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CLINICAL APPROVAL CONCORDANCE AND TUMOUR CONTEXT IN PRECLINICAL ANTICANCER DRUG RESPONSE

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ABSTRACT
Both preclinical tumour suppression and approval of an anticancer treatment for a specific cancer are used to prioritize treatments, but the correlation is not clear. An observational analysis was performed to assess the number of xenografts with a US Food and Drug Administration approval matched to the cancer type and the number of xenografts which are not approved by the US Food and Drug Administration and whether there were any differences in drug response. A 2025 data set of 1,173 cases of xenografts was screened, of which 736 single agent records with valid tumour-volume ratios were analysed for breast, lung and colorectal cancers. Response was primarily defined as ≥ 40% reduction in tumour volume compared to the control group. Response occurred in 230 of 441 approval-matched records (52.2%) and 126 of 295 nonmatched records (42.7%). The association was unclear after adjustment for tumour type and clustering by publication (odds ratio 1.42, 95% confidence interval 0.93-2.17; p = .103) and was not supported by any interaction with approval-by-tumour. A stricter 60% reduction threshold yielded an odds ratio of 1.61 (95% confidence interval 1.05–2.49; p = .030). These retrospective correlations don't prove that the xenograft results translate to patient outcomes or eventual approval.

Keywords: anticancer drugs, mouse xenografts, FDA approval, tumour context, preclinical drug response

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1. Introduction

Decisions regarding efficacy, tolerability and patient needs are necessary before an anticancer drug progresses from laboratory experiments to clinical use. Tumour suppression in vivo means it is active but no patient benefit for the same tumour has yet been obtained. Besides, trial design, dose selection, pharmacokinetics, and being used alone or as combination (Fudio et al., 2022) are crucial for clinical development. This is evident in the field of immuno-oncology, where the immune context within the model and other immunological markers have an influence on the interpretation of preclinical results (Franklin et al., 2022). Looking at experimental responses and clinical approval can shed light on one limited component of translation.

With mouse xenografts, investigators can view the growth and drug activity of the tumour in vivo over time. They depend on the characteristics of the implanted tumour, the host animal, and other molecular characteristics of the tumor; seemingly similar models may not respond in the same way (Zaky et al., 2025). Patient-derived models can retain patient-relevant traits for selection of treatments, but the results depend on the conditions of the model generation and experiments (Genta et al., 2022). Additionally, the representativeness of cell lines selected as tumor surrogates varies from cancer to cancer (Bose & Bozdag, 2024). This example of a patient-derived, characterized vulvar xenograft highlights the value and cancer specificity of patient-derived models (Lin et al., 2026).

A careful meaning of "concordance" is required here. For safety, agreement between animal toxicity findings and adverse events in humans is required, but for antitumour efficacy, such agreement is not required, and cannot establish that xenograft responses predict patient benefit (Liu & Fan, 2026). A review of animal toxicity data for existing drugs used for cancer also covered clinical safety endpoints (Choi et al., 2026). A more focused question for a retrospective response data set is whether xenograft response is different between approved and unapproved drugs for the same cancer. This explores the relationship between being affiliated with an existing approximation label. It is unable to determine when approval was given, what experiment a trial was based on, or if a patient would respond.

This distinction is significant in the context of a tumour. The meaning of a response will differ depending on the histology, as biological pathways, acquired resistance and treatment exposure are different for different cancers. Combining tumour transcriptomics and pharmacologic data of each individual could help fill in missing information not captured by tissue labels (Rusciano, 2026). Patient-derived organoids (PDO) from malignant pleural effusions (MPEs) offer a tool to study therapeutic sensitivity in thoracic cancers (Ferreiro-Miguens et al., 2026). Organoid studies provide additional avenues for the analysis of response, whilst maintaining elements of tumour heterogeneity (Bai et al., 2026). With these approaches, the xenograft summaries ask the question, is the correlation between approval and measured response maintained or changed across tumour types?

The answer is influenced by the model that is chosen. Although cell cultures, organoids, and animal systems are all employed in cancer drug screening, they each have their own advantages and disadvantages in terms of scalability and biological fidelity (Motohashi et al., 2025). Some efficacy assessments can be done in 3-dimensional cultures, which could also reduce the need for animal experiments, and/or give additional information (Tosca et al., 2023). Newer approaches to regulating models also involve consideration of a purpose and evidence standard (Mirlohi et al., 2025). When dealing with drug combinations, measured synergy is dependent on the tested agents and biological system; and single-agent inhibition cannot be used to establish a combination effect (Xu et al., 2026). Analyses that combine drugs, tumour types and endpoints without making comparisons are therefore likely to overestimate generalizability.

