A Report on "1-year Risks of Cancers Associated with COVID-19 Vaccination: A Large Population-based Cohort Study in South Korea" by Kim et al. (2025)

Reviewer 2

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I am wiser than this person; for it is likely that neither of us knows anything fine and good, but he thinks he knows something when he does not know it, whereas I, just as I do not know, do not think I know, either. I seem, then, to be wiser than him in this small way, at least: that what I do not know, I do not think I know, either.

Plato, *The Apology of Socrates*, 21d

To err is human. All human knowledge is fallible and therefore uncertain. It follows that we must distinguish sharply between truth and certainty. That to err is human means not only that we must constantly struggle against error, but also that, even when we have taken the greatest care, we cannot be completely certain that we have not made a mistake.

Karl Popper, 'Knowledge and the Shaping of Reality'

Overview

Citation: Kim, H. J., Kim, M. H., Choi, M. G., and Chun, E. M. (2025). 1-year Risks of Cancers Associated with COVID-19 Vaccination: A Large Population-based Cohort Study in South Korea. *Biomarker Research*. Vol. 13, No. 114.

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Abstract Summary: This large-scale population-based retrospective study in Seoul, South Korea, estimated the cumulative incidences and subsequent risks of overall cancers one year after COVID-19 vaccination using data from over 8.4 million individuals. The study found that COVID-19 vaccination was significantly associated with an increased risk of six specific cancer types, with associations varying by age, sex, and vaccine type.

Key Methodology: Large-scale population-based retrospective cohort study using the Korean National Health Insurance database (8,407,849 individuals), propensity score matching (PSM), and multivariable Cox proportional hazards models to assess cancer risks (Hazard Ratios, *HR*s).

Research Question: What are the cumulative incidences and subsequent risks of overall cancers one year after COVID-19 vaccination in a large population-based cohort in South Korea?

Summary

Is It Credible?

Kim et al. present a large retrospective cohort study claiming that COVID-19 vaccination is associated with a significantly increased risk of six specific cancers—thyroid, gastric, colorectal, lung, breast, and prostate—within one year of administration. Using data from the Korean National Health Insurance database, the authors report hazard ratios ranging from 1.20 to 1.69 for these malignancies (p. 2). Based on these findings, they suggest that clinicians should prioritize monitoring for gastric cancer in relation to booster doses (p. 3).

The credibility of these claims is severely compromised by a fundamental conflict between the study's timeframe and the known biology of cancer. Solid tumors such as lung, colorectal, and prostate cancer typically require years, often decades, to develop from initiation to clinical detectability. The assertion that vaccination could cause a 53% increase in lung cancer or a 69% increase in prostate cancer within a single year is biologically implausible as a causal phenomenon (p. 2). It is virtually certain that the excess cases identified were pre-existing, asymptomatic tumors detected incidentally. This phenomenon, known as surveillance bias, occurs because vaccination involves interaction with the healthcare system, potentially leading to additional screening, physical exams, or imaging. For instance, mRNA vaccines are known to cause axillary lymphadenopathy, which can mimic breast or lung malignancies on imaging, triggering biopsies that detect unrelated, pre-existing cancers.

The study's own data supports the surveillance bias hypothesis over a causal mechanism. The cumulative incidence curves diverge almost immediately, with significant differences appearing as early as one month post-vaccination (Supplementary File 2, p. 2). Furthermore, the age-stratified data for thyroid cancer reveals an anomalous pattern where incidence is highest in the youngest cohort (under 65) and decreases

with age, contradicting the established epidemiological fact that cancer risk increases with age (Supplementary File 3, p. 6). This inversion strongly suggests that the observed "risk" is a function of detection intensity—likely higher in younger populations or those with more reactive immune responses—rather than carcinogenesis.

Methodological inconsistencies further weaken the article's conclusions. The analysis of booster doses contradicts the main findings; in the booster cohort, the increased risks for thyroid, colorectal, lung, breast, and prostate cancers disappear, becoming statistically non-significant (p. 3). The authors pivot to highlighting a new association with pancreatic cancer in this sub-analysis, but they fail to adequately explain why the strong signals observed in the primary analysis vanish upon re-exposure to the antigen. Additionally, the exclusion criteria appear to have been applied asymmetrically; the article explicitly states that vaccinated individuals with a cancer history within one year were excluded, but does not confirm the same exclusion for the unvaccinated group (Supplementary File 1, p. 7). While this specific asymmetry might theoretically bias results against the observed finding, the lack of clarity regarding cohort construction raises significant doubts about the comparability of the groups. Ultimately, the study likely documents an increase in cancer *diagnoses* driven by healthcare utilization, not an increase in cancer *incidence* caused by vaccination.

