the initiating mutation and the emergence of the parental non-metastatic founder clone, followed by several additional years before metastatic capability developed [26]. This illustrates why short follow-up cannot resolve all delayed outcomes, while early increases may reflect detection effects. Stronger inference necessitates individual-level infection and Long COVID phenotyping, baseline cancer-risk assessment, adequate follow-up, and recovered controls.

Temporal sequence alone does not determine causation. Attribution depends on an appropriate comparator, control of confounding, a plausible latency structure, and outcome-specific evidence.

6. Research Priorities for Resolving the Separate Outcomes

Research should be structured around specific outcomes rather than a single concept of cancer risk. For de novo incidence, the priority is a longitudinal cohort comparing patients with well-characterized Long COVID with matched recovered SARS-CoV-2 survivors, excluding active cancer at baseline. Matching or adjustment should include age, sex, smoking, obesity, baseline cancer risk, acute severity, vaccination, reinfection, healthcare utilization, and screening participation.

Cancer endpoints should be verified through cancer registries and analyzed by cancer site. New primary cancer, recurrence, metastatic progression, treatment efficacy, toxicity, and tolerability—including treatment modifications or interruptions—should be assessed as distinct outcomes. Mechanistic biomarkers are most informative when prespecified and linked to an outcome-specific intermediate or clinical endpoint rather than used as surrogate evidence of cancer risk.

Cancer-survivor cohorts should investigate recurrence and tumor dormancy while accounting for time since treatment, residual disease risk, tumor subtype, infection severity, the definition of Long COVID, and inflammatory trajectories. Treatment cohorts should prospectively evaluate treatment efficacy, toxicity, and

tolerance (including treatment modification or interruption). Long COVID phenotype and duration should be documented rather than inferred from a general post-COVID label.

This Perspective does not propose a Long COVID-specific cancer-screening strategy. Such a strategy cannot be inferred from mechanistic overlap or the contextual studies reviewed here. Any future surveillance recommendation would depend on outcome-specific evidence and evaluation within established cancer-specific guideline processes.

7. Conclusions

Long COVID and cancer biology converge through inflammatory, immune, metabolic, vascular, and persistence-related pathways, but several clinical questions remain unresolved. Direct Long COVID-specific incidence data remain insufficient to determine whether de novo cancer risk differs from that of matched recovered controls. Experimental and infection-associated studies support further investigation of tumor dormancy and established malignancies, whereas evidence regarding treatment-related outcomes remains limited and heterogeneous. These represent distinct research questions rather than a single causal sequence.

Future studies should employ explicit Long COVID definitions, recovered controls, adequate latency, baseline cancer-risk assessment, and outcome-specific endpoints. The current evidence warrants neither reassurance nor alarm but instead underscores substantial uncertainty. Although mechanistic overlap and selected post-infectious signals have been documented, whether Long COVID affects specific cancer outcomes, which populations may be affected, and over what time frame remain insufficiently studied.

List of Abbreviations

CI	Confidence Interval
COVID-19	Coronavirus Disease 2019
IL-6	Interleukin-6
SARS-CoV-2	Severe Acute Respiratory Syndrome Coronavirus 2

Author Contributions

The author is solely responsible for the conceptualization, writing—original draft, visualization, writing—review and editing. The author has read and agreed to the published version of the manuscript.

Consent for Publication

Not applicable. This Perspective contains no identifiable individual patient information.

Conflicts of Interest

The author declares no conflicts of interest.

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AI Declaration

The author did not use generative AI or AI-assisted tools to prepare the manuscript previously submitted for editorial review. OpenAI ChatGPT (Codex, GPT-5.6) assisted with implementing the requested editorial revisions, preparing the revised schematic, and drafting the response to the editor for the present revision. The author takes full responsibility for the accuracy, originality, integrity, and scientific content of the manuscript. The author did not use AI tools for the original modifications made in Microsoft Paint.

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