A computational approach provides a different viewpoint. A link between patient-derived xenograft responses and clinical patient data, which is available in some cases, is shown to demonstrate the value of linking experimental and human outcomes where possible (Yang et al., 2025). Preclinical screening reviews have highlighted the inconsistencies in model conditions, as well as limited clinical predictiveness, as barriers to translation (Yadav et al., 2026). Approval status can be considered a contextual clinical marker rather than a measure of individual benefit if there is no linked patient-level outcomes. It is still possible to observe if there is a higher prevalence of tumour-volume

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suppression between drug–cancer pairs marked as approved and if this pattern differs between histologies. Conclusions must be within those limits of observation.

There is a curated xenograft dataset available as of 2025, which contains 1,173 samples of breast, colon, colorectal, and lung cancers (Lee, 2025). It contains information on the drug, the type of tumour, the measurement of the tumour volume after treatment compared to the control tumour, the publication identifier and the FDA-approved annotation for cancer. Combining colon and colorectal data allows for comparison with breast and lung cancer data, and the statistical adjustment for publication-level clustering corrects for dependence between observations from the same publication. Single drug focus on eligible single drug records prevents assigning combination effects to one drug. In this analysis, the association of preclinical response and approval annotation is explored, without assuming that approval is evidence of clinical effectiveness for an experimental record, and in the context of tumour type. The Objectives of this study are as follows:

1.Compare tumour-volume response between approved and unapproved drug–cancer pairs in eligible single-agent xenograft records

2.Determine whether the association between approval status and preclinical response varies across breast, lung, and colorectal cancers

3.Assess the robustness of observed associations using stricter response thresholds and publication-aware statistical analysis

2. Methodology

2.1 Research Design

This analysis was performed on data from mouse xenografts to determine if responses correlated with approval data for the same drug–cancer pair (Lee, 2025). Each treatment record, rather than an individual mouse or patient, was the analytic unit. Single drugs treatments were used as the main comparator as combination approvals are not immediately obvious from drug names. Differences across breast, lung and colorectal contexts were analysed and the potential modification of the approval association by tumour type was tested. When the same record appeared more than once from a publication, it was considered to be a related observation. Design does not determine clinical effectiveness, causality or predictive ability.

2.2 Data Source

Data is from the Version 1 release of the 2025 Census compiled by Lee (2025). It has 1173 xenograft treatment records in its workbooks, including the name of the drug used, the dose administered, the tumour model used, the histology of the tumour, the relative tumour volume, any noted toxicity, approval annotations and the source PMIDs. The “Sheet” tab was used for the analysis; ancillary calculation sheets were not considered to be additional observations. Tumour-volume ratios indicated outcomes measured against controls, while the approval field indicated if the drug matched an indication for the cancer tested. Experimental findings from animal-models only, no patient-level clinical treatment outcomes.

2.3 Sample Selection and Data Preparation

Combination treatments (those with a plus sign, slash or arrow in the drug name) were considered separately and excluded from the primary analysis. Records that were labelled Clinical phase, records without a numeric tumour-volume ratio, and negative ratios were not used in the single-drug approval comparison. Any ratio larger than one was kept because the volume of the tumour can be larger than the control volume; a sensitivity check was performed without the ratios greater than three. Colon and Colorectal labels were combined, and Breast and Lung were retained. IDs for samples were preserved for exclusion trace; PMIDs were normalized for publication identifiers.

2.4 Response and Approval Measures

The primary endpoint was a tumour volume reduction of ≥40% from the control (treated/control ratio of ≤0.60). The stricter threshold (at or below 0.40) of at least 60% reduction was examined separately. Exposure was defined by the drug–target cancer matched approval label (Y/N) on the workbook. The annotation provided was used as provided and historical dates of approval of individual indications

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were not independently verified. Therefore, concordance is not a patient response, but rather a cross-sectional association of a preclinical measurement and recorded approval.