The Bottom Line

The claim that COVID-19 vaccination causes rapid increases in solid tumors within one year is not credible. The short timeframe is biologically incompatible with the development of new cancers, indicating that the study has likely measured the increased detection of pre-existing tumors (surveillance bias) rather than true incidence. Furthermore, the disappearance of these risks in the booster dose analysis suggests the primary findings are statistical or methodological artifacts rather than

robust biological effects.

Potential Issues

Ambiguous application of exclusion criteria: The study's methodology for cohort selection appears to apply a critical exclusion criterion asymmetrically, which could introduce a significant bias. The methods section and study flowchart state that individuals with a "prior medical history of overall cancers within 1-year based on index date" were excluded from the vaccinated group (Supplementary File 1, p. 7; Figure S1A, p. 6). However, this same exclusion is not explicitly mentioned for the unvaccinated control group. If individuals with a recent cancer diagnosis were included in the unvaccinated cohort but excluded from the vaccinated cohort, this would create a strong bias against finding an increased cancer risk in the vaccinated group. While the study used propensity score matching on the Charlson Comorbidity Index (CCI), which includes cancer as a component, the lack of explicit confirmation that this key exclusion criterion was applied to both groups represents a significant failure of methodological transparency that complicates the interpretation of the study's foundational comparison.

High susceptibility to surveillance bias and biologically implausible timeframe: The study's findings are highly susceptible to surveillance bias, and the one-year follow-up period is biologically implausible for the development of de novo solid tumors. The authors acknowledge these critical limitations, stating that the "one-year follow-up period is relatively short for evaluating cancer incidence" and that the "possibility of reverse causation or surveillance bias cannot be excluded" (Supplementary File 1, p. 4). The act of vaccination involves healthcare interactions that may increase medical surveillance and lead to the diagnosis of pre-existing, asymptomatic cancers. This is the most plausible explanation for the observed associations, particularly the immediate divergence in cancer incidence seen at just one month post-vaccination (Supplementary File 2, p. 6, Table S4). The study's conclusion of an association is therefore undermined by the high probability that the results re-

flect increased detection rather than a true increase in cancer incidence.

Failure to test the surveillance bias hypothesis: Despite acknowledging surveillance bias as a key limitation, the study omits a standard analysis that could have tested this alternative explanation for its findings. If the observed associations were due to increased medical detection, the effect would likely be stronger for cancers commonly found through screening (e.g., prostate, breast, thyroid) than for those typically diagnosed based on symptoms. The study's data appears to follow this pattern, with significant hazard ratios for screen-detected cancers but not for others like brain cancer (Figure 1A, p. 2). A formal comparison of these cancer types would have been a logical step to assess the plausibility of surveillance bias. The failure to perform or discuss such an analysis represents a missed opportunity to rigorously evaluate the most likely confounder.

Contradictory findings in the booster dose analysis: The sub-analysis comparing boosted versus non-boosted individuals produces results that are inconsistent with, and in some cases directly contradict, the study's main findings, suggesting the associations may be statistical artifacts rather than robust biological effects. In this analysis, the increased risks for five of the six cancers highlighted in the main analysis (thyroid, colorectal, lung, breast, and prostate) became statistically non-significant (Table 1, p. 3). Conversely, a large, statistically significant risk emerged for pancreatic cancer (HR 2.25), which was not significant in the main analysis. Furthermore, the article highlights new risks for gastric and pancreatic cancer but omits discussion of a statistically significant *protective* association found for leukemia (HR 0.56), which is reported in Table 1. These unexplained inconsistencies between the main and sub-analyses undermine confidence in the stability and validity of the reported associations.

Inadequate lookback period for defining incident cancer: The study uses a one-year lookback period to exclude individuals with a prior cancer diagnosis, a timeframe that is likely insufficient to reliably distinguish new (incident) cancers from

pre-existing (prevalent) ones (Supplementary File 1, p. 7). Many solid tumors develop slowly, and a person diagnosed with cancer 13 months prior to their index date would be included in the cohort. If this pre-existing cancer were detected again during post-vaccination follow-up, it would be misclassified as a new case. This methodological choice significantly increases the risk that the study is counting prevalent cancers as incident, which could create a spurious association if vaccinated individuals were more likely to undergo screening or medical evaluation.