2.5 Statistical Analysis

The study sample and approval groups were summarized by frequencies, percentages, and response proportions. The crude association was tested at each threshold using Pearson's chi-square test. Approval–response odds ratios derived from logistic regression and adjusted for pooled tumour type (95% confidence interval based on publication-clustered sandwich standard errors). A joint approval-by-tumour interaction was tested for variation across cancer groups. The sensitivity checks used were the tougher response threshold and the removal of ratios greater than three. Statistically significant two-sided p values were defined as < 0.05, and the threshold was considered with caution due to multiple related comparisons being made.

3. Results

3.1 Sample Composition

Of the 1,173 treatment records uploaded in the 2025 workbook, 806 included information on one drug and 367 on combinations. Single-drug record with usable volume ratio and binary approval label: primary comparison. As can be seen from Table 1, 42 single-drug records were noted as Clinical phase, 27 records did not have a numeric ratio, and one record had a negative ratio. The other 736 records were from 146 PMIDs and included 356 breast observations, 240 lung observations, and 140 pooled colorectal observations. These counts are not of independent animals or experiments (Figure 1) but of treatment records.

Table 1. Sample selection and tumour composition

Stage or characteristic	Records, *n*	Denominator, *n*	Percent
Full dataset	1,173	1,173	100.0
Single-drug records	806	1,173	68.7
Combination records	367	1,173	31.3
Single-drug records excluded: Clinical phase	42	806	5.2
Single-drug records excluded: unavailable volume ratio	27	806	3.3
Single-drug records excluded: negative volume ratio	1	806	0.1
Primary analytic sample	736	806	91.3
Breast cancer	356	736	48.4
Lung cancer	240	736	32.6
Pooled colorectal cancer	140	736	19.0

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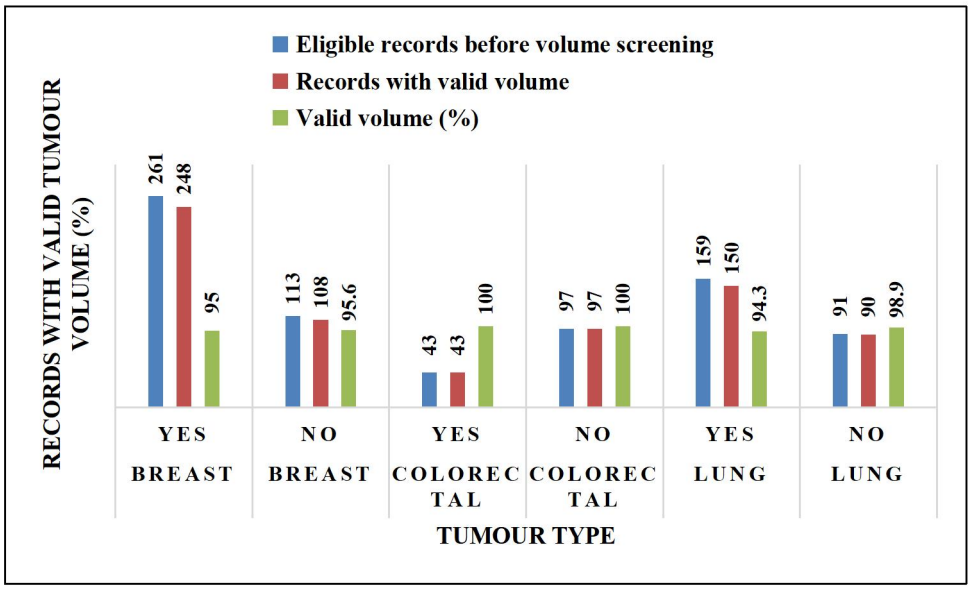

Figure 1. Availability of tumour-volume measurements

3.2 Response at the 40% Threshold

Tumour-volume reduction was seen with at least 40% reduction in 230/441 matched approval records (52.2%) and 126/295 non-matched approval (42.7%). An observed higher proportion of cancers in the approved group showed response. The highest number difference in pooled colorectal cancer is demonstrated in table 2. The overall crude association was statistically significant ($\chi^2 = 6.31$, $p = 0.012$). The adjusted analysis is more appropriate because these proportions do not necessarily reflect differences in the composition of the tumours or the repeated observations from the same publication (Figure 2).

Table 2. At least 40% tumour-volume reduction by tumour type and matched approval

Tumour type	Matched approval	Records, *n*	Responders, *n*	Response, %
All types	Yes	441	230	52.2
All types	No	295	126	42.7
Breast	Yes	248	138	55.6
Breast	No	108	51	47.2
Lung	Yes	150	68	45.3
Lung	No	90	36	40.0
Colorectal, pooled	Yes	43	24	55.8
Colorectal, pooled	No	97	39	40.2

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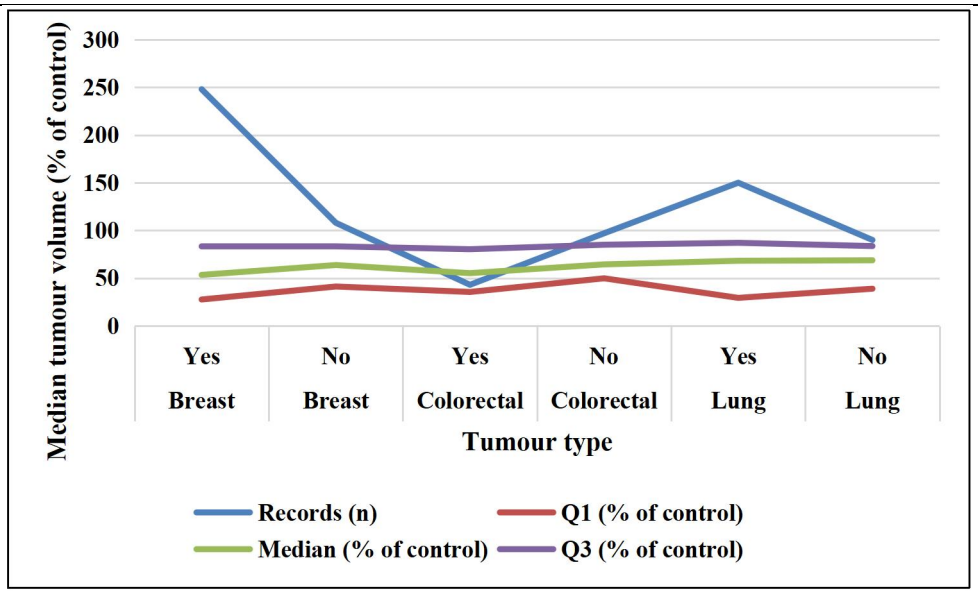

Figure 2. Tumour volume by cancer type and approval match

3.3 Adjusted Approval Association

Crude difference between approval groups was less certain after adjustment for tumour type and clustering of records by publication. For the adjusted model, there was no difference in the odds of response between the two cancer types – lung and pooled colorectal cancer – and breast cancer. Table 3 shows an approval odds ratio of 1.42 at the 40% response threshold (95% CI: 0.93–2.17; p = 0.103). There was no evidence for different associations by tumour type in an approval-by-tumour interaction test (p = 0.805). Thus, there was no clear approval–response relationship for the primary analysis (Figure 3).

Table 3. Adjusted odds of at least 40% tumour-volume reduction

Variable	Comparison	Adjusted OR	95% CI	*p* value
Matched approval	Yes vs No	1.42	0.93–2.17	0.103
Tumour type	Colorectal vs Breast	0.83	0.46–1.50	0.534
Tumour type	Lung vs Breast	0.69	0.43–1.10	0.118

Note. The model included 736 records. Confidence intervals and p values account for clustering within the 146 source PMIDs.