Anomalous age-stratified findings: The study's age-stratified data contains anomalous patterns that are inconsistent with established cancer epidemiology and provide further evidence for surveillance bias. For thyroid cancer, the 1-year cumulative incidence in the vaccinated group shows an inverted pattern, decreasing with age from 8.46 per 10,000 in those under 65 to 3.66 in those 75 and older (Supplementary File 3, p. 6, Table S5). This runs contrary to the typical trend of increasing cancer incidence with age and suggests that younger, vaccinated individuals may have undergone more intensive screening or medical evaluation. This internal inconsistency was not discussed by the authors and weakens the interpretation that vaccination is causally associated with cancer risk.

Insufficient transparency in propensity score matching: The article lacks sufficient detail regarding its propensity score matching (PSM) methodology, preventing a full assessment of its quality. Standard practice for demonstrating the effectiveness of PSM includes providing a table of baseline characteristics for the cohorts both before and after matching; however, a pre-match table for the main cohort is not provided. Furthermore, the post-match standardized mean difference (SMD) for income was 0.090, a value very close to the common threshold for imbalance (0.1), suggesting the possibility of residual confounding by socioeconomic status (Supplementary File 2, p. 4, Table S2). These omissions and potential weaknesses in the matching process reduce confidence that the two groups were adequately balanced on key covariates.

Potential for immortal time bias in the booster analysis: The design of the booster

dose sub-analysis may be affected by immortal time bias. To be included in the booster group, an individual must have survived without a cancer diagnosis for the period between their second and third doses. This period represents "immortal time" during which the outcome could not occur by definition. The non-booster group does not have an equivalent protected period, which could bias the results toward finding a lower risk in the booster group. The study's methods do not describe a landmark analysis or the use of time-dependent covariates, which are standard techniques to mitigate this type of bias, and the definition of the index date for this comparison is ambiguous (Supplementary File 1, p. 7).

Misleading generalization across different vaccine types: The study's abstract and conclusions generalize the findings to "COVID-19 vaccination" as a whole, which obscures substantial differences in the risk profiles observed between vaccine technologies. For several cancers, the association was predominantly or exclusively driven by cDNA (adenoviral vector) vaccines, which showed much higher hazard ratios than mRNA vaccines. For example, a significant association with gastric cancer and prostate cancer was found only for cDNA vaccines (Supplementary File 3, pp. 2, 4). While mRNA vaccines were also associated with increased risks for some cancers, pooling all vaccine types together in the main conclusions is an oversimplification that masks potentially important heterogeneity in the data.

Overstatement of clinical implications and lack of risk-benefit context: The article makes a clinical recommendation that appears premature given the study's acknowledged limitations. Despite stating that the findings do not establish a causal relationship, the authors conclude that "clinicians should prioritize monitoring the risk of gastric cancer in relation to COVID-19 booster doses" (p. 3). This is a significant interpretive leap. The article also fails to contextualize the findings by discussing the very small absolute risk increases in the context of the established benefits of COVID-19 vaccination in preventing severe disease and death. This lack of a risk-benefit framework may lead to misinterpretation of the study's public health

significance.

Presentation and clerical issues: The manuscript contains several clerical errors that affect its transparency and clarity. The cohort flow descriptions in the methods section contain arithmetic discrepancies, with tens of thousands of individuals unaccounted for in the exclusion totals (Supplementary File 1, p. 7). Additionally, several figure captions in the supplementary materials are inconsistent or erroneous. For example, the caption for Figure S2 refers to panels that do not match the figure's labels, and the caption for Figure S5 incorrectly describes a panel on breast cancer as showing data for lung cancer (Supplementary File 3, pp. 5, 12). These errors reduce confidence in the overall presentation and clarity of the manuscript.

Future Research

Control for surveillance intensity: Future research must quantify and control for the frequency of healthcare interactions. By adjusting for the number of medical visits and imaging procedures (e.g., CT scans, ultrasounds) performed in the year following vaccination, researchers could distinguish between increased disease incidence and increased detection.

Landmark analysis with extended follow-up: To mitigate reverse causation and detection bias, future studies should employ a landmark analysis that excludes the first year of follow-up. Assessing cancer risk starting from year two or three would provide a more biologically plausible window for observing potential oncogenic effects, filtering out prevalent cases detected during the vaccination process.

Differentiation of detection method: Future work should stratify cancers by the method of detection—symptomatic presentation versus incidental finding during screening. If the association is driven by surveillance bias, the excess risk should be concentrated in early-stage, screen-detected cancers (e.g., thyroid microcarcinomas, PSA-detected prostate cancer) rather than advanced, symptomatic disease.

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