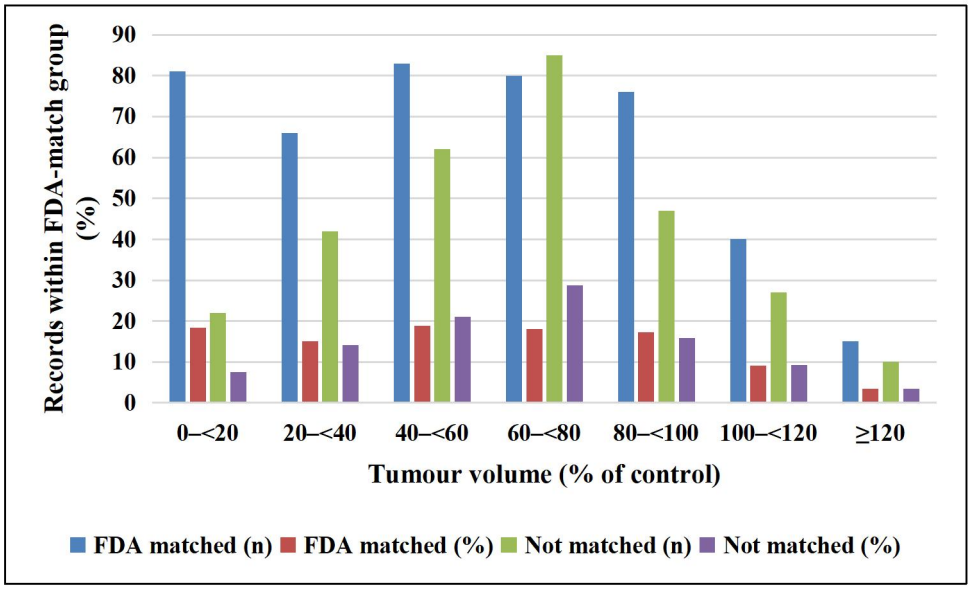

Figure 3. Distribution of tumour-volume measurements

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3.4 Sensitivity Analysis at the 60% Threshold

In 441 matched-approved records, 147 (33.3%) met the stricter 60% response threshold, while 64 of 295 (21.7%) matched-nonapproved records did. Adjusted approval association was also checked following the adjustment of tumour types and publication clustering. The number and percentages of each cancer type are displayed in Table 4. The adjusted odds ratio was 1.61 (95% CI: 1.05–2.49; p = 0.030). An estimate was then recalculated without the four exceptionally large positive ratios and remained similar (OR = 1.61; p = 0.033). Tumour interaction was not significant (p = 0.454) and this result should be interpreted with caution (Figure 4).

Table 4. At least 60% tumour-volume reduction by tumour type and matched approval

Tumour type	Matched approval	Records, *n*	Responders, *n*	Response, %
All types	Yes	441	147	33.3
All types	No	295	64	21.7
Breast	Yes	248	87	35.1
Breast	No	108	27	25.0
Lung	Yes	150	47	31.3
Lung	No	90	24	26.7
Colorectal, pooled	Yes	43	13	30.2
Colorectal, pooled	No	97	13	13.4

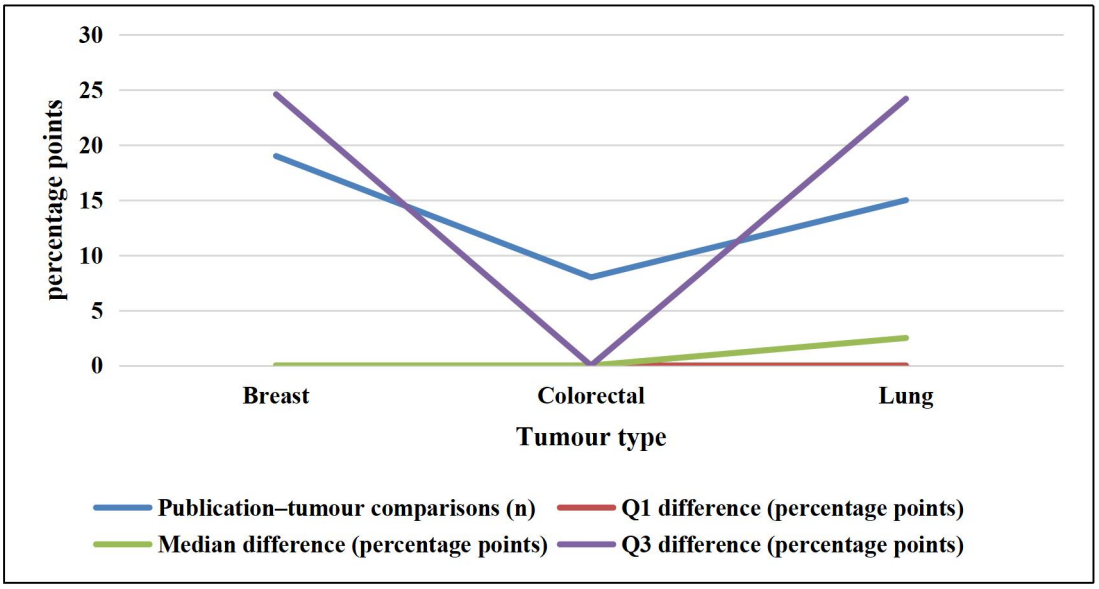

Figure 4. Within-publication differences by tumour type

4. Discussion

The eligible single-agent xenograft records revealed a weak positive correlation with cancer-matched FDA approval with tumour-volume suppression. Approved drug/cancer pairs had higher proportion of pairs that met response criterion than nonapproved pairs (52.2% vs 42.7% respectively) at the primary threshold of at least 40% reduction. There was a suggestion of a difference on the unadjusted comparison, and an adjusted odds ratio of 1.42 (95% CI 0.93–2.17; p = .103) in the comparison after

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adjustment for tumour type with publication-clustered uncertainty. The primary analysis thus offers weak evidence for the independent distinction of responsive experiments by approval matching. According to the description, the difference was the highest in colorectal cancer, the lowest in breast cancer and the lowest in lung cancer. There was no support for the approval-by-tumour interaction ($p = .805$) and these rankings do not demonstrate that the association varies by cancer type. At the stricter threshold of at least 60% reduction, the adjusted odds ratio was 1.61 (95% CI 1.05–2.49; $p = .030$). This finding should be interpreted with caution because it may be influenced by the definition of response.

The uncertainty of a pooled xenograft association is a testament to confusion that pervades the space of experimental models, which have retained varying aspects of the biology relevant to treatment response. Drug testing in a three-dimensional context was possible in scaffold-free renal-cancer tumoroids, which maintained tumour-related cellular characteristics (Séraudie et al., 2023). Pancreatic tumour-slice cultures maintained the tissue organisation and exhibited exploratory links between ex vivo drug sensitivity and patient outcome, where outcomes serve as a form of validation, for which there is no available data at present (Heckelmann et al., 2026). Patient-derived organoids and xenografts for gastric cancer have also been used in combination to enable more patient-specific drug testing (Yan et al., 2026). On the other hand, across cancers, the suitability of cell lines as proxies to patient tumours also hinges on the biological context (Bose & Bozdag, 2022).

Other work demonstrates what extra information a translatorial analysis can make use of. The identification of breast-cancer drug prioritization by the gene regulation mechanism include molecular information that are not available in the existing tumour-volume records (Huang & Ovcharenko, 2026). In precision-oncology research, molecular and clinical data are also taken into account beyond one experiment endpoint (Goda & Abdel-Aziz, 2025). Approval and the clinical indication are important to discuss, as demonstrated with the development of antibody–drug conjugates for platinum-resistant ovarian cancer (Zhao et al., 2025). The manufacturing and regulation challenges of oncology nanotheranostics also highlight why model effectiveness does not necessarily translate to clinical approval (Alum et al., 2026). In an expert-driven decision support system evaluated on simulated scenarios, tumour-specific evidence, approval status and guidelines were combined (Lammert et al., 2024). We cannot compare the results of these studies with the approval association reported here because they vary in terms of model and outcomes.

Approval matching will provide the clinical context for a series of otherwise disparate xenograft experiments. The designated drug–cancer pairing maintains the distinction of a drug being approved for one cancer but not for another. The baseline response differences are also hidden by stratifying by tumour type. However, the analysis of interaction is not enough to determine the strength of the concept of approval concordance for breast, lung and colorectal cancers. Additional data for future datasets would be useful to include approval date, molecular subtype, tumour of origin, dose, length of therapy and whether therapy was used in combination or singly. Then, by correlating experiments with patient outcomes, clinical translation could be directly assessed. These findings can be used to determine model and treatment contexts that are worth further investigation until such links are present. They cannot be used to select a particular patient's treatment.

Approval was provided by the dataset annotation and not reconstructed at the date of each original experiment. The result of an experiment may thus include a drug in the "approved" group so it is difficult to interpret the temporal prediction. Tumour-volume ratios were obtained from various dose, route, schedule, control and xenograft studies and may not be equivalent for the same treatment effect. A number of records were from the same publication. Statistical dependence was partially mitigated by clustering uncertainty by publication, but was unable to fully account for repeated testing on specific drugs and models, nor could it remove selection bias. Combination treatments were not used as the effect would not be directly attributable to the single agent and this could only be used in designing multidrug treatments. Further sample reduction was due to missing or invalid measurements. Above all, there are no associated patient responses or survival data. Clinical evidence, safety and regulatory decisions are also included in the FDA approval. The observed association is

thus with an approval label and not with a proven ability to predict patient benefit based on xenograft response.

5. Conclusion

The xenograft data for 2025 shows a weak correlation between FDA approval in cancer and preclinical tumour-volume suppression. A total of 1,173 cases were recorded of which 736 were single-agent records with a valid tumour-volume ratio and an approval annotation. A 40% reduction threshold was the primary one, with 230 out of 441 matched records (52.2%) and 126 out of 295 nonmatched records (42.7%) responding. When tumour type and clustering within publications were taken into account the association was weakened (adjusted OR 1.42; 95% CI 0.93 to 2.17; p = .103). There was descriptive difference greatest for colorectal cancer and lowest for lung cancer, but there was no analysis to conclude that the approval agreement varied according to tumour type. A more rigorous cut-off of ≥ 60% reduction resulted in a more robust adjusted association (OR = 1.61; 95% CI, 1.05 to 2.49; p = .030), suggesting that the definition of response is somewhat arbitrary. These data argue for the retention of tumour context in xenograft analyses and for granting approval based on the matching cancer indication. They do not demonstrate that any one experiment predicted later approval or outcome of a patient's treatment. No patient responses were associated with the records, approval dates were not reconstructed, and experimental regimens varied. Results are not clinical predictive accuracy, but association with approval status.

References

1. Alum, E. U., Obasi, D. C., Uzor, S., Eji, M. O., Amadi, E. B., Emeruwa, A. P., ... & Alum, B. N. (2026). Navigating the translational Valley of death in oncology nanotheranostics: manufacturing, biological, clinical, and regulatory barriers to implementation. Nanomedicine, 1-20.
2. Bai, M., Liu, M., Wang, S., Ran, M., Ye, J., Xie, Y., ... & Li, H. (2026). Organoids in cancer therapy: translational applications and clinical promise. Molecular Cancer.
3. Bose, B., & Bozdag, S. (2022). Finding the best cell lines across pan-cancer to use in pre-clinical research as a proxy for patient tumor samples considering immune cells, multi-omics, and cancer pathways. bioRxiv, 2022-12.
4. Bose, B., & Bozdag, S. (2024). Identifying cell lines across pan-cancer to be used in preclinical research as a proxy for patient tumor samples. Communications Biology, 7(1), 1101.
5. Choi, G., Bae, S., & Lim, K. M. (2026). Analysis of predictive value of animal repeated dose toxicity study results for clinical safety of US FDA-approved anticancer drugs between 2019 and 2023. Regulatory Toxicology and Pharmacology, 106031.
6. Ferreiro-Miguens, R., Diez-Grandio, I., Soto, R., Ferreiro, L., Garcia, J., Otero-Alen, M., ... & Leon-Mateos, L. (2026). Proof-of-concept study in patient-derived organoids from malignant pleural effusion for functional testing in thoracic tumors. Biomedicine & Pharmacotherapy, 203, 119816.
7. Franklin, M. R., Platero, S., Saini, K. S., Curigliano, G., & Anderson, S. (2022). Immuno-oncology trends: preclinical models, biomarkers, and clinical development. Journal for immunotherapy of cancer, 10(1), e003231.
8. Fudio, S., Sellers, A., Pérez Ramos, L., Gil-Alberdi, B., Zeaiter, A., Urroz, M., ... & Lubomirov, R. (2022). Anti-cancer drug combinations approved by US FDA from 2011 to 2021: main design features of clinical trials and role of pharmacokinetics. Cancer Chemotherapy and Pharmacology, 90(4), 285-299.
9. Genta, S., Coburn, B., Cescon, D. W., & Spreafico, A. (2022). Patient-derived cancer models: Valuable platforms for anticancer drug testing. Frontiers in oncology, 12, 976065.
10. Goda, R. Y., & Abdel-Aziz, A. K. (2025). Exploiting artificial intelligence in precision oncology: an updated comprehensive review. Journal of Translational Medicine, 23(1), 1397.
11. Heckelmann, B., Fetzner, D., Lapshyna, O., Hidam, A. S., Brauer, A., Wandmacher, A. M., ... & Braun, R. (2026). Tumor slice cultures enable rapid ex vivo profiling of patient-specific drug

sensitivity in pancreatic ductal adenocarcinoma. npj Precision Oncology, 10(1), 325.
12. Huang, X., & Ovcharenko, I. (2026). Using Deep Learning Models of Gene Regulation to Guide Drug Prioritization. Pharmaceuticals, 19(7), 1097.
13. Lammert, J., Dreyer, T., Mathes, S., Kuligin, L., Borm, K. J., Schatz, U. A., ... & Tschochohei, M. (2024). Expert-guided large language models for clinical decision support in precision oncology. JCO precision Oncology, 8, e2400478.
14. Lee, H. (2025). Supplementary dataset_Dataset of anticancer drug responses in mouse xenograft models (Version 1) [Data set]. Mendeley Data. https://doi.org/10.17632/rymw4t4r8r.1
15. Lin, Y., Chang, C., Liu, N., Chai, D., Cai, L., & He, Y. (2026). A clinically concordant vulvar cancer patient-derived xenograft model retaining primary molecular signature for preclinical drug sensitivity assessment. Open Medicine, 21(1), 20261419.
16. Liu, X., & Fan, F. (2026). A Large-Scale Concordance Study of Toxicity Findings Across Preclinical Species and Humans for Small Molecules and Biologics in Drug Development. bioRxiv, 2026-01.
17. Mirlohi, M. S., Yousefi, T., Aref, A. R., & Seyfoori, A. (2025). Integrating new approach methodologies (NAMs) into preclinical regulatory evaluation of oncology drugs. Biomimetics, 10(12), 796.
18. Motohashi, S., Katsuta, E., & Ban, D. (2025). Advances and challenges in drug screening for cancer therapy: A comprehensive review. Bioengineering, 12(12), 1315.
19. Rusciano, D. (2026). Molecular oncodiagnostics in precision oncology: integrating tumor transcriptomics, patient pharmacogenetics, and Ex Vivo chemoresistance testing to improve individual chemotherapy response. Journal of Personalized Medicine, 16(4), 176.
20. Séraudie, I., Pillet, C., Cesana, B., Bazelle, P., Jeanneret, F., Evrard, B., ... & Filhol, O. (2023). A new scaffold-free tumoroid model provides a robust preclinical tool to investigate invasion and drug response in Renal Cell Carcinoma. Cell Death & Disease, 14(9), 622.
21. Tosca, E. M., Ronchi, D., Facciolo, D., & Magni, P. (2023). Replacement, reduction, and refinement of animal experiments in anticancer drug development: the contribution of 3D in vitro cancer models in the drug efficacy assessment. Biomedicines, 11(4), 1058.
22. Xu, T., Xu, M., Ngan, D. K., Zhao, T., Chen, C. Z., Zheng, W., & Huang, R. (2026). In vitro bioactivity profile-driven discovery of drug synergies in hepatocellular carcinoma. Journal of Translational Medicine.
23. Yadav, R., Singh, S., Kumar, S., Kolay, A., Sharma, H., Dorai, R., ... & Yadav, M. K. (2026). Preclinical screening models in anticancer drug development: strengths, limitations, and translational challenges. Frontiers in Pharmacology, 17, 1822764.
24. Yan, H., He, X., Hou, X., Liao, G., Li, H., Duan, X., ... & Tang, J. (2026). Integrating Patient-derived Organoid and Patient-derived Xenograft Models to Guide Precision Chemotherapy for Gastric Cancer. Anticancer Research, 46(9), 4895-4908.
25. Yang, J., Wu, T., Xue, F., Gong, J., & Niu, X. (2025). TRANSPIRE-DRP: a deep learning framework for translating patient-derived xenograft drug response to clinical patients via domain adaptation. Journal of Translational Medicine, 23(1), 1340.
26. Zaky, M. Y., Lamloum, N. S., Ahmed, N. A., & Ahmed, O. M. (2025). Preclinical in vivo animal xenograft models: heterogeneity and biomarkers of therapeutic response and resistance. In Preclinical cancer models for translational research and drug development (pp. 109-127). Singapore: Springer Nature Singapore.
27. Zhao, Y., Yuan, H., Li, N., & Wu, L. (2025). A mini-overview of antibody-drug conjugates in platinum-resistant ovarian cancer: A preclinical and clinical perspective. International Journal of Biological Macromolecules, 304, 140767.